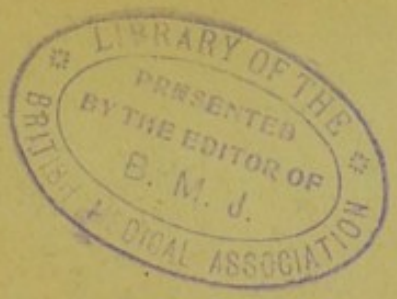
Diseases of the skin : including radiotherapy and radiumtherapy / by Ernest Gaucher ; translated and edited by C.F. Marshall.

DISEASES OF THE SKIN

E. GAUCHER


LIBRARY OF THE
PRESENTED
BY THE EDITOR OF
B. M. J.
BRITISH MEDICAL ASSOCIATION

DISEASES OF THE SKIN

DISEASES OF THE SKIN

INCLUDING RADIOTHERAPY AND RADIUMTHERAPY

BY ERNEST GAUCHER

PROFESSOR OF CUTANEOUS AND SYPHILITIC DISEASES AT THE FACULTY OF MEDICINE AND PHYSICIAN TO THE ST LOUIS HOSPITAL, PARIS

TRANSLATED AND EDITED BY

C. F. MARSHALL,

M.Sc., M.D., F.R.C.S.

SURGEON TO THE BRITISH SKIN HOSPITAL; LATE ASSISTANT SURGEON TO THE HOSPITAL FOR DISEASES OF THE SKIN, BLACKFRIARS

WITH NUMEROUS ILLUSTRATIONS

LONDON

JOHN MURRAY, ALBEMARLE STREET, W.

1910

PREFACE

THIS book comprises the second edition (1909) of the volume on Diseases of the Skin in the *Nouveau Traité de Médecine* (Brouardel, Gilbert and Thoinot), written by Professor Gaucher in collaboration with other authorities; together with the chapters on Cutaneous Syphilides and the Treatment of Syphilis from Professor Gaucher's *Précis de Syphiligraphie.*

The most recent developments of Radiumtherapy are described by Wickham, Degrais and Domenici. The treatment of Nævi by X-rays is described by Gastou, and general Radiotherapy has been revised by Zimmern. Special articles have been written on Sporotrichosis by Monier-Vinard; on Blastomycosis by Rubens-Duval; and on Mycosis fungoides by Domenici. A brief account of Yaws has been added by the Editor.

The illustrations are mostly from models in the St Louis Hospital Museum and from Professor Gaucher's private collection. Two figures of the *Spirochæta pallida* have been kindly lent by Dr Levaditi and Mr J. E. R. M'Donagh. One is used by courteous permission of the proprietors of the *Medical Annual.*

C. F. M.

LONDON, *May* 1910.

CONTENTS

DISEASES OF THE SKIN

INTRODUCTION

ALL cutaneous affections can be reduced to elementary lesions, which characterise the disease at its period of maturity, and the study of these lesions should precede the description of diseases of the skin considered individually. The special descriptions should also be preceded by an exposition of the general etiology of dermatoses; for, it is not enough to diagnose the lesion, it is also necessary to recognise the nature of the disease, to ascertain whether it is of microbial, toxic or diathetic origin. In this way we can establish a precise and complete diagnosis and institute efficacious treatment.

As this work does not pretend to be anything more than a manual of practical dermatology, I have not devoted a special chapter to general pathological anatomy, but have mentioned the more important and characteristic histological lesions in the description of each individual disease. For the same reason, instead of writing a chapter on the general treatment of cutaneous affections, I have given the treatment of each disease after its symptomatic description; confining myself, moreover, to useful methods of treatment, the value of which I have proved by personal experience. For, it must be borne in mind that, especially in recent times, the treatment of skin diseases has been unnecessarily complicated, each dermatologist endeavouring to invent a new remedy, which has only an ephemeral existence. There are, indeed, but few efficacious medicaments, and it is only these which I have retained and recommended.

This work is, therefore, divided into three parts:—

1. Elementary lesions of the skin;
2. General etiology of diseases of the skin;
3. Special description of diseases of the skin.

The order in which the cutaneous affections are described cannot be that of a natural classification, because we are still ignorant of the true nature of certain dermatoses. Thus, the first division—to

which I give the name of common inflammatory dermatoses—includes at the same time the principal diathetic dermatoses, and diseases of variable or undetermined nature, such as the erythemas, purpura, herpes, pemphigus, pityriasis rosea, pityriasis rubra, and pityriasis pilaris. It is by reason of the form of their elementary lesion that the latter have been placed with other erythematous, vesicular or squamous affections of more definite nature.

The other divisions constitute more natural groups:—Diseases of the pilo-sebaceous follicles; diseases of the sweat glands; diseases of the hairs; diseases of the nails; dyschromatous dermatoses; vascular and lymphatic dermatoses; hypertrophic dermatoses (epidermic, epidermo-papillary, dermic hypertrophies); atrophic dermatoses; neoplastic dermatoses.

The preceding divisions are, to a certain extent, anatomical. The following are etiological:—Microbial dermatoses; parasitic dermatoses (vegetable and animal parasites).

Although these divisions are not all established on the same principles, they have the advantage of grouping together diseases which are more or less similar.

ELEMENTARY LESIONS OF THE SKIN.

The elementary lesions, on which depends the morphological diagnosis of every disease of the skin, are divided into two main classes—primary and secondary.

The *primary* lesions include: macules or spots, squames, vesicles, bullæ, pustules, papules, tubercles or tuberosities (the word tubercle is used here in its morphological sense), tumours, hypertrophies and atrophies.

The *secondary* lesions are: excoriations, ulcerations, fissures, crusts and cicatrices.

Primary Elementary Lesions.

Macules.—We must first of all distinguish between *congestive* or *inflammatory macules*, due to hyperæmia of the papillary vessels; *vascular macules* and *hæmatic macules*, the former produced by permanent dilatation of the vessels, the latter by infiltration of blood into the skin; *hyperchromic* and *achromic macules*, due to the accumulation of pigment or some colouring matter in the cells of the Malpighian layer, or to the absence of pigment in these cells.

Congestive Macules.—*Erythema.*—These lesions are transitory and disappear after death. Sometimes the epidermis presents cell lesions; a certain number of cells of the Malpighian layer show vesicular changes consisting in swelling of the nucleolus, then of the

nucleus which fills the whole cell. The colour of the erythematous spots varies from rose to red, and disappears momentarily under pressure of the finger. Their shape is generally rounded or polycyclic. These spots usually give rise to slight burning or itching, and terminate by desquamation. Their size is variable; sometimes the redness consists of spots limited to certain regions of the body, more or less abundant (*macular erythema*), or of spots which become fused with each other (*diffuse erythema*); sometimes the spots extend over the whole surface of the skin, as in measles (*morbilliform* or *rubeoliform erythema, roseola*). At other times the rash forms a diffuse redness over the whole cutaneous surface, similar to that of scarlet fever (*scarlatiniform erythema*).

But the spots may assume other forms:—*Marginate erythema* is characterised by rounded spots with raised margins; *circinate erythema* forms a circle with normal skin in the centre. In other cases the erythema presents raised projections above the surface of the skin (*papular, papulo-tubercular erythema, erythema urticans, urticaria*). Sometimes the projection is deeply imbedded in the skin (*erythema nodosum*). Lastly, the congestion of the erythematous spots may be sufficient to cause abundant exudation and elevation of the horny layer of the epidermis (*vesicular* and *bullous erythema*). The vesicle may occupy the centre or the whole surface of the erythematous spot. Another variety of vesiculo-bullous erythema is *herpes iris* or *hydroa* (see Polymorphous Rheumatic Erythema).

Vascular Macules—Hæmatic Macules.—*Vascular macules* are formed by permanent dilatation of the small vessels of the skin, constituting what are called *vascular nævi*, or *telangiectases*. They differ from congestive macules in the absence of burning or itching, and desquamation. They are generally permanent, but disappear momentarily under pressure. Formed by the dilatation of pre-existing capillaries and by the formation of new capillaries, they are sometimes of a red colour, sometimes violet like lees of wine.

Hæmatic or *purpuric macules* are due to infiltration of blood in the dermis or papillæ. They differ from congestive and vascular macules in not disappearing under pressure of the finger. They have a purple colour, which afterwards becomes green and then yellow owing to changes in the blood effused in the tissues. Sometimes hematic macules are not due primarily to extravasation of blood by rupture of a vessel, but to exaggerated dilatation of the small vessels (telangiectasis) which precedes their rupture. This lesion occurs especially around the pilo-sebaceous follicles.

Hyperchromic Macules—Achromic Macules.—*Hyperchromic macules*, usually due to accumulation of pigment normally present in the deep cells of the epidermis, constitute pigmentary or melanodermic spots. But they may be produced by another colouring

matter; for instance, the spots occurring in persons who have absorbed nitrate of silver; the blue spots due to the secretions of *pediculi pubis*, etc. These macules cause neither pain nor itching; they are rather disfigurements than true diseases.

Achromic macules result from atrophy of pigment; they are areas in which the normal cutaneous pigment is absent (partial albinism, vitiligo, etc.).

Squames.—These are epidermic scales of various sizes; when very fine, they are called furfuraceous squames. They adhere more or less to the subjacent surface, which is sometimes red, sometimes white, according to the presence or absence of inflammation. Squames may be primary or secondary. Primary squames result from exaggerated proliferation of the cells of the epidermis, accompanied by deficient keratinisation; the cells being continually produced without becoming stratified to form the horny layer. Secondary squames are consecutive to other elementary lesions of the skin—erythemas, vesicles, bullæ, pustules, papules, tubercles.

The primary squamous dermatoses comprise pityriasis simplex, pityriasis rosea, pityriasis pilaris, psoriasis, exfoliative dermatitis, the squamous syphilide, pityriasis alba (trichophytic), pityriasis versicolor, and ichthyosis.

Vesicles.—These are transparent elevations, hemispherical or acuminate in form. Varying in size from a millet-seed to a hemp-seed, they enclose a liquid which is generally clear and alkaline, sometimes hæmorrhagic, formed by exudation from the capillaries; except in the case of *sudamina* and *dysidrosis*, which are vesicular lesions with acid contents formed by the sudoriparous secretion.

Histologically, there are two kinds of vesicles: one belonging exclusively to eczema, the other including all other vesicles, of which the herpetic vesicle is the type.

The evolution of the eczematous vesicle takes place in the following manner:—First of all a red spot appears, resulting from papillary congestion, and accompanied by a little exudation; this exudation becomes more and more abundant and accumulates in the Malpighian layer to form the vesicle. The cells of the Malpighian layer present at the same time a lesion described under the name of vesicular degeneration; the cells become globular and finally rupture, opening into one another; their partial destruction resulting in the formation of a cavity which becomes filled with liquid and raises the epidermis. After a time, the contents of the vesicles either become absorbed, or break through and form a superficial ulceration, which becomes covered by a crust; this eventually falls off, and all trace of the lesion disappears.

In all other vesicles, of which herpes may be taken for the type, the vesicular cavity is situated, not in the Malpighian layer of cells

as in eczema, but underneath the horny layer, which becomes raised by the collection of liquid beneath it.

The vesicular dermatoses comprise eczema, vesico-bullous erythema, hydroa, herpes, some eruptions caused by irritants, certain eruptions of toxic origin, the vesicular syphilide, miliaria, dysidrosis, and trichophytic circinate herpes.

Bullæ.—These are hemispherical elevations, transparent, or opaque when containing pus, sometimes hæmorrhagic. They are large vesicles, resulting, like the vesicle of herpes, from elevation of the horny layer of the epidermis. The type of the bulla is that produced by the application of a blister, or the phlyctenule of a burn of the second degree. The liquid contents of bullæ is alkaline or neutral, like that of vesicles; it is albuminous, and contains epithelial cells and leucocytes.

The bulla has the same evolution as the vesicle. There is first of all congestive redness with serous exudation, which raises the epidermis; sometimes the raised epidermis ruptures and forms a superficial ulceration; sometimes the liquid dries up in the form of a crust; sometimes the liquid becomes absorbed, and the wall of the bulla forms a fine squame which eventually falls off.

The dermatoses characterised by the formation of bullæ include all the varieties of pemphigus, the bullous erythemas, and the bullous syphilide or syphilitic pemphigus. The application of cantharides powder to the skin also gives rise to bullæ.

Pustules.—Pustules are purulent vesicles. Like the vesicle, the pustule commences as an erythematous macule; underneath this occurs serous infiltration in the papillæ, which become swollen and transform the macule into an elevation called a *papule*. This infiltration penetrates the epidermis and loosens its cells, forming a cavity, which is nothing more than a vesicle. Some of the cells also undergo vesicular or vacuolar degeneration. In each vesicle thus formed are found, along with altered cells, granules and microbes, leucocytes which increase in number, filling the cavity and transforming the vesicle into a pustule. The pustules, when they dry up, form a crust. After the crust falls off, either an insignificant cicatrix is produced, when the lesion is chiefly epidermic and the papillæ almost intact; or else there remains an indelible cicatrix, when the dermis is partly destroyed, the papillary vessels having been obstructed by masses of leucocytes formed around them. Hence the distinction established by Virchow and Rindfleisch into parenchymatous pustules and catarrhal pustules, a distinction made long ago by Willan, who divided pustules into two groups: *phlysaciate* pustules and *psydraciate* pustules.

Phlysaciate (φλυζειν, "to burn") pustules are large, with a hard base, and surrounded with a red periphery. Under the crust the dermis is ulcerated, and these pustules leave cicatrices.

Psydraciate (ψυδραίχια, "pustule") are superficial, have no red periphery, and give rise to no cicatrix.

In smallpox, vaccinia, the pustular syphilide, ecthyma, we find phlysaciate pustules; in impetigo, and in certain eruptions due to irritant applications, the pustules are psydraciate.

Papules.—These are red, solid elevations, varying in size from a pin's head to a lentil, localised or generalised, isolated or agglomerated. Of all the elementary lesions of the skin, it is these which cause the most intense itching, and which lead to the most scratching. Papules excoriated by itching become covered with reddish crusts, formed by dried blood. Exception must be made for syphilis, the papular eruptions of which are nearly always, but not quite always, non-pruriginous.

Anatomically, the papule is only a papilla increased in its dimensions, or an agglomeration of hypertrophied papillæ. There is first of all congestion of a certain number of papillæ, then exudation of serous liquid which swells the papillæ, and at the same time proliferation of the connective tissue. The horny layer of the epidermis is intact, or is only modified secondarily. The situation of the papule in the papillary body accounts, to a certain extent, for the violent itching which characterises papular eruptions; for we know that it is in the papillæ that the nerve-endings are found. Papules generally terminate by simple subsidence of the projections.

The principal dermatoses characterised by papules are lichen, strophulus, prurigo, papular syphilide, scabies, pediculosis, etc.

Among the papular dermatoses there is a special kind which develops round the pilo-sebaceous follicles (*folliculitis*); the type of this is acne with all its varieties.

In folliculitis, a conical papule is formed around the excretory duct of the follicle. In most cases this papule becomes a pustule, under the influence of the agents of suppuration which penetrate it. On the chin, inflammation of the pilo-sebaceous follicles produces a pustular eruption called *sycosis*.

Tubercles—Tumours.—As already mentioned, the word tubercle applies not only to bacillary tuberculosis; it is synonymous with tuberosity or nodosity. Tubercles vary in size, so that it is difficult to say where tubercle ends and tumour begins: as a rule, the word tumour is applied to tubercles which exceed the size of a nut. The structure of tubercles differs according to the disease to which they belong; but, in a general way, they are formed by embryonic proliferation—a connective tissue neoplasia—developed in the superficial part and even in the deeper parts of the dermis. The termination of tubercles varies according to the cause which produces them.

The principal dermatoses in which tubercles are met with are: tuberculous lupus, verrucose tuberculosis, anatomical tubercle, tuber-

culous gummas, tubercular syphilides, syphilitic gummas, tubercular leprosy, and Oriental boil.

Tumours include *fibroma molluscum*, keloid, xanthelasma, mycosis fungoides, sarcoma and epithelioma.

Hypertrophies—Atrophies.—The skin may be affected by hypertrophies and atrophies.

Hypertrophies may affect the epidermis, the papillary body, the dermis, or all parts of the skin together. Hypertrophies of the epidermis include ichthyosis, corns, horns, and keratosis. Papillary hypertrophy gives rise to warts, papillomas, condylomas, and vegetations. The dermis is hypertrophied in elephantiasis Arabum, myxœdema, and in sclerodermia, at any rate at the beginning of the latter disease.

Atrophy of the skin may be general, as in senile cutaneous atrophy, or partial, and may be limited to one of the constituent parts of the skin.

Secondary Elementary Lesions.

Excoriations—Ulcerations—Fissures.—*Excoriations* are losses of substance involving only the epidermis; *ulcerations* involve the papillary body and the superficial or deep parts of the dermis; *fissures* are linear ulcerations situated in the folds of the skin, especially observed around the natural orifices (mouth and anus), and sometimes in the folds of flexion of the joints.

Excoriations are secondary to vesicular, bullous, or superficial pustular eruptions; they occur in eczema, pemphigus and impetigo.

Ulcerations are secondary to phlysaciate pustules—the pustules of ecthyma, for example. Tubercles and tumours may also become ulcerated. Ulcerations have often an important diagnostic value, on account of the different characters which they present, according to the nature of the disease in which they occur. Thus, *tuberculous* ulcerations are irregular, with a sanious base and detached borders, and have little tendency to cicatrisation: *syphilitic* ulcerations, on the contrary, have more regular contours and usually little discharge; their borders are sharply cut out, and they may cicatrise partially and spontaneously.

Fissures may complicate eczema of the lips, anus and vulva, plantar and palmar eczema and psoriasis, and eczema of the articular folds.

Crusts.—These result from the drying of the discharge which bathes the surface of ulcerations.

Crusts have also diagnostic characters of some value. The crusts of eczema are soft, grayish, thin and fragile; those of impetigo are yellow and raised by purulent matter; those of ecthyma are thicker and more deeply imbedded, and of a brown colour. The crusts

covering tuberculous ulcerations are soft and non-adherent; those of syphilis, on the contrary, are thick, stratified, and adherent.

Cicatrices.—These are also very characteristic. The cicatrices of smallpox are irregular and scalloped; those which succeed tuberculous ulcerations are irregular, bridled, hypertrophic, and at first of a violet colour. Syphilitic cicatrices are very different; they are flat, smooth and regular; they are pigmented, and their pigmentation disappears progressively from the centre to the circumference, leaving finally a flat, white, depressed surface.

GENERAL ETIOLOGY OF SKIN DISEASES.

Diseases of the skin are produced by many causes, which can be divided into six groups: (1) traumatic non-parasitic agents; (2) parasites; (3) medicaments and foods; (4) diathesis, such as arthritism; (5) diseases or disturbances of the nervous system; (6) defects in conformation.

Traumatic Agents.—These are very diverse in nature, and include radiant heat, sunlight, electric light, cold, continued pressure from prolonged confinement to bed, irritating substances of animal, vegetable or mineral origin. But the majority of these causes only act on subjects who are predisposed; for instance, cold only causes chilblains in lymphatic subjects, and many irritating substances only produce eruptions in predisposed individuals. On the other hand, the part played by morbid predisposition must not be exaggerated, for certain substances applied to any person invariably determine the same effects.

Parasites.—We distinguish between animal parasites and vegetable parasites.

Among the *animal parasites*, some are intra-epidermic, such as the acarus of scabies; others, such as lice, live on the skin: hence the distinction into *dermatozoa* and *epizoa*. There are other parasites called *hæmatozoa* which reach the skin or subcutaneous cellular tissue by way of the circulation; these are the *filaria sanguinis hominis*, and the cysticercus of the cellular tissue.

The *vegetable parasites* include (1) vegetable parasites proper, and (2) microbes.

The former are divided into—(1) epidermic parasites, such as *microsporon furfur*, which causes pityriasis versicolor, and *Microsporon minutissimum*, which causes erythrasma; (2) parasites of the hairs and epidermis, such as *Trichophyton tonsurans* and *Achorion Schonleinii;* parasites of the cellular tissue, such as *Actinomyces* which causes actinomycosis, and *Sporotrichum*, which produces sporotrichosis.

The microbes are streptococci and staphylococci, which cause impetigo, ecthyma, and the various forms of pyodermatitis; the bacilli of tuberculosis, leprosy, glanders, anthrax, etc.

There are some microbes which appear to be protozoa; for example, the parasite of Oriental boil and the spirochætes of syphilis and yaws.

Parasites and microbes, some of them at any rate, do not act in the same way in different subjects: for example, scabies, which is always caused by the *acarus*, does not give rise to the same lesions in robust persons as it does in lymphatic individuals. Again, impetigo, which is always caused by pyogenic microbes, does not spread with the same facility in all subjects; the successive auto-inoculations occur more readily in children, and especially in lymphatic and scrofulous subjects, than in healthy individuals. Ecthyma also extends more rapidly in cachectic persons than in the robust. The trichophyton fungus of ringworm is never seen on the scalp of adults, while we know with what tenacity it clings to that of children.

Microbes produce dermatoses in several ways. In the first place, they may be inoculated directly in one or more parts of the body, like the pyogenic microbes of impetigo and ecthyma, or the tubercle bacillus in anatomical tubercle. They may invade the body by way of the respiratory or digestive tracts, and then give rise to various lesions on the surface of the skin; in this way tuberculous gummas are formed. On the other hand, these microbes may poison the body by the toxins which they form; it is probable that toxins give rise to the generalised eruptions so often observed during the course of eruptive fevers—enteric, diphtheria, cholera, and puerperal fever.

Lastly, several parasites may attack the same subject successively; for instance, scabies very often becomes complicated by ecthymatous eruptions due to secondary infection with pyogenic microbes, the infection being produced by scratching.

Aliments—Medicaments.—Some persons cannot eat mussels, strawberries, etc., without suffering from urticaria. A number of drugs—iodides, bromides, mercury, antipyrin, etc.—may also give rise to special eruptions. In all these cases the toxic substance acts either through the vaso-motor system, or by direct cutaneous elimination, whether the intoxication is of mineral, vegetable, or animal origin, like the ptomaine which is present in abnormal quantity in mussels.

But in all intoxications, both medicamentous and alimentary, we must allow for individual susceptibility. One person will support with impunity large doses of iodide of potassium, while another will present erythema or acne after the smallest doses. With

mercury also, idiosyncrasy plays a great part. This individual susceptibility is generally innate, but it may also be acquired and transitory. This modification in tolerance for drugs generally depends on changes in the kidney or renal insufficiency.

Auto-intoxications — Diathesis — Arthritism.—Auto-intoxications, especially those which depend on chronic affections of the kidneys, may also give rise to a certain number of dermatoses. When the kidneys act imperfectly, they allow waste products to accumulate in the blood, and these when eliminated by the skin may cause eruptions. These eruptions in patients affected with chronic nephritis may, to a certain extent, ward off an attack of uræmia, which soon manifests itself when the dermatoses disappear.

Abnormal fermentations, associated with disorders of digestion, especially with dilatation of the stomach, the products of which pass into the circulation, have been rightly accused as the causes of certain dermatoses.

The term *diathesis* must be reserved for another form of auto-intoxication, due to primary disorders of nutrition. The doctrine of diathesis has been the subject of much controversy; nevertheless, it should be maintained at any rate in part. In certain subjects there is a sort of constitutional condition, a general disposition of the organism, which causes these subjects to be dermopathic in the absence of any apparent external cause for the eruptions which they present. Sometimes the same type of eruption appears always on the slightest occasion; most often eczema. Sometimes eruptions of different types succeed each other from infancy to old age. Again, different eruptions may be observed in different members of the same family; urticaria in one, acne in another, eczema or psoriasis in another, and these different dermatoses originate from the same general cause. Another proof of the existence of a constitutional diathesis as a primary cause of these dermatoses, is that the cutaneous affection is seldom isolated, but belongs to a morbid series, including multiple visceral manifestations. Gout, rheumatism, asthma, pulmonary emphysema, repeated bronchitis, biliary or urinary lithiasis, early arterio-sclerosis, and dyspepsia are often observed in the dermopathic subjects themselves or in members of their family. In fact, there is a sort of substitution of one affection for another, either in the same subject or from one generation to another; for all these affections are of the same nature, and depend on the same cause.

What, then, is the nature of this constitutional state or diathesis? Is it simple, or are there several diatheses? Bazin described four diatheses: scrofula, syphilis, arthritism and herpetism. Recent discoveries have diminished the number.

The majority of scrofulides, lupus, and scrofulous gummas are

forms of tuberculosis; others, less numerous, are manifestations of late hereditary syphilis; others again, such as impetigo, formerly classed among the superficial scrofulides, are special microbial affections. The conception of scrofula has changed greatly in recent times. Nowadays we must regard scrofula as a chronic toxi-infection of microbial origin, a kind of toxinic impregnation of the organism, arising from the germination of microbes accumulated in the naso-pharynx in subjects affected with adenoid vegetations.

Syphilis is an infective disease, the microbe of which is the *Spirochæta pallida*, discovered by Schaudinn and Hoffmann. It is a disease of external origin, not a diathesis.

Bazin was wrong in separating arthritism and herpetism; the disorders of nutrition and the cutaneous manifestations are the same in arthritic as in herpetic subjects. Arthritism and herpetism arise from the same cause; but there are two types of arthritics: fat arthritics with a gouty tendency, and thin arthritics, which represent the old herpetics.

To resume, since scrofula and syphilis are separated from the group of diatheses, there only remains one diathesis, which corresponds to the old arthritism. All diathetic dermatoses arise from arthritism; they include eczema, pityriasis simplex, psoriasis, prurigo, the different forms of lichen, acne rosacea, etc.

The fundamental character of arthritism is a disturbance of nutritive changes, an impairment of nutrition, on account of which substances absorbed and assimilated undergo incomplete oxidation. But this cause of diathetic dermatoses is often modified by superadded scrofula, and the association of these two causes results in the production of hybrids, or diathetic cutaneous affections which imprint a special stamp on the soil in which they develop. For example, eczema in scrofulous subjects is especially moist, and easily becomes impetiginous; the skin of lymphatic subjects is very prone to suppuration.

Metastasis.—The conception of the diathesis as the fundamental cause of the majority of cutaneous affections leads to the discussion of the old doctrine of metastasis. By *metastasis* was formerly understood the transport of the essence of the disease from one part of the body to another; in the domain of dermatology, the replacement of a benign cutaneous manifestation of the diathesis by a visceral determination more grave and sometimes fatal. A too rapid cure of eczema in children, or its spontaneous disappearance, may produce grave metastases: bronchitis, dysenteric enteritis or pulmonary congestion, sometimes followed by death. In adults, especially in old people, the disappearance of generalised eczema may be followed by the same accidents, or by other manifestations of arthritism, such as asthma, rheumatism, etc. These metastases also

occur in other dermatoses than eczema; prurigo may alternate with bronchitis; psoriasis with rheumatism, dyspepsia, etc. What takes place in diathetic eruptions may be compared with what occurs in gout, where metastatic visceral lesions follow the sudden disappearance of the joint affections.

There are thus *autogenous morbid poisons* which provoke alternately cutaneous eruptions and internal affections. The skin is an emunctary for these morbid poisons, the dermatosis is a safeguard, and when it disappears the visceral manifestations appear.

We must now consider the nature of these morbid poisons produced in the body. We have seen that the characteristic of arthritism (according to Bouchard) is incomplete oxidation of nitrogenous matter. We know that the ultimate product of physiological combustion of nitrogenous matter is urea, a crystalline body very soluble and dialysable, and easily eliminated in the urine. It is not the same with other unassimilated, incompletely transformed substances called *extractive matters.* These include uric acid, leucine, tyrosine, creatine and creatinine, xanthine and hypoxanthine, etc., substances which are very slightly soluble, and only excreted by the kidneys in very small quantities. These substances are highly toxic and irritating. By their irritant action on the renal epithelium they may, when present in large quantities, give rise to epithelial nephritis. They must be equally irritating to the skin when they are eliminated by the cutaneous secretions, and their tendency to become eliminated by the skin increases when the kidney only offers them insufficient filtration on account of their slight solubility. The cutaneous secretions, therefore, in arthritic subjects play the part of vicarious secretions; hence the frequency of cutaneous eruptions in the gouty, and in all those in whom nutrition is impaired.

This idea of regarding the diathetic dermatoses as *autogenous toxidermias* is by no means imaginary; in fact, as I have shown, and as I have taught for a long time, in patients affected with eczema and psoriasis, there is an increase in the production of nitrogenous extractive matters eliminated in the urine, and at the same time a diminution in the nitrogenous output, which is much below the normal; this indicates insufficient elaboration of nitrogenous matter in the body.

On these grounds, I have defined *diathesis* as a *chronic auto-intoxication by nitrogenous extractive matters.*

The pathogeny of diathetic dermatoses, as I have just explained it, accounts for the gravity of metastases in diseases of the skin. When cutaneous elimination is arrested by suppression of the dermatosis, as elimination by the kidney is often insufficient, these toxic matters accumulate in the internal organs, and give rise to

visceral metastatic lesions, varying in severity according to the situation of the metastasis.

Diseases of the Nervous System.—The nervous system may affect dermatoses in several ways.

A. In some cases superficial eruptions are under the influence of the nervous system; these are the *reflex erythemas* which are symptomatic of diseases of the urethra or uterus; also the erythemas of the first dentition. The dermatosis may even be quite transient, like emotional erythema. These reflex eruptions are not due to nerve lesions, but simply to functional disorder of the nervous system. There are other cutaneous affections produced directly by nerve lesions; these are the *trophic dermatoses,* due to wounds or contusions of nerves, to neuritis, or to lesions in the gray matter of the spinal cord.

B. There are other dermatoses in which the nervous system plays only an intermediary part. In this case the nervous affection is caused by an intoxication; this is observed in zona and urticaria.

The primordial cause of zona acts through the medium of a nerve lesion, and from this point of view zona is a trophoneurosis; but the nerve lesion (neuritis) is caused either by an external injury, or by an infective agent of undetermined nature, or by an intoxication; for example, intoxication by carbonic oxide. Urticaria, again, is produced, not by a nerve lesion, but by a functional disorder of the nervous system, a vaso-motor disturbance, which is generally due to an intoxication.

There is another infective cutaneous disease, the parasite of which is well known, in which this parasite acts sometimes through the medium of the nervous system; this is *leprosy.* Besides tubercular leprosy, the cutaneous lesions of which are directly produced by the bacillus of Hansen, there is a nervous form of leprosy, called *anæsthetic* or *trophoneuritic,* in which the bacilli are situated in the nerves, and in which the cutaneous lesions are trophic in nature and result from changes in the nerves. Again, sclerodermia and symmetrical keratosis of the extremities are cutaneous diseases which appear to depend on nerve lesions, but these lesions arise from causes apart from the nervous system.

C. The nervous system also exerts a secondary influence in diathetic cutaneous affections. In eczema and psoriasis it is not uncommon to observe perfect symmetry in the lesions.

The nervous system also plays an important part in essential pruritus, in which there is no eruption at all, or only a few papules of prurigo. In this nervous pruritus, as in urticaria, the eruption, if not entirely produced by scratching, is certainly much increased, and often provoked by it; but scratching is only the result of itching,

and this is a nervous manifestation — the reaction of the nerve-endings affected by diathetic auto-intoxication.

Defects of Conformation. — All dermatoses due to external causes, and all those of diathetic and nervous origin, are acquired. There is another important group of dermatoses which are due to developmental defects in the tissues or anatomical elements, which may be called congenital. These dermatoses are rather deformities than diseases.

Etiological Classification.—In accordance with the preceding views, we can thus classify cutaneous affections, etiologically, into six principal groups:—

First Group.—*Dermatoses of external origin, non-parasitic*, caused by heat, the action of the solar rays, electric light, cold, continued pressure, or irritants of any kind.

Second Group.—*Parasitic dermatoses.*

A. Dermatoses produced by animal parasites: Scabies, Pediculosis. Eruptions due to bugs, fleas, etc.

B. Dermatoses produced by vegetable parasites: Favus, Ringworm, Erythrasma, Pityriasis versicolor.

C. Microbic dermatoses: Impetigo, Ecthyma. Certain folliculites, Oriental boil, Tropical ulcer, Perlèche, Botriomycosis, Warts, Molluscum contagiosum, Rhinoscleroma.

Malignant tumours of the skin probably belong to this class; and probably also Alopecia areata.

Tuberculosis, Sporotrichosis, Blastomycosis, Leprosy, Syphilis, Glanders, Anthrax, Actinomycosis.

Third Group.—*Toxic, medicamentous and alimentary dermatoses:* auto-intoxications (from lesions of the kidneys and abnormal digestive fermentations).

Fourth Group.—*Diathetic dermatoses* (Arthritism): Eczema, Pityriasis simplex, Seborrhœa, Psoriasis, Prurigo, Lichen, Acne and Rosacea.

Fifth Group.—*Dermatoses of nervous origin.*

Sixth Group.—*Congenital dermatoses:* Ichthyosis, Keratosis pilaris, Nævi.

This etiological classification, very different from morphological classification, which is based on the elementary lesion, is the only scientific one; but it is still very imperfect, on account of our want of knowledge as to the nature and origin of a certain number of dermatoses. For example, where can we place pityriasis rubra, the benign tumours, xanthelasma, etc.? With regard to xanthelasma, we shall discuss later on Cohnheim's theory, which attributes this affection to a developmental defect in the connective tissue. Certain forms of chronic pemphigus are infective in nature; others, especially pruriginous pemphigus (dermatitis herpetiformis) appear to be of nervous origin. Elephantiasis has a multiple pathogeny, sometimes of animal parasitic origin (filaria), sometimes of microbial nature, sometimes of nervous origin; all these causes may lead to obstruction

of the lymphatic or venous circulation. These dermatoses can only be differentiated by their morphology and evolution.

We have thus adopted a classification based as far as possible on etiology. But we are compelled to classify dermatoses of unknown nature according to the form of their elementary lesions; we are thus reduced to a system of mixed classification. Moreover, in the most recent classifications the majority of the dermatoses are classified according to their morphological aspect, only parasitic affections, recognised as such for half a century (ringworm, scabies, pediculosis), being grouped apart. If morphological classification is not very scientific, it has at least the advantage of being practical; for example, it allows the erythemas to be studied together, and thus facilitates their diagnosis.

DESCRIPTION OF DISEASES OF THE SKIN

COMMON INFLAMMATORY DERMATOSES.

ERYTHEMA.

ERYTHEMA consists of congestive spots, of a red colour, which disappears momentarily under pressure of the finger. But, by exaggeration of the cutaneous congestion, the primordial macular element may be complicated by secondary elements, papular, tubercular, vesicular or bullous, resulting either from small-celled infiltration of the papillæ, or from elevation of the epidermis by serous exudation. Thus, besides simple erythema, these are papular, tubercular and vesico-bullous erythemas. Other varieties, such as diffuse erythema, marginate erythema, and circinate erythema, are named after the form of the elementary lesion.

These morphological varieties may occur alone, or may coexist in the same subject, constituting what is called *polymorphous* or *multiform erythema.*

Pathologically, erythema is a cutaneous hyperæmia, due to the action of the nervous system on the papillary or subpapillary vascular network; it is thus an angioneurosis. The nervous reaction which provokes erythema depends on several causes, which may be divided into five groups: (1) external causes; (2) medicamentous and alimentary intoxications; (3) lesions or disorders of the nervous system; (4) infective diseases; (5) diathetic causes.

The external causes include parasitic and non-parasitic cutaneous irritations. These determine, not only erythema, but also other lesions; it is therefore preferable to include in the same description all eruptions due to external causes, whatever the form of their elementary lesion, whether this is a simple erythema or a papular, vesicular or other eruption. In the same way all forms of parasitic eruptions will be described with the parasites which give rise to

them. The medicamentous and alimentary eruptions, erythematous and others, will also be studied together, and the eruptions of nervous or trophic origin. Lastly, special chapters will be devoted to polymorphous erythema, rheumatic or idiopathic; to idiopathic scarlatiniform erythema; to the infective erythemas; and to pellagra, in which erythema is only one symptom of the disease. There still remain essential roseola and rubeola, but these diseases belong rather to general medicine than to dermatology.

We have to consider: (1) the *erythemas*, including (*a*) polymorphous erythema; (*b*) scarlatiniform erythema; (*c*) erythemas symptomatic of infective diseases; (2) *dermatoses of external origin* and *pellagra;* (3) *medicamentous and alimentary eruptions;* (4) *eruptions of nervous origin.*

Idiopathic Polymorphous Erythema (Erythema Multiforme).

Etiology. — The older authors considered that polymorphous erythema (apart from the symptomatic polymorphous erythemas which appear in the course of certain febrile diseases, or after the absorption of certain medicaments) was always rheumatic in nature.

The majority of contemporary authors regard idiopathic polymorphous erythema as an infective disease of undetermined nature, and the concomitant joint affections, which occur at the same time as the erythema, as pseudo-rheumatic manifestations. The bacteriological researches of Haushalter and others have been alleged to support this hypothesis. In two cases of erythema observed by Haushalter a micrococcus was cultivated on agar-agar from the contents of the bullæ, and also from the patient's urine. Subcutaneous injections of the culture were made in several guinea-pigs, and caused death in two cases. But attempts at culture from the patient's blood were negative, and inoculation in animals failed to produce an eruption like polymorphous erythema. These researches are, therefore, inconclusive and of little value.

On the other hand, there is now a great tendency to regard rheumatism as an infective disease, although its specific microbe has not yet been discovered (perhaps it is Achalme's bacillus). In order to reject the rheumatic nature of polymorphous erythema, it would be necessary to prove that the pathogenic agent found in the eruption is different from the pathogenic agent of rheumatism, which has not yet been done. The bacteriological researches mentioned above in no way invalidate the old theory. I therefore think that primary polymorphous erythema is generally rheumatic in nature, together with the arthritic, cardiac and pleural complications which so often accompany it.

Moreover, it is possible that the infective parasitic agent of

polymorphous erythema is not always the same, and that several infections of different nature and origin are capable of giving rise to it. Landouzy has recently upheld the tuberculous origin of certain polymorphous erythemas, and I have observed a case of generalised polymorphous erythema probably connected with tuberculosis.

Polymorphous erythema is an affection of adult age, and is more common in women. It is liable to recur, and may be periodic, recurring every year, or after a few years' interval.

Symptomatology. — The eruption is generally preceded by prodromal symptoms, such as fever, which sometimes ceases with the eruption, sometimes increases with or appears during the eruption; gastro-intestinal disturbances, diarrhœa, etc.; also symptoms depending on rheumatism, myalgia, arthralgia, bronchitis, and joint affections. The joint affections generally precede the eruption or coincide with it, more rarely they appear after the eruption.

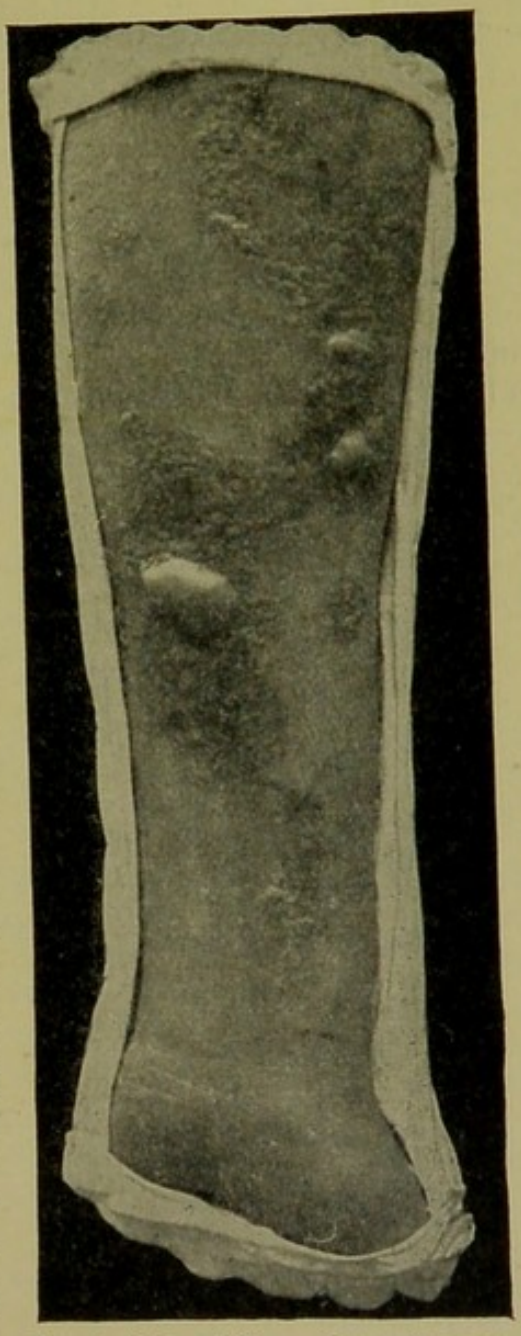


Fig. 1.—Vesico-bullous polymorphous erythema. (St Louis Hospital Museum.)

The erythema may appear in one of three forms; the first consists of erythematous red spots, sometimes covered with papules and tubercles; in the second, the epidermis of the erythematous spots is raised in the form of vesicles and bullæ; the third is characterised by the formation of nodes in the dermis, this is *erythema nodosum*. Sometimes these three forms coexist in the same subject, but usually they develop separately, preserving the same appearance during their whole duration. Erythema nodosum especially remains distinct, and some authors describe it as a special disease.

(1) **Maculo-papular Erythema.**—This form includes macular or diffuse erythemas, papular, papulo-tubercular, circinate, marginate erythemas, etc. It usually occurs on the dorsal surface of the hands,

fingers and wrists, and around the elbows and knees. It is generally accompanied by burning and itching. Sometimes there are patches of urticaria (urticarial erythema), or hæmorrhagic patches due to rupture of some capillaries (purpuric erythema), mixed with the eruption. The elements of the eruption are at first bright red, afterwards becoming violet in the centre, while the periphery remains red; they last from one or two weeks to a month, then gradually fade, leaving slight desquamation.

(2) **Maculo-vesicular Erythema.**—This is characterised by red spots, on the surface of which the epidermis is raised to form vesicles or bullæ. This form is more painful than the first. The eruption is situated around the wrists or other joints; it may also occur on the abdomen and face, and I have observed a case affecting the glans penis. This form is of somewhat longer duration than the first; the vesicles and bullæ dry up and form crusts, which after a time fall off. The eruption sometimes invades the bucco-pharyngeal mucous membrane, forming red patches, with elevation of the epithelium in the centre, on the soft palate and tongue. It may even occur on the conjunctiva. The vesicles may rupture and give place to superficial ulcerations, which cicatrise rapidly.

Hydroa—Herpes Iris—Erythema Iris.—Hydroa, which some authors have described as a distinct affection, may be included here. It is the same as Bazin's *hydroic erythema* and Bateman's *herpes iris.* Each element of hydroa consists of a small bulla situated on an erythematous spot, which forms a circle around it. The eruption shows itself chiefly on the backs of the hands and wrists, but it may affect the rest of the body and even the face. The elements are always quite separate from each other and few in number, even when the eruption affects the whole surface of the body.

Each element of hydroa develops as follows: first of all a rounded red spot appears, in the centre of which a vesicle forms. On the second day the centre of the vesicle dries up and forms a crust, while the periphery remains raised around the crust; finally, erythematous circles appear around the vesicle, of a rose-red colour, becoming paler towards the limits of the lesions, suggesting somewhat the colours of the rainbow: hence the term *herpes iris.* In other cases the eruption is less marked, and consists only of an abortive vesicle in the centre of a red spot. Sometimes the vesicle is absent altogether, constituting simple *erythema iris.* At the end of five or six days, the redness disappears and the crust falls; but the disease may last for three weeks, in the form of successive crops.

On the mucous membranes hydroa presents special features; it forms a recurring eruption affecting the tongue, lips, and the parts around the anus and genital orifices. Buccal hydroa commences in the form of an erythematous patch on which is produced a vesicle;

this ruptures and leaves an opaline ulceration, which cicatrises after seven or eight days. Hydroa of the conjunctiva, which is always accompanied by a similar eruption on the face, consists of two or

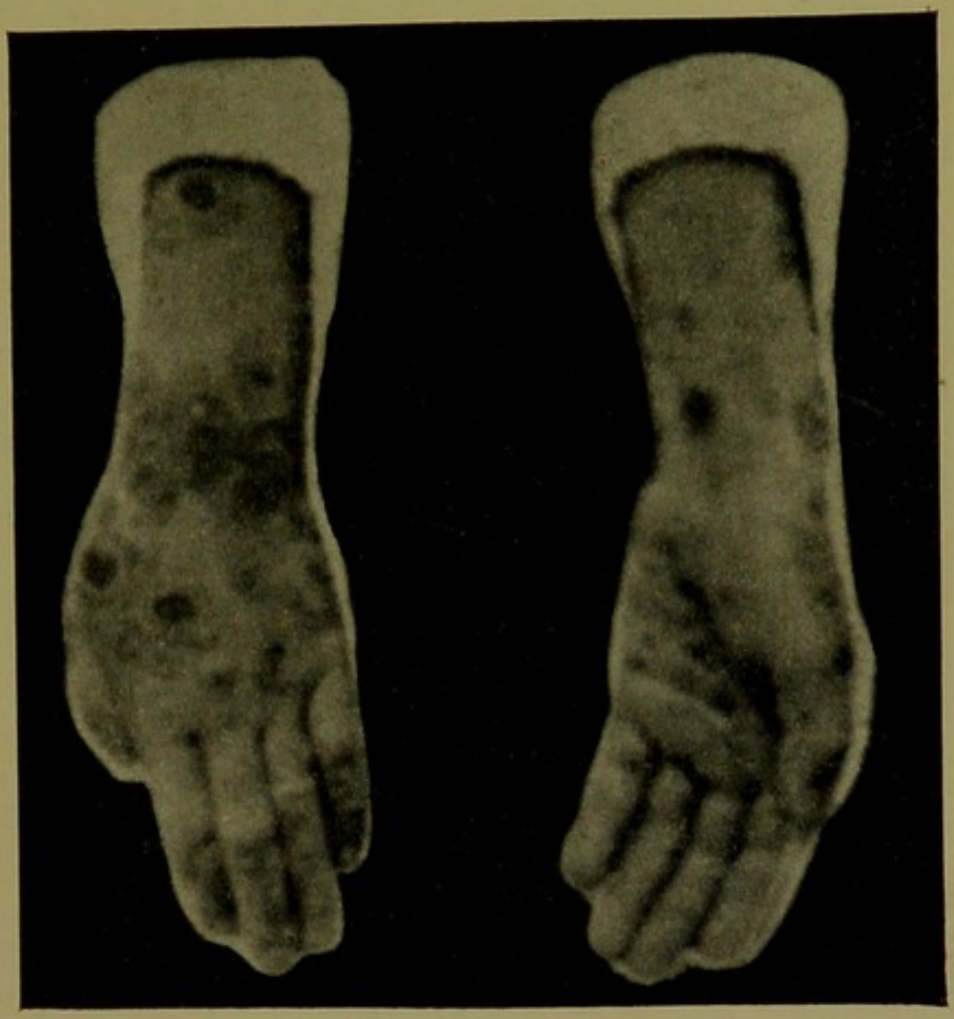


Fig. 2.—Polymorphous erythema—Hydroa.

three small isolated vesicles with a red periphery, which soon disappear.

(3) **Erythema Nodosum.**—This sometimes occurs with the preceding forms, but more usually by itself. It consists of dermic nodosities, round or oval, varying in size from a nut to a pigeon's egg. These nodosities are imbedded in the dermis and subcutaneous cellular tissue, and are painful to the touch. They are most often observed on the lower part of the legs, but may occur on all four limbs, especially round the joints. They are rare on the face. Erythema nodosum is preceded by fever, sometimes very high, and gastric disturbance, which, in children, may be mistaken for typhoid fever. The nodosities are at first hard and red, later on they become soft and livid; after eight or ten days they become absorbed, leaving a bluish hæmorrhagic spot, which changes from green to yellow like

an ecchymosis. In fact, erythema nodosum is not only congestive, but hæmorrhagic; the congestion of the capillary vessels is so great that it ruptures some of them and gives rise to infiltration of blood in the dermis. The eruption sometimes appears in several successive crops.

COMPLICATIONS.—In its three forms, polymorphous erythema may present complications which depend on the same cause as the eruption; *i.e.*, most often rheumatism. The joints may be affected with arthritis, especially the shoulder and knee; sore-throat may occur similar to that of rheumatism. Laryngitis, bronchitis, pulmonary congestion, pneumonia, and even pleurisy may also occur, and endocarditis and myocarditis are not uncommon. Cases of phlebitis, congestive lesions of the kidneys, presenting all the characters of rheumatic nephritis, cases of congestion of the liver and spleen, and adenopathics have also been reported in connection with the eruption.

The prognosis of polymorphous erythema, therefore, depends on the complications which may arise during the course of the cutaneous affection.

DIAGNOSIS.—Diffuse erythema must not be mistaken for *erysipelas*, which has a raised border and is accompanied by high fever; nor for *lymphangitis*, which presents inflammatory tracts along the lymphatics. Macular erythema is easily distinguished from *idiopathic roseola*, which affects the whole surface of the body, is characterised by small rose-coloured elements, and has a very different evolution; from *measles*, which is accompanied by ocular, nasal and bronchial catarrh. At the commencement of *variola* there is often a rash resembling diffuse erythema, but the diagnosis can be made by the concomitant symptoms.

Macular *syphilides* have a characteristic coppery colour; the macules of *leprosy* have a tawny colour, and are also anæsthetic.

Vesicular and bullous erythema must be distinguished from *acute pemphigus;* bullæ occur in polymorphous erythema, but in pemphigus they constitute the principal element.

The ulcerations secondary to hydroa of the mucous membranes differ from the *mucous patches* of syphilis in being more diffuse and more painful; on the lips, hydroa chiefly affects the middle part, while mucous patches occur most often at the commissures. *Aphthæ* are small ulcerations, round and excavated, and deeper than the ulcerations of hydroa. Lastly, in *ulcero-membranous stomatitis* there is a peculiar fœtor of the breath, with salivation and glandular swelling.

Erythema nodosum may be mistaken for *tuberculous* and *syphilitic gummas* of the skin, but the nodosities of erythema nodosum are red, while gummas are colourless at first, and only become red when they are about to open: erythema nodosum never ulcerates.

Erythema induratum, the toxi-tuberculous nature of which appears to be now demonstrated, differs from erythema nodosum by its special etiology; it only occurs in young and lymphatic subjects who are compelled to stand for prolonged periods. It also differs by the absence of fever, by its chronicity, and by the ulcerations which sometimes succeed the nodules in the skin.

TREATMENT.—This includes rest in bed and the administration of a purgative if there is gastric trouble. Alkalis may be prescribed, in the form of bicarbonate of soda or Vichy water. When the joints are affected, salicylate of soda or sulphate of quinine should be given. I am not in favour of iodide of potassium, which has been recommended for polymorphous erythema, as it may predispose to cutaneous hæmorrhages.

Locally, inert powders, such as starch, oxide of zinc and talc, may be applied. In erythema nodosum, which is especially painful, opium or chloroform liniment may be used. In the vesiculo-bullous form, a mixture of 20 parts powdered starch and 5 parts boric acid is useful, or linimentum calcis. Large bullæ may be pricked with a needle, previously passed through a flame.

Idiopathic Scarlatiniform Erythema.

Idiopathic scarlatiniform erythema must be distinguished from symptomatic scarlatiniform erythemas.

Some authors connect scarlatiniform erythema with subacute exfoliative dermatitis or pityriasis rubra, and consider it as an attenuated form of this disease; but exfoliative dermatitis, although it constitutes an erythemato-squamous dermatosis, is much more squamous than erythematous; moreover, it progresses more slowly, and has a graver prognosis than erythema.

ETIOLOGY.—Its etiology is unknown. It has been attributed to rheumatism, but this connection is not proved. Some authors attribute it to the action of cold, or to seasonal influences.

SYMPTOMATOLOGY.—Scarlatiniform erythema is usually preceded by prodromal symptoms—rise of temperature (38° to 40° C.), general malaise, vague pains in the thorax, gastric disturbance, and sometimes diarrhœa. These symptoms last for twenty-four or forty-eight hours, after which the eruption appears, in the form of red patches, at first isolated, but soon coalescing to form a diffuse eruption like that of scarlatina. The patches are situated at first in the groins, trunk, and lower limbs, then over the whole surface of the body, even in the face. The eruption is sometimes localised, sometimes generalised. Sometimes the epidermis is raised in places, forming fine vesicles which coalesce in groups and then dry up. Scarlatiniform erythema may affect the pharyngeal, nasal, and conjunctival mucous mem-

branes in the form of more or less intense redness. Desquamation occurs on the third or fourth day, becomes general on the fifth, and ends on the tenth day, except on the hands, where it lasts longer. It is furfuraceous on the face, lamellar on the trunk and limbs; but on the hands and feet it forms large strips, as in scarlatina. In some cases the hair and nails are shed, and the nails are sometimes furrowed. These facts, rare it is true, establish a gradual transition between scarlatiniform erythema and exfoliative dermatitis, in which the loss of hair and nails is constant. On the other hand, there are cases in which the disease is much more benign and of shorter duration, the eruption less intense, and the desquamation hardly appreciable. The eruption causes more or less severe itching.

Such is the regular evolution of this dermatosis. A second eruption often appears when the first is undergoing desquamation; there may even be several successive outbreaks. These relapses seem to be more common in the localised form. Recurrences are also observed which take place regularly every year or after an interval of several years; hence the name recurrent desquamative scarlatiniform erythema which is often given to this disease.

Prognosis.—In spite of the tendency to recurrence, the prognosis is benign.

Diagnosis.—The first thing to determine is whether the eruption is idiopathic, or due to the absorption of drugs, or whether it occurs during the course of some infective disease. In the case of a patient affected for the first time with scarlatiniform erythema, the question whether it is scarlatina must be decided. The two affections have many symptoms in common, but one is contagious and the other is not. In scarlatina the sore-throat is more marked and precedes the eruption, while in scarlatiniform erythema it is slight and accompanies the eruption. Again, the eruption of scarlatina does not appear in crops, like scarlatiniform erythema, and does not recur. In scarlatiniform erythema, desquamation occurs as early as the second day; in scarlatina, desquamation occurs later. *Acute eczema rubrum* differs from scarlatiniform erythema in the redness being less generalised, and in the presence of small vesicles on the red patches, which rupture easily and cause exudation.

Treatment.—This consists in the application of powders of starch, talc, oxide of zinc, or subnitrate of bismuth.

Erythemas Symptomatic of Infective Diseases.

The name *symptomatic erythema* is given to erythemas and roseolas which are observed in the course of certain infective diseases.

In *variola*, erythematous rashes sometimes occur before the

appearance of the characteristic eruption. Sometimes these rashes resemble measles (*rubeoliform*); sometimes they simulate scarlatina (*scarlatiniform*). They do not as a rule affect the whole body, but are situated chiefly in the groins, on the lower part of the abdomen, and on the flanks, towards the axillæ. Their colour is deep red, sometimes becoming hæmorrhagic. Their diagnosis is very important, because, in malignant hæmorrhagic variola, these rashes constitute the only eruption, and patients may die before the characteristic eruption appears.

In *varicella*, erythematous rashes are very rare. Sometimes they are premonitory, sometimes they follow the characteristic eruption. They are scarlatiniform erythemas, but do not desquamate.

Vaccinia sometimes gives rise to a rubeoliform erythema, which appears between the second and eighth days of the vaccinal eruption. It commences around the points of vaccination and becomes more or less generalised.

Measles is sometimes followed by polymorphous eruptions which occur from two to eight days after the disappearance of the rubeolar eruption.

Scarlatina sometimes presents, during convalescence, an eruption which has been regarded as a recurrence; but fever is often absent, and the erythema is rather morbilliform than scarlatiniform.

In *enteric fever*, the special eruption of rose spots on the abdomen may become generalised. But, in addition to this, different forms of polymorphous erythema may occur during the course of the disease. This eruption usually affects the wrists, elbows, knees and buttocks, more rarely the trunk and face. It is symmetrical and of short duration. Sometimes it recurs, especially in severe cases. Besides these benign erythemas, Hutinel has described cases of erythema followed by death in two or three days; the eruption being preceded by aphthæ and fissures on the tongue and lips. These cases were accompanied by prostration and vomiting, and the temperature rose to 40° C. before death. Le Gendre has reported a fatal case of desquamative scarlatiniform erythema which occurred during convalescence from enteric fever.

In *cholera*, at the period of reaction, erythemas occur which are of good omen, contrary to those of other infective diseases, which aggravate the prognosis. Sometimes they resemble measles, sometimes they consist of flat papules, in other cases they form scarlatiniform patches situated chiefly on the limbs.

Pyæmia and *septicæmia* may give rise to all forms of erythema. One of the most common is a diffuse erythema, or one occurring in large patches, which may occur in all kinds of suppuration. In puerperal infection scarlatiniform erythema occurs. Other forms

of erythema observed are morbilliform and papular, and erythema nodosum.

The same erythemas are also seen in *ulcerative endocarditis.*

In *epidemic cerebro-spinal meningitis* an erythema sometimes occurs which affects the form of roseola. In *diphtheria* a diffuse or rubeoliform erythema is sometimes met with which has a tendency to become hæmorrhagic. This is always of grave prognosis, especially when it appears at a late stage in the disease.

Streptococcal angina may also give rise to erythemas, which, in themselves, are always of benign prognosis.

Gonorrhœa, apart from cases in which patients have taken copaiba, may sometimes produce polymorphous erythema, macular, papular or scarlatiniform.

The *syphilitic roseola* usually presents itself in the form of a macular roseola, about six weeks after the chancre; but it may recur in the form of recurrent roseola, which often assumes an annular form. Syphilitic roseola of the neck is the origin of the *pigmentary syphilide* of the neck. Macular roseola gives rise to the macular pigmentary syphilide; annular roseola to the areolar pigmentary syphilide. This pigmentary syphilide is therefore always secondary, and not primary, as many authors have maintained. Mauriac has described a syphilitic erythema nodosum as an early manifestation of syphilis, but this is probably only a coincidence; a case of simple erythema nodosum occurring during syphilitis.

It is probable that the majority of the erythemas which sometimes occur in the course of measles, scarlatina, enteric fever and diphtheria, are not directly due to these diseases, but to a secondary infection which aggravates the disease. Hutinel found that all children affected with erythema presented fissures of the lips, and buccal or pharyngeal ulcerations, in which he found streptococci. These erythemas may therefore be of toxic origin, and caused by the absorption of streptococcal toxins.

ERUPTIONS OF EXTERNAL ORIGIN.

Eruptions of external origin are due to irritation of the skin produced by external agents, such as radiant heat, sunlight, electricity, cold, continual pressure and irritating substances.

Erythema ab Igne.—This is caused by excessive radiant heat. It shows itself as a diffuse redness with swelling and smarting of the skin, followed by desquamation. It occurs especially in workmen who are exposed to intense heat, such as glassblowers, blacksmiths, smelters and stokers.

Under the prolonged action of heat the affection becomes chronic, and then presents the following appearance: the skin is reddish brown, thickened, hard and wrinkled, with characteristic marbling, affecting the parts exposed to heat—the face, hands and forearms.

Erythema Solare.—This presents the same aspect as the above: redness and smarting, and sometimes desquamation after a few days. In severe cases blisters may be produced. Solar erythema is caused by the chemical rays of the solar spectrum—the violet and ultra-violet rays. This is why it is more common in the spring and at sunrise, because the sunlight at these times is richer in chemical rays. In sailors, under the action of sun and salt air, diffuse pigmentary spots are formed on the face.

The *electric light* may also cause a similar erythema. The most important of these electric erythemas is that produced by the X-rays, and which is known by the name of *radio-dermatitis.* This lesion is often observed in patients who have been exposed to the action of the X-rays. It varies from simple reddening of the skin to more or less extensive scarring. Sometimes it does not appear till after a considerable interval of time.

Pellagroid Erythema.—This will be described later, in the article on Pellagra.

In all these eruptions, *treatment* consists in the application of emollient lotions or powders.

Hydroa Vacciniforme.—Some persons when exposed to the sun's rays, especially in spring or summer, are subject to an eruption of umbilicated vesico-pustules. This eruption was described by Bazin in 1862, and by Hutchinson in 1888, under the name of "summer eruption"; more recently cases have been published by Bowen and Brocq. The affection occurs especially in children, and may recur several times during the summer, disappearing in adult age. Each eruption lasts from one to several weeks. It affects the face, ears, nape of the neck, dorsal surface of the hands, and also the feet when these are exposed. It is characterised by opaline vesicles situated on a red base. Some vesicles rupture and dry up, others become umbilicated, simulating the pustules of variola; after six to fifteen days the crusts fall, leaving a varioliform cicatrix which is permanent. Treatment is chiefly prophylactic, and consists in protection of the face by ointments, and the use of red or yellow veils.

Erythema pernio, or Chilblain.—Like heat, cold may also cause erythema, which is characterised by redness of the skin with swelling of the dermis and sometimes the subcutaneous cellular tissue. This erythema gives rise to severe smarting, especially when the patient passes from cold into warmth. It affects chiefly the fingers and toes, ears, heels, cheeks, and tip of the nose; in fact all regions where the circulation is less active. Chilblains may be complicated by bullæ,

fissures, and painful ulcerations, which are difficult to heal. Simple erythema pernio terminates by desquamation. In lymphatic subjects it may become chronic, or at any rate recur every winter.

TREATMENT.—Baths prepared with decoction of walnut leaves or lotions of ammonium chlorhydrate (1 in 10) are useful. At night the following ointment may be applied: glycerole of starch 50 grammes, tannin 50 centigrammes, covered with a simple powder. Another useful ointment consists of equal parts of white vaseline, anhydrous lanoline, oxygenated water and oxide of zinc. Ulcerated chilblains should be dressed with boric vaseline. In lymphatic subjects tonic treatment is indicated—cod-liver oil, iron, iodo-tannic syrup.

Erythema Paratrimma.—This occurs in patients who are confined to bed for long periods (paralysis, enteric fever, etc.), and is aggravated by the irritating action of the fæces. It affects the buttocks and sacral region, and often leads to ulceration (bed-sores).

Eruptions due to Animal Irritants.—Erythema may be produced by contact with certain animals (*vide* Animal Parasites). We need only mention here the special eruption observed among silk-winders, which is characterised by vesicles situated on the fingers, generally between the thumb and index finger.

Erythema Intertrigo.—The irritation caused by the cutaneous secretions in parts where skin comes in contact with skin, gives rise to *erythema intertrigo.* This is especially observed in the inguinal, intergluteal, cruro-scrotal and cruro-vulvar folds, in the axillæ, and under the breasts of fat women, especially when the cutaneous secretions are allowed to accumulate. Intertrigo is characterised at first by redness, most marked at the bottom of the folds of skin, less marked at the borders of the erythematous surface; changes in the cutaneous secretions produce a special odour; the epidermis is sometimes macerated, and fissures and excoriations may form, accompanied by a sero-purulent discharge. In *diabetic subjects* intertrigo presents special features: it affects chiefly the prepuce, glans penis and labia majora, parts which come in contact with the sugar-containing urine. Besides erythema, there is also an eczematiform eruption, which becomes covered with crusts, due to the action of the irritating urine, and aggravated in diabetes by the bad nutrition of the skin.

In the macerated epidermis of skin affected with intertrigo, microbes are naturally found, but the part played by these has been exaggerated.

Erythema Neonatorum.—Like intertrigo, this erythema is of external origin, and is due to irritation of the skin by urine and fæces, aggravated by want of cleanliness, defective nutrition of the skin, and bad general condition. Most commonly situated on the buttocks and thighs, the erythema may extend to the legs and even

to the feet; it consists in bright redness of the skin, followed by desquamation, sometimes by excoriations and ulcerations. Sevestre and Jacquet have described a special variety of infantile erythema, which has often been mistaken for one of the manifestations of congenital syphilis. This occurs in two forms, *vesicular* and *lenticular*, and affects the buttocks, perineum, and genital organs, and may extend to the thighs and legs. The *vesicular* form begins by the formation of small vesicles which dry up and leave red spots, isolated, or forming confluent patches; these spots may become excoriated or even ulcerated. The *lenticular* form, which follows the preceding, is characterised by smooth, lenticular papules with a depressed centre, slightly exudative, reddish brown or violet in colour; between the papules are found vesicles which have not yet undergone transformation into papules, brownish spots, the remains of flattened papules, erosions and even ulcerations, more or less irregular by confluence of the constituent elements. This form simulates in a striking manner the papular or papulo-erosive syphilides, and for this reason Sevestre and Jacquet have given it the name "*syphiloide post-erosive.*"

Sometimes this erythema resembles the eruption of vaccinia, and this form has received the name of *herpes vacciniforme.*

Diagnosis.—Erythemas of the newly born are often mistaken for congenital syphilis. They differ from syphilides by being usually confined to the parts around the anus and genital organs, by the absence of other signs of congenital syphilis, and by yielding to simple treatment in ten to fifteen days.

The *treatment of intertrigo* consists in absolute cleanliness. As soon as the inflammation has subsided, the parts should be treated with lotions of saturated boric acid or 1 per cent. resorcin, and then powdered with powdered talc containing 1 per cent. salicylic acid. The cutaneous surfaces should be separated by fine linen. If there is much itching, a little camphor may be added to the powder. The same treatment applies to erythemas of the newly born.

Eruptions due to Vegetable Irritants.—Besides the vegetable parasites, which will be described in special articles, there are numerous vegetables which produce erythemas. Bitter oranges may give rise to erythema, and even vesicles and pustules, on the hands of the workers who peel them. Flax may cause an eczematiform eruption, etc.

Eruptions due to Chemical Irritants.—All chemical substances obtained from the mineral or vegetable kingdoms may give rise to eruptions. Tincture of arnica, when applied in a pure or concentrated form, causes erythema, and even papules and vesicles. Quinine produces erythema, vesicles and pustules in the workmen

who prepare it. The eruption caused by thapsia is well known; on the face it may simulate erysipelas. The application of mercurial ointments and strong solutions of perchloride of mercury often cause erythema and sometimes eczematiform eruptions; the same with ointments of pyrogallic, salicylic and chrysophanic acids. These erythemas may be simple, scarlatiniform, or similar to exfoliative dermatitis; sometimes they are vesiculo-bullous or pustular.

The application of iodoform in powder or as an ointment may give rise to a localised red eruption with the production of vesicles; this sometimes becomes generalised.

Arsenic, among the workmen engaged in the manufacture of arsenical colours, causes erythema, vesicles, pustules and ulcerations on the face, especially about the commissures of the lips; on the nasal septum, where they often cause necrosis; on the hands, and on the scrotum, where they may be mistaken for mucous patches. But, apart from simple erythema, all the other cutaneous lesions are rather trophic disorders caused by chronic arsenical poisoning, than lesions produced by the external action of arsenical colours.

Lastly, eruptions may be caused by turpentine, oil of cade, tar, petroleum, impure soap, sugar, carbolic acid, salol, bichromate of potash, aniline dyes, etc. Formol, which is used to disinfect vanilla damaged by mould, gives rise to an eruption which has been wrongly attributed to vanilla. Salol, especially, is very irritating to the skin, and should never be used as an external application.

Professional Eruptions.—These occur in bakers, grocers, masons, bricklayers, dyers, druggists, manufacturers of coloured paper, tanners, etc. But these are most often cases of eczema, or some other dermatosis of diathetic origin, occurring in subjects who are predisposed, on account of repeated irritation of the skin.

In all artificial eruptions the main point is to suppress the primary cause of the eruption; after which we can prescribe emollient lotions, starch baths, and sedative powders. Ointments should be avoided, as they often increase the irritation.

PELLAGRA AND PELLAGROID ERYTHEMA.

Pellagra is a general disease, chronic and subject to vernal exacerbations, characterised by disorders of the digestive and nervous systems, and leading, under the influence of insolation, to erythema, affecting the parts directly exposed to the sun's rays.

Pellagra may be endemic or sporadic. *Endemic pellagra* occurs chiefly in Italy, and in some parts of Spain and Austria. It was formerly observed in the southern parts of France, but, owing to the

progress of hygiene, no longer occurs endemically. *Sporadic pellagra* has been observed in Vienna, Westphalia and Greece, and in Paris and other parts of France.

There are two special forms of pellagra—pellagra of the insane, and pellagra of alcoholics—which have been regarded by some authors as "pellagroid erythemas" developed under the influence of physical or mental decadence in the insane, and in alcoholics. Disorders of digestion and nutrition are common in the insane, and these prepare a soil which is favourable for the production of pellagroid erythema under the influence of the sun's rays. Alcohol, owing to its harmful action on the nutrition of the tissues, predisposes to pellagra.

SYMPTOMATOLOGY.—Pellagra is a chronic disease, which usually passes through three stages. At first intermittent, it appears in the spring and ceases in the winter; then it becomes remittent. The symptoms persist during the winter, and become exacerbated in the spring; finally, the disease becomes chronic and ends fatally.

The *prodromal* symptoms consist in lassitude and prostration, anorexia, or sometimes boulimia, with nausea, vomiting, and diarrhœa; sometimes headache and vertigo. These symptoms increase till the erythema appears in the spring. At its period of acme the disease presents three groups of symptoms—cutaneous, digestive, and nervous.

Cutaneous symptoms.—The characteristic erythema is dark red, sometimes erysipeloid, and is accompanied by burning pain. It affects the dorsal surface of the hands and first phalanges, the face and neck, the posterior surface of the forearm, the upper part of the chest and dorsal surface of the feet (when these parts are exposed); in fact, all parts of the body exposed to the sun's rays, the erythema ceasing where the skin is covered by the clothes. Sometimes vesicles or bullæ develop, giving rise to gray or blackish crusts. The eruption generally lasts till May, when the red colour fades, the skin desquamates, and resumes its normal appearance in the winter. At its onset, pellagroid erythema is thus intermittent and annual. When it has recurred for some years, the skin becomes rough, dry, brown, and somewhat atrophied; finally, the epidermis constantly desquamates in the form of gray powder.

Some authors consider the cutaneous lesion as an erythema of internal origin; others hold that it is chiefly due to the action of the sun's rays. The latter view is held by Bouchard, who puts forward the following arguments in its favour: the situation of the lesion, its appearance in the spring, the possibility of avoiding the eruption by covering the face and hands, etc., prove that pellagroid erythema is nothing more than a solar erythema developed in predisposed subjects. It may be absent in persons affected with pellagra when,

by change of occupation, they are no longer exposed to the sun. Bouchard and others have shown that the erythema is caused by the violet and ultra-violet rays of the spectrum; hence its appearance in the spring when these rays are present in greatest quantity. Pellagra, by producing defective nutrition, renders the skin more susceptible to the action of the sun's rays, causing it to lose the power of absorbing the chemical rays (fluorescence), a property possessed in a high degree by the skin of negroes.

Digestive symptoms.—These are not actually intermittent like the erythema, but they undergo exacerbation in the spring and become attenuated in the winter. The lips become dry, livid and covered with black crusts. The buccal mucous membrane is swollen and inflamed, and presents aphthous vesicles and ulcers. The gums may be fungous and bleeding. The inflammation sometimes spreads to the pharynx and œsophagus. The tongue is smooth, red, and denuded of epithelium, or covered with aphthous ulcers; when death is imminent it becomes dry and black. Gastric symptoms consist in gastralgia, dyspepsia, nausea, and pyrosis, sometimes anorexia, but more often boulimia. The pyrosis and buccal inflammation explain the constant thirst of patients affected with pellagra. Diarrhœa is more common than constipation, and is sometimes hæmorrhagic.

Nervous symptoms.—These are intermittent at first, afterwards continuous. They include sensory, motor and mental disorders. *Sensory disorders* include frontal headache, sometimes unilateral, neuralgia, and rachialgia, burning sensations in the palms and soles, formication, and more rarely anæsthesia or analgesia. Disorders of special sensation include perversion of taste, tinnitus, and deafness, flashes of light, muscæ volitantes, hemeralopia, diplopia, amaurosis or complete blindness. The pupils are often unequal, and there are often retinal lesions. *Motor disorders* are nearly always preceded by vertigo. Incoördination of movement precedes paresis of the lower limbs and eventual paralysis; the upper limbs are affected afterwards. If mental troubles are superadded, pellagra then simulates general paralysis. Other motor disorders have sometimes been observed, such as cramps in the muscles of the foot, calf, hand or spine; trismus, tetanoid contractures, epileptiform convulsions, choreiform movements and tremors. *Mental disorders* usually occur at an advanced stage of the disease. At first there is mental depression and diminution in intelligence; later on there may be acute mania, suicidal or homicidal. Suicide is generally by drowning, probably on account of the intense thirst. Dementia, simulating paralytic dementia, is more common than mania.

Complications.—These may affect the respiratory or circulatory systems. Respiratory complications include catarrhal laryngitis or chronic bronchitis, hypostatic pneumonia in the later stages, pleuritic

effusion, and sometimes pulmonary tuberculosis. Circulatory complications include anæmia, diminution in the number of red corpuscles, hæmorrhage from the mucous membranes, or in the skin and subcutaneous tissue. Albuminuria, accompanied by anasarca, retention of urine and cystitis, and gangrene of the leg and scrotum have also been observed.

PROGNOSIS.—Pellagra is generally fatal, but may be cured in young subjects if taken in time. Death may be caused by cachexia, complications or suicide. The duration of the disease is very variable; it may only last three months, or may be prolonged for ten or twenty years or longer. When the patient leaves his country there may be remission, but recurrence when he returns.

ETIOLOGY.—The principal cause of pellagra lies in alimentation. The disease has been attributed to the ingestion of maize affected by a parasitic fungus (*Sporisorium maidis*), which causes the disease of maize called *verdet*. This diseased maize produces an intoxication analogous to that produced by ergot of rye (ergotism). Balardini found that fowls fed on diseased maize suffered from wasting, thirst and tremors, and become cachectic in twenty-eight days. He concluded that the diseased grain contained deleterious substances capable of producing harmful effects in man. These substances have been considered as the cause of the digestive and nervous symptoms of pellagra, for, according to Lombroso and others, ingestion of the *Sporisorium* itself is harmless.

These researches show that diseased maize gives rise to profound changes in nutrition; but these changes are not due solely to the diseased maize. Pellagra is unknown in countries where maize is largely consumed; for instance, in America. Again, pellagra occurs where no maize is consumed; most cases of sporadic pellagra occurring in the absence of any consumption of maize.

In fact, pellagra, whether it occurs among consumers of maize or among persons who never eat maize, is probably caused by defective alimentation, which leads at first to digestive troubles, and later on to nervous disorders, by the absorption of noxious products of a toxic nature.

Pellagra affects both sexes; it is more common in adults and children than in the aged. It is neither contagious nor hereditary, although it is often observed in individuals of the same family, and although those affected may procreate children who are predisposed to contract it. Cases which appear to support heredity can nearly all be explained by the influence of diet and mode of life, which are the same for parents and children.

According to Cuboni and Majocchi, pellagra is of parasitic origin. The latter observer has described a bacillary schizomycete in diseased maize, and claims to have found the same organism in the

brain and viscera in two cases which died of pellagra; but this requires confirmation.[1]

DIAGNOSIS.—This is always easy in countries where pellagra is endemic, but not where it occurs sporadically. The diagnosis is based on the association of erythema, or the lesions which succeed it, with digestive and nervous symptoms; on the season when it appears; and on its special localisation on the hands and face. Pellagroid erythema must not be confused with simple *solar erythema* (sunburn), which occurs in healthy subjects. It is easily distinguished from *eczema* affecting the backs of the hands, which is seldom so limited, and is accompanied by itching and exudation.

Acrodynia—a disease which is not now seen, but which was common in the first half of the latter century—has sometimes been confused with pellagra, but in acrodynia the erythema usually affects the palms and soles, rarely the dorsal surface, and is accompanied by œdema, pain and contracture of the extremities. Pellagra must not be mistaken for convulsive and gangrenous *ergotism*. Lastly, the typhoid symptoms which sometimes occur in pellagra must not be mistaken for typhoid fever.

Alcoholic pellagra is recognised by the concomitant symptoms of alcoholism, occurring in subjects who have not otherwise suffered from misery. In pellagra of the insane, the patients are first insane and afterwards pellagrous.

PATHOLOGICAL ANATOMY.—The post-mortem lesions are very varied and not characteristic. According to Lombroso, they consist in atrophy and fatty degeneration, sometimes in hyperæmia and pigmentation.

The principal lesions observed have been: injection and ulceration of the mucous membrane of the stomach and intestines; fatty degeneration of the liver; atrophy of the spleen; interstitial nephritis; bronchitis, hepatisation or tuberculosis of the lungs, anæmia or congestion of the nerve centres, and meningitis. According to Golgi, in half the cases microscopic examination shows pigmentation in the adventitia of the cerebral capillaries, in the nerve cells, and in the sympathetic and spinal ganglia, with increase of the amyloid bodies in the cerebral and spinal connective tissue. Bouchard found lesions in the posterior columns of the spinal cord, and considered them identical with tabes. Marie points out that there are considerable differences in the spinal cord in tabes and pellagra: in tabes, there are changes in the cornu-radicular zone, but integrity of the internal posterior zone; in pellagra, there is integrity of the cornu-radicular zone, but changes in the internal posterior zone. I have myself observed a case in which there were changes in the cord and meninges almost identical with those described by Marie.

[1] Sambon suggests that pellagra is of protozoal origin.—ED.

In the skin, sclerosis of the papillary and dermic vessels and atrophy of the horny layer have been described. In a piece of skin taken from a patient in the cachectic stage, Raymond found diminution in thickness of the epidermis, atrophy of the papillæ, with desquamation and hyperkeratinisation of the horny layer; the cutaneous glands and nerves were normal. This integrity of the cutaneous nerves is apparently in contradiction with the nerve lesions found by Dejerine in a case of alcoholic pellagra; but these lesions were due to alcoholic neuritis.

TREATMENT.—This consists in nourishing diet, tonics such as quinine and iron, sulphur or salt-water baths. The parts exposed to the sun should be protected. Locally, sedative powders and lotions are indicated. Lombroso recommends arsenic internally; but this has probably nothing more than a tonic action. In fact, the treatment and prophylaxis of pellagra consist in substantial alimentation and good hygiene.

MEDICAMENTOUS AND ALIMENTARY ERUPTIONS.

Medicamentous Eruptions.—These are sometimes simple scarlatiniform or rubeoliform erythemas, sometimes vesicular or bullous erythemas, sometimes urticarial or hæmorrhagic. They are, therefore, polymorphous erythemas, like idiopathic or rheumatic erythema.

As regards their *pathogeny*, there are two points to be considered: first of all the medicament itself; secondly, individual predisposition, which is so strong in certain persons that they cannot take the smallest dose of certain drugs without an eruption. This predisposition is often dependent on an unknown cause, but sometimes it is due to insufficient renal permeability. It is, therefore, important, before administering a drug, to test the patient's urine for albumen, and estimate the output of urea. We will now enumerate the principal drugs, with the different eruptions which they produce.

Salicylic acid and *salicylate of soda* cause diffuse, rubeoliform or scarlatiniform erythemas.

Antipyrin, and all the preparations which contain it, may give rise to patches of erythema affecting chiefly the limbs, or to rubeoliform or scarlatiniform erythemas. These erythemas soon terminate by desquamation. Antipyrin may also give rise to *bullæ*, which usually appear on an antecedent spot of erythema; they soon rupture and dry up. Bullous antipyrin eruptions may also appear on the mucous membranes; they have often been observed in the mouth and about the anus.

Antipyrin may also produce black spots on the skin. These

commence as erythematous spots, which gradually become gray and then black. They sometimes occur on the genital organs. A peculiar characteristic of this black antipyrin eruption is the frequency of recurrence *in situ* after fresh doses of the drug.

Nitrate of silver, after prolonged administration, gives a grayish blue coloration to the skin, due to the deposit of silver in the papillary layer of the dermis.

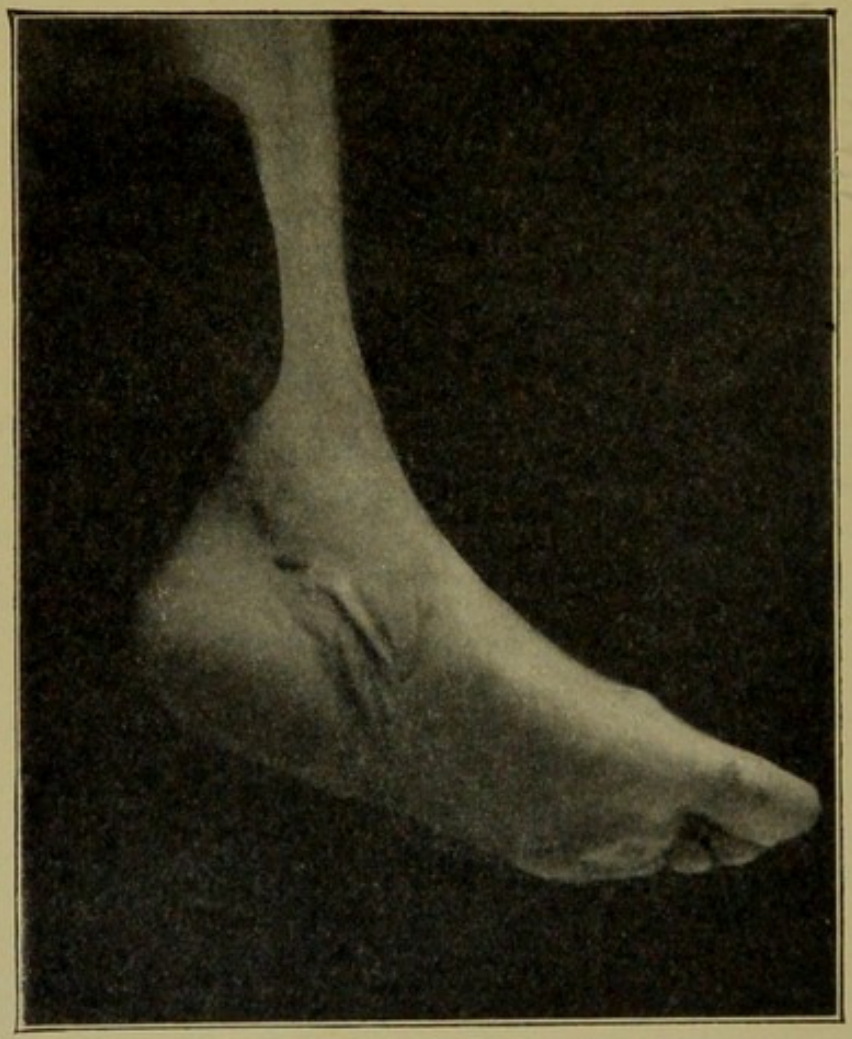


Fig. 3.—Bullous eruption of the foot due to antipyrin.

Arsenic may produce erythemas of all kinds, papular, vesicular (zoniform eruption), bullous or even pustular eruptions, but these are not common. A special form of arsenical dermatosis consists in yellowish or bistre-coloured spots affecting the whole surface of the body, but most marked on parts exposed to repeated friction, especially the neck and round the waist. Chronic poisoning by arsenic may produce keratodermia, affecting the palms of the hands and soles of the feet, and ulcerations on the scrotum and in the interdigital spaces.

Belladonna, aqua laurocerasi, and *datura stramonium* may give rise to simple or scarlatiniform erythemas.

Bromides cause acneiform eruptions resembling ordinary acne; also simple or papular erythemas, which may be complicated by vesicles or pustules. *Bromoform* may also cause an erythematous eruption. Sometimes, especially in young children, bromides give rise to large, reddish tubercles, which simulate hypertrophic mucous patches, and sometimes last a long time. Bromide eruptions are among the most severe of the toxidermias. Tolerance for bromides

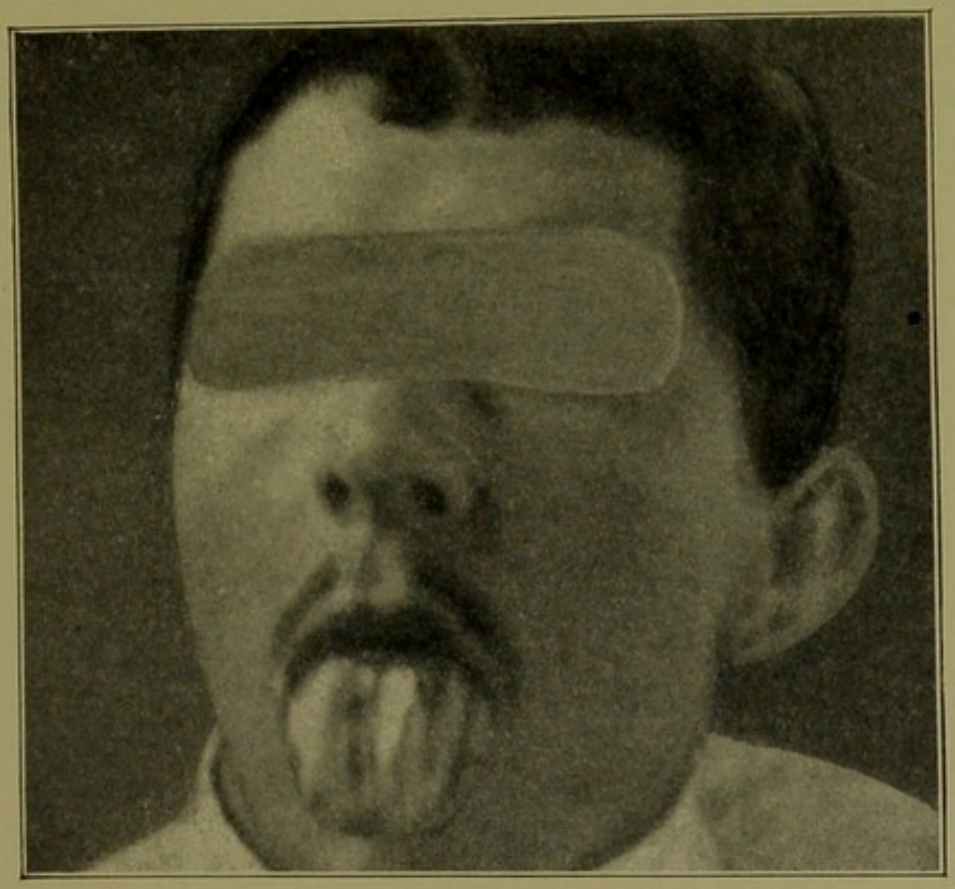


FIG. 4.—Bullous eruption of the tongue and lips due to antipyrin.

can sometimes be attained by practising intestinal antisepsis at the same time.

Chloral produces an erythema, occurring in large red patches on the face or limbs, seldom generalised, sometimes hæmorrhagic.

Quinine and *cinchonine* occasionally give rise to a roseola, affecting chiefly the face and neck.

Copaiba, cubebs, santal, and *turpentine* cause erythemas, sometimes localised, sometimes generalised. The first form, characterised by red, pruriginous, raised patches, situated about the wrist joints, is a benign eruption which disappears in the course of three or four days. In the generalised form the patches are confluent and occupy nearly

the whole surface of the body, even the face, which is red and œdematous. All varieties of polymorphous erythema, from red spots to papular, bullous, urticarial or hæmorrhagic erythemas, have been observed after the administration of balsams. Generalised balsamic erythema may affect the mucous membrane of the pharynx and larynx, and in the latter case may cause œdema of the glottis with considerable obstruction to respiration.

Iodide of potassium and other *alkaline iodides* may produce erythemas, which are often accompanied by other symptoms of iodic intoxication—coryza, lachrymation and sore-throat. All kinds of eruptions have been observed, singly or combined with one another, but the most common are iodic acne and purpura. *Iodic acne* is characterised by pustules situated on the face and neck, due to inflammation of the sebaceous glands, by which the drug is eliminated. *Iodic purpura*, which is of a benign character, chiefly affects the lower limbs; in some cases it may be caused to disappear or be prevented by administering tannin at the same time, in the form of extract of rhatany. Red and painful indurations, somewhat resembling those of mycosis fungoides, have also been reported as the result of iodic intoxication; these sometimes suppurate in one or more places and then form vegetations like those of pemphigus vegetans. Bullous eruptions have also been observed.

Mercury and its salts cause a group of eruptions to which the general name of *hydrargyria* has been given. Idiosyncrasy plays a great part here. This idiosyncrasy, the nature of which is unknown and which occurs without renal lesions, affects certain persons whether mercury is administered internally or applied externally. This susceptibility, however, must not be regarded as contra-indicating mercurial treatment; but, bearing in mind that mercury is not well tolerated, and that small doses may produce the same effects in such persons as large doses in others, it should be given in small doses at first, and then gradually increased.

Three forms of mercurial eruption have been described: (1) a benign form; (2) a febrile form; (3) a malignant form. The *benign form*, which is the most common, is an erythema affecting the lower part of the abdomen, the scrotum and the inner surface of the thighs. It is sometimes complicated by the formation of vesicles, constituting what has been called "mercurial eczema." This eruption causes severe itching, but is not accompanied by fever, and disappears after slight desquamation. The *febrile form* follows the preceding, in cases where mercurial treatment has been continued in spite of the appearance of erythema; but it may occur independently. It is accompanied by fever and gastric disturbance, and may present two varieties of eruption; one localised, the other generalised. The former consists of red spots which become covered with vesicles or

pustules, producing yellow crusts like those of impetiginous eczema. The second form is a scarlatiniform erythema affecting the whole surface of the body, even the face, which becomes swollen; sometimes vesicles and pustules develop on the erythema. The eruption may affect the mucous membrane of the mouth and pharynx. It is followed by abundant desquamation, especially on the hands and feet, and much resembles exfoliative dermatitis. The *malignant form* is rare, and is only an exaggerated form of the preceding. It is due to the administration of mercury in immoderate doses. The eruption is livid red, and covered with vesicles and bullæ which rapidly become purulent and sanious. It is accompanied by mercurial stomatitis, which has a tendency to become gangrenous; the general condition is very bad, the temperature may rise to 40° C., and the patient generally succumbs to mercurial intoxication.

The preparations of *opium* and *morphine* may give rise to simple, papular or scarlatiniform erythemas.

Ergot may produce erythematous patches, followed by rapid gangrene of the skin.

Alimentary Eruptions.—Salt-water fish and all fish which have undergone decomposition, shellfish, crustacea, pork, high game, old cheese, coffee, tea, and alcohol may give rise to outbreaks of erythema (sometimes scarlatiniform), acne, or urticaria (vide *Urticaria*). According to Juhel-Rénoy, tainted pork may give rise to an eruption which may be roseolar, scarlatiniform, urticarial, or polymorphous; but this has been disputed.

The *diagnosis* of toxidermias is very easy when a history of the case is available, otherwise it is difficult. In the case of polymorphous or scarlatiniform eruptions of doubtful nature, enquiry should always be made whether the patient has been taking drugs, or ingested some noxious food.

Treatment consists in removing the cause, and then prescribing milk diet, laxatives and diuretics. Dry eruptions should be treated with emollient applications, moist eruptions with simple powders.

ERUPTIONS OF NERVOUS ORIGIN—CUTANEOUS TROPHONEUROSES.

These eruptions vary from simple vaso-motor disturbance, producing transient erythema, to trophic disorders of the skin.

We can distinguish three kinds of erythema of nervous origin: (1) transient erythemas; (2) reflex erythemas of visceral origin; (3) trophic erythemas.

Transient erythemas are those which appear on the neck and

chest of patients, especially women, when they undress for examination; they soon disappear.

Reflex erythemas, said by some authors to be symptomatic of urethral or uterine lesions, are more lasting than the preceding, and are sometimes polymorphous. *Trophic erythemas* occur after wounds of nerves, in neuritis, and also in affections of the posterior system of the spinal cord (tabes and syringomyelia). They are characterised by a uniform, shiny redness (glossy skin), sometimes bright red, sometimes pale red or violet. The eruption is accompanied by smarting pain, and chiefly affects the extremities. It is followed by desquamation, with thinning of the skin, or may be complicated by fissures and ulcerations.

In other cases there is formation of bullæ and vesicles; this is sometimes observed in locomotor ataxy and in other affections of the posterior or sensory system of the spinal cord. These vesicles may assume the form of zona; the bullæ resemble those of pemphigus (see Zona, Pemphigus and Pemphigoid Eruptions).

The frequently repeated action of the X-rays on the fingers of operators who practice daily radioscopy and radiography, may produce an *ascending neuritis*, which manifests itself after several months by the appearance of cutaneous trophic disorders, similar to those produced by wounds of the nerves:—Thin and glossy skin, telangiectases, ulcerations and fissures, and often papillomas, which may become epitheliomatous. I have seen three cases of this kind.

This *radioneuritis*, peculiar to *operators*, is produced by the repeated action of the X-rays, and the lesions which it causes must be distinguished from *radiodermatitis*, which occurs in *patients* who have been exposed to too prolonged radiotherapy, and whose skin has absorbed too large a quantity of rays.

In some cases, as the result of changes in the peripheral nerves, erythemato-pustular lesions with superficial scars are observed, affecting only the anæsthetic areas. These lesions are improved by the application of occlusive dressings, which seems to show that the nerve lesions produce changes in the skin which render it more vulnerable and predisposed to microbic invasion.

The nerve lesions may be followed by spontaneous ulcerations, which spread slowly and serpiginously, and are nearly always accompanied by intense neuralgia pains and anæsthesia; these correspond to the area of distribution of certain nerves. *Perforating ulcer* occurs most often in tabes; according to Duplay and Morat, it is due to a degenerative neuritis. *Gangrene* may occur as the result of nerve lesions; either spontaneously or after slight injuries, especially in hysterical subjects. The gangrene is preceded by vaso-motor phenomena; sometimes a simple erythema, sometimes an

urticarial erythema, sometimes a bullous eruption. The gangrene may be superficial or deep, affecting nearly the whole of the dermis; the wound left after the slough has separated heals very slowly. The affection is of long duration, and occurs in successive outbreaks.

Symmetrical gangrene of the extremities (*Raynaud's disease*) is due to disease of the nervous system.

In sclerodermia, anæsthetic leprosy, palmar and plantar keratosis, and in zona, the nervous system only plays an intermediary part; these diseases are not properly speaking nervous affections. The same with vitiligo, which has always a nervous pathogeny, but in which the cutaneous nerve changes are generally secondary (see *Vitiligo*).

The nails are often shed as the result of lesions of the peripheral or central nervous system. The same with the hairs, and the hair of the head; but this partial loss of hair must not be mistaken for alopecia areata, which is a contagious disease.

FIG. 5.—Hysterical gangrene complicating a bullous eruption. (St Louis Hospital Museum.)

TREATMENT.—In the case of reflex erythemas, treatment should be directed to the affections which are the cause of these reflexes. In trophic eruptions, the lesion of the peripheral or central nervous system must be treated in the usual way. The eruptions should be covered with occlusive and antiseptic dressings.

In many cases of trophic ulcers of the fingers, secondary to traumatic neuritis caused by crushing of the hand, I have had good results from local hydro-faradic baths. The two rheophores of an induction coil are placed in a vessel filled with salt water; the patient places his hand in the water for ten minutes daily.

URTICARIA.

Urticaria is characterised by the rapid appearance of large flat red papules, or of papuliform patches, white in the centre and red at the edges. The sudden appearance and the histological lesions of this dermatosis connect it with the erythemas.

Urticaria may be acute or chronic. Acute urticaria may be febrile or not; there is a special form, *autographic urticaria*, which should be described apart. Chronic urticaria includes two forms: common urticaria and pigmentary urticaria.

Acute Urticaria.—In *febrile* urticaria the eruption is preceded by slight fever and shivering and signs of gastric disturbance; epigastric pain, nausea, vomiting, and sometimes diarrhœa, lasting from a few hours to two days. In *non-febrile* urticaria the eruption is neither preceded nor accompanied by general phenomena. Whatever the mode of onset of urticaria, the eruption always presents the same characters; it is preceded by itching and burning sensations in the parts affected. It has been stated that the eruption of urticaria is secondary to scratching, that it is a pruritus which becomes eruptive. In certain subjects, it is true, the pruritus and the eruption may be suppressed on the limbs by covering the skin with a layer of wool, thus preserving them from external irritation; but it does not follow that the urticaria is exclusively due to the pruritus and not a neurodermatitis. The pruritus is simply the first manifestation of the congestive process which takes place in the skin, under the influence of many causes, which we shall study later on.

The eruption appears most often on the trunk, buttocks, thighs, shoulders and arms. It is sometimes local, sometimes general. It is formed by raised spots or wheals, varying in diameter from that of a sixpence to that of a half-crown; some of the spots are round, some oval. The colour is red or pink; sometimes the spots are pale in the centre and red at the periphery. The eruption is attended by intense itching, which leads to scratching, and this tends to produce fresh spots. Each spot lasts a few hours, or even only a few minutes; but, as the spots appear in successive crops, the eruption lasts for twenty-four or forty-eight hours, or sometimes a little longer. The eruption disappears without desquamation.

Varieties.—Sometimes urticaria occurs in the form of large white patches situated on a red base (*porcelanous urticaria*); sometimes there are raised nodules almost as large as a nut (*tuberous* or *nodose urticaria*); sometimes there is a single, very large nodosity (*giant urticaria*).

The urticarial spots are produced by acute congestive œdema of the skin; in some cases, the œdema predominates over the congestive

phenomena and forms *œdematous urticaria*. The latter occurs in regions provided with loose cellular tissue—the eyelids, prepuce and scrotum; the labia majora, vaginal orifice and urinary meatus in women. Around the anus, the œdema may simulate external hæmorrhoids.

In rare cases the vascular exudation is still more abundant and raises the epidermis in the form of bullæ (*bullous urticaria*); the ruptured bullæ give place to crusts, which eventually fall off and sometimes leave pigmentation. Lastly, the congestion may be intense enough to cause rupture of some capillaries (*hæmorrhagic urticaria*); surrounding the spots is a red ring, which passes through all the tints of ecchymoses.

Internal Urticaria.—Urticaria may also affect the mucous membrane of the mouth, nose, pharynx, larynx, and even the bronchial tubes. In the mouth and pharynx the eruption appears in the form of raised red patches with considerable œdema, chiefly situated on the tongue, soft palate and uvula. In the larynx it gives rise to œdema of the glottis, and causes symptoms of asphyxia. Gueneau de Mussy has described the alternation of cutaneous urticaria with asthma, the latter being due to urticaria of the bronchial mucous membrane; but this asthma is not essential asthma, it is a dyspnœa caused by the internal eruption. Gueneau de Mussy even went so far as to regard the vomiting and diarrhœa, which precede the cutaneous eruption, as due to urticaria of the digestive tract; but this is anatomically impossible, for the urticarial papule can only develop on the skin or on a dermo-papillary mucous membrane. The gastro-intestinal symptoms are only the first manifestations of the intoxication which produces the urticaria. In some cases these symptoms alternate with, instead of preceding, the urticarial eruption on the skin; they are then, like the urticaria, manifestations analogous to arthritism.

Autographic Urticaria, or Dermographism.—This eruption is easily provoked in subjects with very sensitive skins; nervous subjects who may or may not be subject to actual hysterical attacks, but who are generally affected with latent hysteria. If the finger or a blunt-pointed instrument is drawn along the skin, the parts touched become rapidly raised in the form of a red wheal; in this way letters and figures of all kinds can be traced. The wheals, at first red, afterwards become pink, and then white, surrounded by a red zone. They sometimes last for several hours, but are not accompanied by itching.

Chronic Urticaria.—This has the same aspect as acute urticaria, and is characterised by the reappearance of the eruption every day or several times a day for long periods. It is sometimes diurnal, sometimes nocturnal, and may be produced by the least change of

temperature; it sometimes appears regularly every morning and evening. The affection may last for months or even years; it persists in spite of all kinds of treatment, and may lead to a sort of decline, caused by continual itching, and loss of sleep and appetite.

Besides this chronic form, there is a form of intermittent urticaria which is a manifestation of malaria (*febris intermittens urticata*). This yields to sulphate of quinine.

Pigmented or Pigmentary Urticaria.—This form, which is undoubtedly a special disease, was first described by Nettleship (1867), and afterwards studied by Colcott Fox and others. It generally appears in infancy, a few months after birth; but it is sometimes observed in adolescents, and Besnier reported two cases in adults. It is formed by spots or patches, more or less raised like those of urticaria, and at first of a red colour. According to the degree of elevation of the eruptive elements, three forms may be distinguished: (1) a macular form, represented by spots; (2) a nodular form, formed by large papules; (3) a mixed form, combining the two preceding.

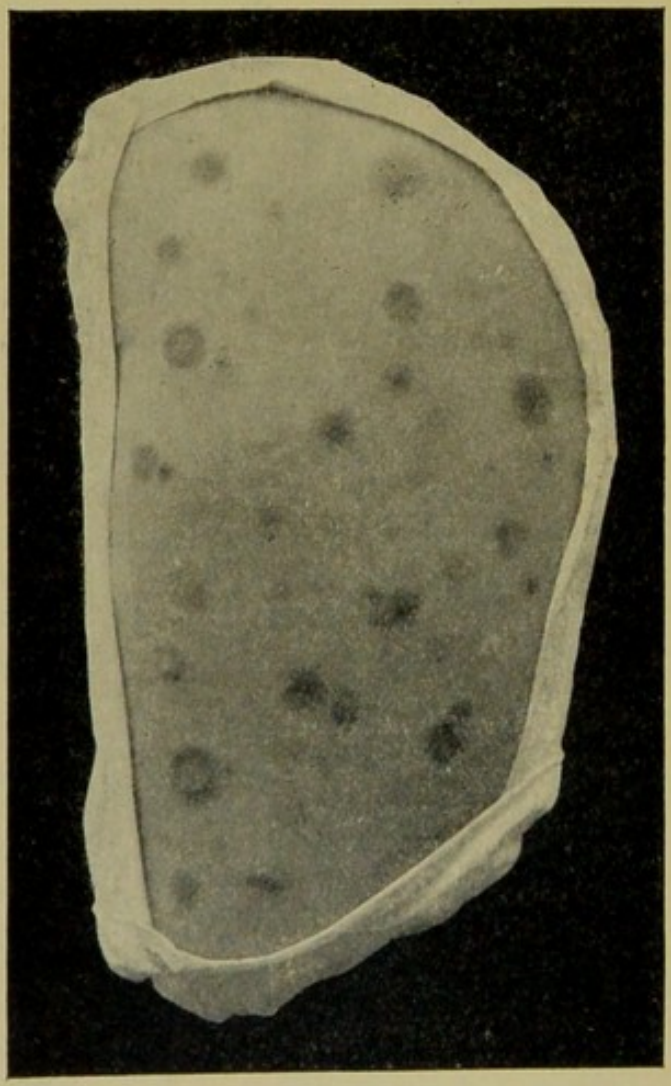


Fig. 6.—Pigmentary urticaria. (St Louis Hospital Museum.)

These papular spots or patches, of different sizes, round or oval in shape, occur on the whole surface of the body; trunk, limbs and face. After a time they disappear, leaving pigmentation, at first yellow, afterwards brownish; this pigmentation is characteristic. The eruption appears in successive crops, sometimes separated by intervals, so that recent urticarial elements and the pigmented spots left by former elements are met with at the same time. Moreover, from time to time erythematous eruptions appear,

on the spots or on the healthy skin, spontaneously or as the result of scratching (this form of urticaria is very pruriginous). Sometimes vesicles and small bullæ are developed on the spots.

For about a year successive outbreaks of eruption occur; then follows a long period of retrogression, during which the pigmentation finally disappears without leaving scars. The disease lasts for eight or ten years. Its etiology is quite unknown. Microscopic examination has shown the presence of dilated vessels in the papillary body, and an agglomeration of cells with special characters (mast-cells) in the dermis. The pigmentation of the spots is due to these cells, to crystals of hæmatin in the papillæ, and to pigmentation of the Malpighian cells.

Diagnosis.—The diagnosis of urticaria is very easy; the raised spots, the rapid disappearance of the eruption, and the intense itching differentiate urticaria from all the varieties of erythema. However, we must bear in mind that there is an urticarial form of erythema which forms a transition between erythema and urticaria. Again, urticaria often occurs along with other dermatoses, such as dermatitis herpetiformis and chronic prurigo. The diagnosis between urticaria and facial *erysipelas* is sometimes difficult: in urticaria, there is œdematous swelling with little redness; in erysipelas, on the other hand, the redness is much more marked, and has a characteristic raised border; in erysipelas the febrile and general symptoms are more intense than in urticaria.

Pathological Anatomy.—According to Renaut, Vidal, and Rindfleisch, urticaria is a kind of acute œdema of the papillary body with congestion of the papillæ; the latter is even more marked than in the erythemas. Renaut caused artificial urticaria by injecting a few drops of water into the dermis by means of a Pravaz syringe. The white coloration in the centre of the urticarial spots is due to serous extravasation, which compresses the vessels and produces anæmia in the centre of the spots, while the peripheral redness is due to collateral hyperæmia.

Etiology.—Urticaria is caused by vaso-motor disturbance. This may be provoked by external agents, as in eruptions caused by contact with nettles, medusæ and certain caterpillars, and by bugs. The same vaso-motor disturbance may result from an intoxication, which may be of alimentary, medicamentous or infective origin. Lastly, urticaria may be caused by primary disorders of the nervous system.

In certain persons the ingestion of mussels, oysters, salt-water fish, crayfish, pork, etc., gives rise to an urticarial eruption; some persons are even so susceptible that they cannot eat strawberries, eggs, etc., or drink seltzer water or champagne without suffering from urticaria. In these cases the eruption is often preceded by

vomiting and diarrhœa, which are the first signs of the intoxication. This intoxication has been attributed to the presence of ptomaines in the substances ingested. Bouchard has pointed out the frequency of urticaria in persons affected with dilatation of the stomach; in this case the eruption is due to the absorption of products of fermentation formed in the dilated stomach.

Medicamentous urticaria may follow the ingestion of certain drugs, such as the balsams, chloral, etc.

In certain infective diseases, such as variola and malignant diphtheria, urticarial eruptions are observed, and sometimes in the second week of enteric fever. Malaria may also give rise to urticaria, in two different forms: sometimes the eruption accompanies the febrile attacks; sometimes it replaces them, in cases of latent fever, and yields to sulphate of quinine. All these urticarias are due to the absorption of soluble products of infective origin.

The urticaria which is due to the penetration of liquid from a hydatid cyst into the peritoneum is allied to *toxic urticaria.* After puncture or spontaneous rupture of a cyst into the peritoneal cavity, an urticarial eruption sometimes appears.

Urticarias of *nervous origin* are those which occur after strong emotion or anger. In the same group may be placed the eruptions which are produced by simply touching the skin, the type of which is dermographism; also the urticarias of reflex origin, especially uterine.

But above all these causes must be placed the diathetic influence, or *neuro-arthritism;* for drugs and foods only cause urticaria in persons who are predisposed.

Treatment.—If the urticaria is due to an external cause, to an article of diet or a drug, the irritating cause must be suppressed. In cases of alimentary intoxication, a mild purgative should be given, followed by intestinal antisepsis by means of salicylate of bismuth or magnesia, and benzonaphthol. Alkalis and milk diet are sometimes useful. If there is dilatation of the stomach, this must be treated.

Locally, hot lotions containing a third part of vinegar, or 1 per cent. carbolic acid, should be applied. Ointments containing menthol or guaiacol (1 per cent.) are also useful; these should be covered with starch or talc powder containing a little camphor. Finally, if local treatment fails, we may attempt to ease the itching by preparations of valerian.

In chronic urticaria, sulphate of quinine may be administered; but this drug only acts efficaciously in malarial urticaria. Mineral waters may be prescribed to counteract neuro-arthritism.

In urticaria of the respiratory passages, with dyspnœa or asthmatic attacks, spirit of ether should be prescribed.

PITYRIASIS ROSEA.

Described for the first time and individualised by Gibert, this dermatosis differs from the other forms of pityriasis by its pseudo-exanthematous character.

Symptomatology.—It is an erythemato-squamous eruption (squamous roseola) which generally appears without any prodromal

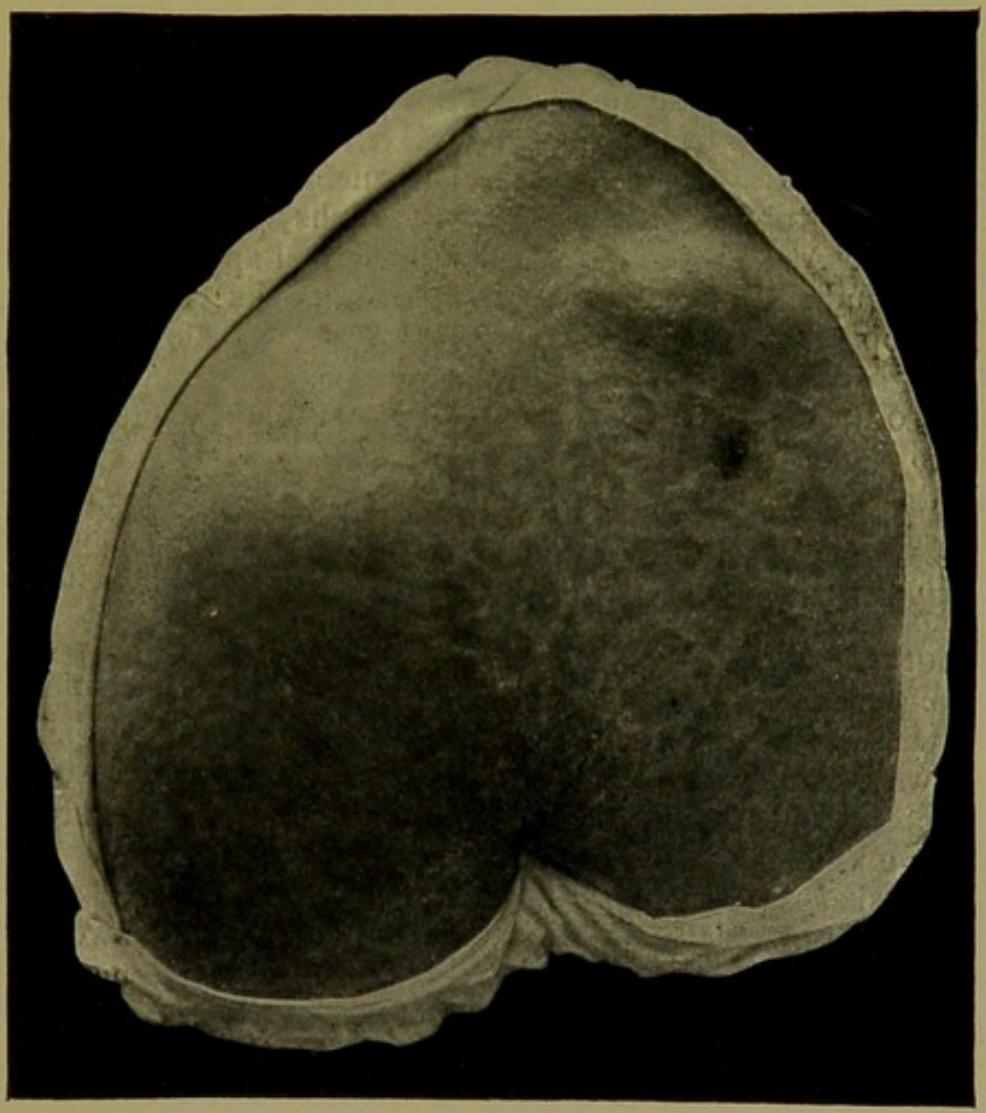


Fig. 7.—Pityriasis rosea. (St Louis Hospital Museum.)

symptoms; very rarely there is a slight rise of temperature, with slight gastric disturbance and malaise.

According to Brocq, a single patch appears at first, which progresses for eight or ten days, and is then followed by the generalised eruption. The latter consists of isolated, rounded spots, of a rose colour and the size of the finger-nail; these disappear momentarily

under pressure. This form, which is the most common, is *pityriasis maculata.* Another form, with larger elements in the form of complete or incomplete rings with healthy skin in the centre, is *pityriasis circinata.* This form is nothing more than *pityriasis maculata,* in which the elements have undergone resolution in the centre, while the peripheral parts remain.

The redness of the eruption rapidly fades, without disappearing altogether, and the spots become covered with small squames. These are easily detached at the centre of the spots, but are more adherent at the periphery. The spots increase in size more or less regularly, generally remaining isolated but sometimes becoming confluent. The eruption appears in successive crops, so that spots of different appearance are seen at the same time.

Pityriasis rosea commences on the neck, chest and shoulders, then extends to the arms, abdomen, thighs and legs. Itching is very slight. At the end of a fortnight the red spots become yellow, then fade, and disappear at the end of five weeks; but as there are several crops, the affection may last for two months or longer.

The *prognosis* is always benign.

Etiology.—The etiology is unknown. Bazin attributed pityriasis rosea to rheumatism, and called it a "pseudo-exanthematous arthritide"; but there is nothing to justify this hypothesis. Bouchard has observed pityriasis in cases of dilatation of the stomach, and considers that there is a connection between the two affections.

Diagnosis.—Pityriasis rosea must not be mistaken for *trichophytic circinate erythema.* This affects chiefly the backs of the hands, wrists, neck and face, *i.e.,* the exposed parts of the skin; the spots always spread eccentrically, and the borders are often vesicular; lastly, microscopic examination shows the characteristic spores, while there is no known specific parasite in pityriasis rosea.

Pityriasis rosea is most often mistaken for *syphilitic roseola;* but the latter occurs chiefly on the abdomen, flanks, and lower part of the chest and forearms; pityriasis rosea is situated more on the upper part of the body. The spots of syphilitic roseola are more coppery than red, and are not squamous. Lastly, in syphilis there will be other characteristic signs.

The spots of *pityriasis versicolor* have a special café-au-lait colour, and scratching with the finger-nail removes thick squames.

Vernal roseola is an exanthem characterised by a general eruption of spots without squames. It is unnecessary to discuss the diagnosis from *measles.*

In *psoriasis* the squames are pearly white, larger, thicker, and more abundant than in pityriasis rosea. However, when psoriasis is acute and generalised, and consists of small elements, the diagnosis

may be less easy; the existence of former outbreaks; scratching the patches, which renders the squames more apparent in psoriasis, and reveals beneath them a red, flat, papular elevation which bleeds easily, will serve to make the diagnosis.

Eczema, when moist, cannot be mistaken for *pityriasis rosea;* in the case of dry eczema confusion will be avoided by remembering that the squames of eczema are soft and occupy large areas, and that the eruption is more tenacious. The diagnosis from *seborrhœic eczema* is more difficult, but in this dermatosis the patches are situated principally on the trunk, and are covered with soft, seborrhœic squames. However, there are cases of generalised seborrhœic eczema, in the form of round, squamous patches of small dimensions, which so closely resemble pityriasis rosea, that the existence of transitional forms between these two diseases is not impossible.

TREATMENT.—This is very simple. Every other day a bath of starch and borate of soda should be taken. An ointment of zinc oxide or glycerole of starch may be applied, but it is generally preferable to abstain from any local applications. Irritating ointments should be avoided.

PURPURA.

The term *purpura* is applied in a general way to hæmorrhages in the skin. The elementary lesion consists of spots called *petechiæ*, or *spontaneous ecchymoses*, according to their dimensions.

SYMPTOMATOLOGY.—*Petechiæ* are punctiform or lenticular spots, bright red or bluish at first, but afterwards becoming brown and then yellow, in accordance with the changes in extravasated blood, before disappearing altogether. They do not disappear under digital pressure. Petechiæ are chiefly met with on the lower limbs, on the anterior surface of the legs; they are rare on the upper limbs, but sometimes occur on the back of the forearm; they are still more rare on the trunk and face. They are more or less confluent, and when present in great numbers they are mingled with ecchymoses.

Purpura appears in successive crops, so that spots in different degrees of evolution are found in the same region. Fresh outbreaks are provoked by walking and by prolonged standing, while the horizontal position hastens their disappearance.

Petechiæ are sometimes observed on the mucous membrane of the mouth and tongue, where they assume the aspect of small black grains.

Spontaneous ecchymoses are larger than petechiæ; usually the diameter of a sixpence, they may attain much larger dimensions, and even occupy nearly the whole of a limb.

In some cases petechiæ and ecchymoses coincide with polymorphous erythematous lesions, such as papular erythema or erythema nodosum; these erythemas themselves are often purpuric, not disappearing completely under pressure. Sometimes petechiæ begin as papules, which soon disappear, but the colour remains. There is not always, therefore, a sharp line of demarcation between these different eruptions. Sometimes hæmorrhagic bullæ develop on the ecchymoses, in the same way that bullæ form in polymorphous erythema.

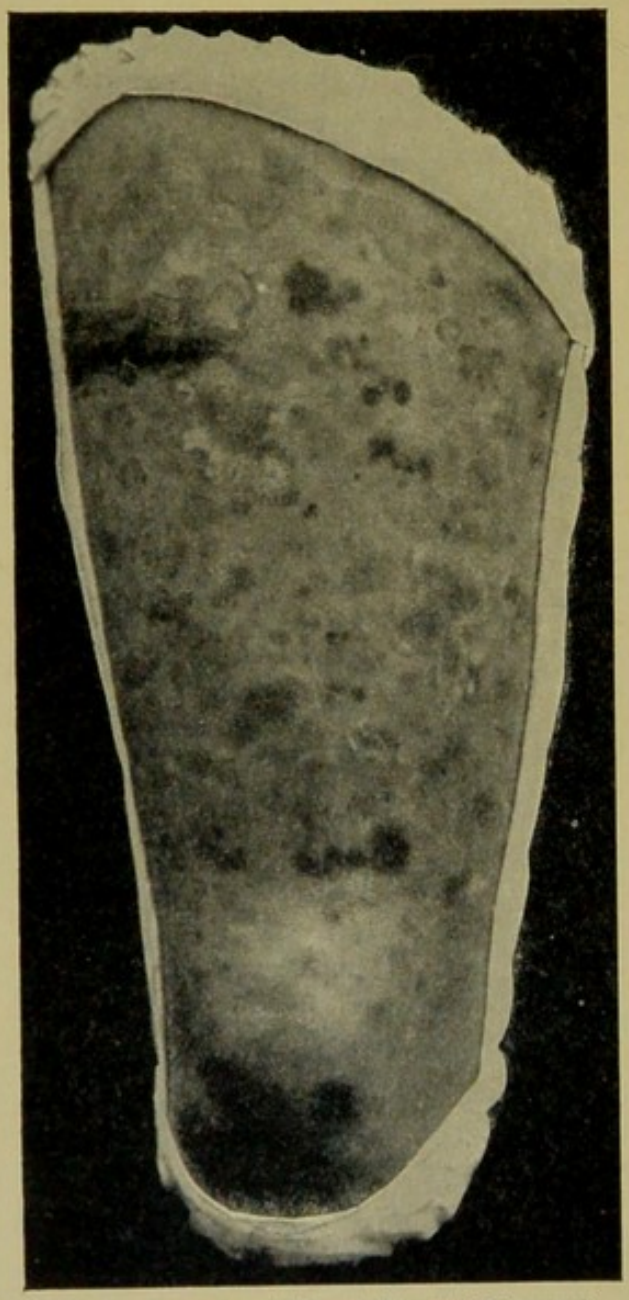


Fig. 8.—Purpura. (St Louis Hospital Museum.)

Some infective purpuras may be complicated by ulceration and cutaneous gangrene.

Lastly, cutaneous hæmorrhages may coincide with general phenomena, functional disorders, and hæmorrhage from the mucous membranes and in the viscera.

The *prognosis* of purpura is very variable. Some cases are very benign, while others, on account of their complications and the visceral hæmorrhages which accompany them, are of grave prognosis.

This accounts for the old division of purpura into two forms: *purpura simplex* and *purpura hæmorrhagica*. Hayem has shown that this clinical and prognostic distinction corresponds to a different condition of the blood in the two forms. In purpura simplex the blood-clot is retractile, as in the normal state; in purpura hæmorrhagica the clot is not retractile, and preparations of blood show a precipitation of hæmatoblasts.

Classification.—Purpura may be primary or secondary, accord-

ing as it constitutes the principal phenomenon of the morbid evolution, or is only a complication.

Punctiform hæmorrhages occur in certain forms of chronic eczema and psoriasis affecting the lower limbs, and in some syphilides. A localised purpura sometimes occurs after the lightning pains of tabes and certain neuralgias (purpura of nervous origin). Purpuric spots may be caused by violent muscular efforts or by prolonged standing.

Secondary purpuras include secondary infective purpuras, toxic and cachectic purpuras, and those of scurvy and hæmophilia. Primary purpuras are subdivided into: (1) rheumatic purpura; (2) primary infective purpura; (3) Werlhof's disease.

Secondary Purpura.

Infective Secondary Purpura.—This is met with in the course of infective diseases, such as variola, scarlatina, measles, intermittent fever, pyæmia, puerperal infection, ulcerative endocarditis, acute tuberculosis (of which purpura may be a premonitory phenomenon), diphtheria, enteric fever, typhus, plague, malignant jaundice, pneumonia, cerebro-spinal meningitis, infective tonsillitis, and gonorrhœa.

Toxic Purpura.—The absorption of certain substances is often followed by purpura, but a certain predisposition is necessary for this to occur. Toxic purpura manifests itself in the form of petechiæ, chiefly situated on the legs, sometimes also on the wrists. Iodides may give rise to purpura (iodic purpura), also to hæmorrhage from the gums and severe epistaxis. Purpura has been also observed after the administration of chloral, sulphate of quinine and arsenic, and is common in phosphorus poisoning. It sometimes occurs in alcoholic poisoning, and has been observed in the legs in a case of alcoholic paralysis.

Cutaneous hæmorrhages are seen in secondary malignant jaundice, and may occasionally occur in uræmia.

Cachectic Purpura. — Purpura may complicate pernicious anæmia, leucocythæmia, diseases of the spleen and advanced cardiac disease. In old people, it occurs on the posterior surface of the forearm and on the back of the hand. Cutaneous hæmorrhages are sometimes observed in cancer, pellagra, and malarial cachexia.

Under the name of *chronic angio-sclerotic purpura*, I have described a special and not uncommon variety of purpura situated on the lower limbs, which may be attributed to vascular sclerosis. This form of purpura consists of brown spots, chiefly affecting the lower part of the legs. The spots are persistent, and result from changes in extravasated blood, arising from the rupture of degenerate and friable capillaries. In exceptional cases this variety of purpura, instead of

remaining limited to the lower limbs, may spread over nearly the whole surface of the body in successive crops, the spots of which, at first purpuric, end in the formation of brown patches of indefinite duration.

Scorbutic Purpura. — In scurvy, petechiæ, ecchymoses and œdema appear on the lower limbs, at the same time as hæmorrhage from the swollen and fungoid gums. The situation of the lesions round the hairs has been considered wrongly as being peculiar to scurvy. Cases of scurvy have been reported in which the lesions were limited to a few spots on the limbs and slight bleeding from the gums.

Lastly, we must mention the severe ecchymoses and hæmorrhages which occur in *hæmophilia.*

Primary Purpura.

Rheumatic Purpura.—This was and is still called *rheumatic* by those who, like myself, believe that the concomitant joint affections are of rheumatic origin (rheumatic peliosis). This view is, however, not accepted by many contemporary authors. Mathieu has proposed the term *rheumatoid purpura* to avoid all ambiguity, but I still hold that this form of purpura is closely related to primary acute articular rheumatism. It is generally accompanied by rheumatoid and gastro-intestinal symptoms of various degrees of severity.

Rheumatic purpura is usually a simple purpura; but along with purpura limited to the lower limbs (rheumatic peliosis), may be observed a generalised purpura, affecting the whole body and aggravated by the production of internal hæmorrhages.

This affection generally occurs in young people as the result of prolonged walking, or excessive fatigue. It manifests itself at first by pain in the knees, sometimes in the instep; there is a little periarticular or articular swelling due to synovial effusion. Petechiæ appear from the first on the lower limbs; in more severe cases, on the upper limbs and even on the trunk. Sometimes there are ecchymoses, in the annular form of certain erythemas. In cases of generalised purpura, there is swelling of the hands and elbows. Besides the hæmorrhagic spots, œdema, papular erythema, or even erythema nodosum with a hæmorrhagic tint are sometimes observed on the lower limbs, showing the relations which exist between purpura and polymorphous erythema. In these cases the congestion of the vessels has caused rupture of their walls.

This form of purpura usually presents few general manifestations; fever is absent, but there may be epigastric pain, vomiting, colic and diarrhœa. In severe cases there are rigors and fever (38° C.), and hæmorrhage from the nasal, buccal and intestinal mucous membranes. Cardiac and visceral complications are exceptional.

A peculiarity of this form of purpura is its evolution in successive outbreaks, so that new spots are found mixed with older ones which have more or less faded. The affection may last for some weeks or months.

Primary Infective Purpura.—Three forms of infective purpura have been described—typhoid, hyperacute, and pseudo-rheumatic.

Typhoid form.—In this form typhoid symptoms are associated with cutaneous hæmorrhages. In some cases the typhoid symptoms precede the cutaneous hæmorrhages; the symptoms then include rigors, pain in the hypochondriac regions, lassitude and headache, depression and complete loss of appetite. After this appear epistaxis, diarrhœic stools, often mixed with blood. All these symptoms suggest typhoid fever, till petechiæ appear on the legs and thorax. The spleen is enlarged, the tongue white, and the temperature may rise to 37° or 40° C. Other hæmorrhages may occur, such as hæmaturia or hæmatemesis. This form generally ends fatally, either from hæmorrhage or exhaustion.

Hyperacute form.—The onset of this form is acute and sudden, its progress is rapid, and it ends fatally in a few days. The symptoms are the same as in the preceding form, viz., rigors, fever, severe general symptoms, petechiæ and ecchymoses.

Pseudo-rheumatic form.—At first sight this resembles acute articular rheumatism with purpuric spots; the pain is acute, there is considerable swelling of the joints, high fever, colic, and vomiting, together with petechiæ and ecchymoses. Sometimes the general symptoms predominate over the articular pains. This form of purpura is also very grave, internal hæmorrhages being often considerable, or dangerous owing to their situation; for instance, in the nervous system. Sometimes gangrene ensues in the purpuric patches, resulting in cicatricial contraction and deformity.

Werlhof's Disease.—This is epitomised by Lasègue as follows:—"No fever nor prodromal malaise. The affection begins by a more or less severe hæmorrhage, most often gingival, sometimes by an epistaxis. On the next day, a petechial eruption appears on the lower limbs, sometimes extending to the trunk and upper limbs. Hæmorrhage or oozing of blood continues, and, according to its amount, causes melæna or hæmatemesis. Rapid improvement, and cure on the eighth or fifteenth day." But the termination is not always so favourable, as fatal cases have been reported.

The etiology of this disease is very obscure; in some cases the exciting cause appears to have been a fall, fright, or violent anger.

Diagnosis.—In infective purpura there is high temperature, typhoid state, and often albuminuria. In rheumatic purpura there are joint pains, but only slight fever. In Werlhof's disease there is no fever, but hæmorrhages predominate. In spite of these differences

it is often difficult to decide to which form of purpura a given case belongs.

Typhus is recognised by the injection of the face at the onset, the petechial exanthematous eruption, the intensity of the nervous symptoms, and the course of the temperature. The history and concomitant symptoms assist in the diagnosis of secondary purpuras, cachectic, toxic, or scorbutic.

Pathological Anatomy.—The vessels of the dermis, especially those of the papillary layer, are dilated and engorged with red blood corpuscles. The nuclei of their endothelial cells are enlarged and sometimes tend to become segmented. In the centre of the purpuric spots most of the vessels present changes in their walls; the endothelial cells undergo proliferation and desquamation, so that the dilated vessels are almost destitute of endothelium. These vessels are obstructed by a fibrinous clot full of red corpuscles.

In this degree, the purpuric lesion is only a vascular dilatation, and the purpura is only telangiectasic; in a more advanced degree, the vascular walls are ruptured, giving exit to red corpuscles which form small hæmorrhagic foci around the vessels in the dermis.

Pathogeny.—In the majority of cases purpura, like polymorphous erythema, is an angioneurosis, due to various causes and characterised by considerable vascular dilatation.

The pathogeny of rheumatic purpura is the same as that of rheumatic polymorphous erythema.

With regard to primary infective purpura, several observers have found micrococci and bacilli in the blood during life. Martin de Gimard, in two cases, found micrococci, and succeeded in making cultures which, when inoculated in rabbits, gave rise to ecchymoses and even hæmorrhagic infiltration in some of the viscera; in two cases gangrenous patches developed. The microbial infection causes the vaso-motor disturbance, from which results dilatation and thus rupture of the vessels.

The pathogeny of secondary infective purpuras is the same as that of primary purpuras. The pathogeny of toxic purpuras is identical with that of erythemas of the same nature.

In certain cases, purpura is possibly caused by embolism; thus, in a case of purpura consecutive to endocarditis and followed by pneumonia, Claisse found, in a purpuric spot, a clot with masses of pneumococci occupying a small vessel in the dermis. In leucocythæmia, the cutaneous hæmorrhages appear to be due to the accumulation of leucocytes in the capillaries and to rupture of these vessels.

In cachectic purpura and in scorbutic purpura the purpuric spots are probably produced by rupture of the vessels, due to changes in their walls. In hæmophilia, purpura appears due to fragility of the

small vessels; the same in senile purpura, where the capillaries are affected with arterio-sclerosis.

TREATMENT.—Purpuric spots disappear after absolute rest in bed. Internal and external hæmorrhage may be checked by subcutaneous injections of ergotine, large doses of lemon juice mixed with powdered sugar, chloride of calcium (3 grammes daily), and subcutaneous injections of adrenalin ($\frac{1}{10}$ milligram). Fever may be abated by sulphate of quinine, and the general condition improved by tonics, alcohol, and a nourishing diet. In cases of iodic purpura, extract of rhatany is recommended.

HERPES.

Herpes is an inflammatory, vesicular dermatosis, characterised by the formation of round vesicles, arranged in distinct groups, and resting on an erythematous base.

CLASSIFICATION.—Herpes may develop under the influence of different etiological conditions, which impart special characters to the affection: (1) herpes is observed as a secondary symptom in the course of certain morbid conditions; this is *symptomatic herpes;* (2) it may also constitute by itself an independent disease, known as *herpetic fever;* (3) it constitutes the peculiar affection known as *zona*, or *herpes zoster;* (4) it may be of external origin, and represent a simple local affection.

Symptomatic Herpes.

Symptomatic herpes occurs in febrile gastric disturbance, pneumonia, cerebro-spinal meningitis, enteric fever, malaria, etc. It has a special localisation, being always situated near the mucous membranes, especially round the lips (*herpes labialis*), but sometimes round the nares. Sometimes herpes appears on the glans penis or prepuce (*herpes preputialis*), or round the vulva and anus. Sometimes it forms groups of vesicles on the cheeks, and even on the inner surface of the lips.

There is a form of symptomatic herpes which appears to be due to menstruation, and is known under the name of *catamenial herpes.* Some women are affected with this at each menstrual period, and the herpes always affects the same place in the same woman; sometimes round the lips, sometimes on one cheek, sometimes on the vulva or buttock. These are the four most frequent situations.

The vesicles of symptomatic herpes are not numerous; they soon dry up and form small yellow or brown crusts.

According to Leloir, herpes may be aborted by the application of pure alcohol to the affected region. If this fails, simple powders may be applied.

Herpetic Fever.

Herpetic fever is characterised by fever, and a disseminated herpetic eruption. It may therefore be considered as a special exanthematous fever.

The disease is preceded by prodromal symptoms, consisting in fever of varying degree, malaise and anorexia; sometimes there is epistaxis.

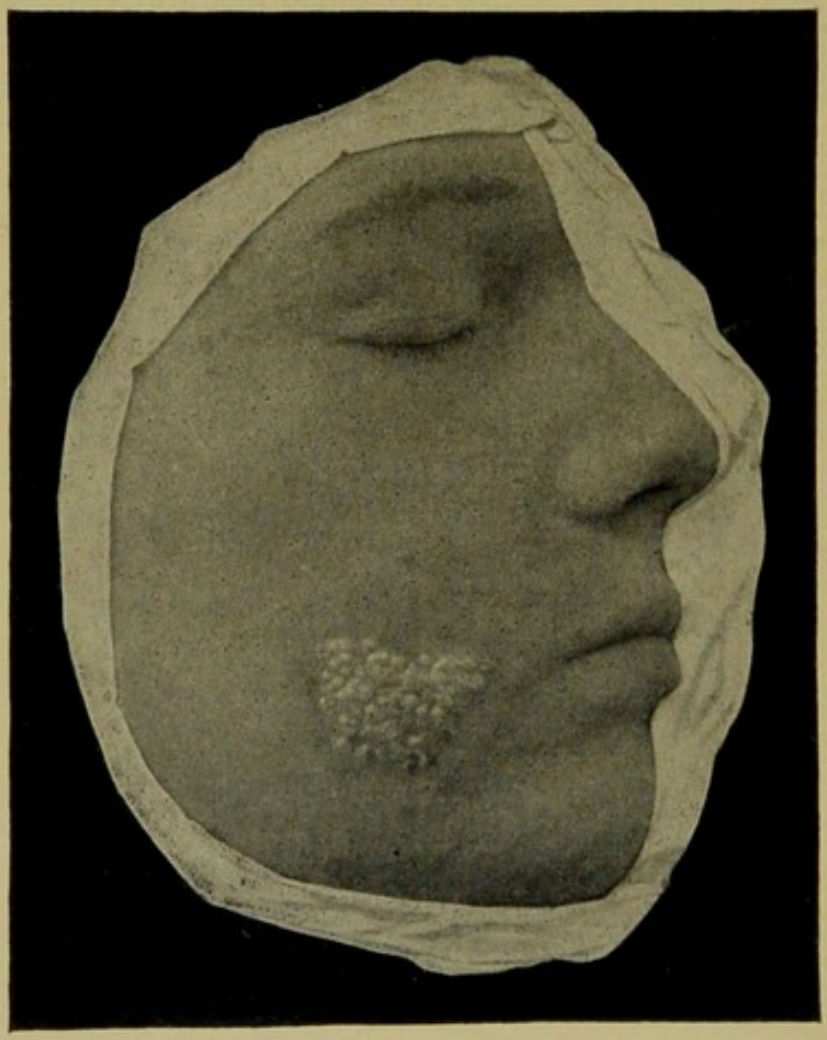


Fig. 9.—Herpes of the cheek. (St Louis Hospital Museum.)

In some cases these general symptoms may cause anxiety, and the temperature may reach 40°: the patient is delirious, and some serious disease is feared; then, at the end of two or three days the temperature falls, the general symptoms subside, and the eruption appears.

The latter consists of herpetic groups which present the usual characters, but are confined to the neighbourhood of the mucous orifices. The vesicles are situated irregularly on each side of the

face, on the cheeks, nose, eyelids and ears. They sometimes extend to the rest of the body, and may occupy the neck and chest and occasionally the limbs. They are always discrete. The eruption may affect the mucous membranes only; vesicles occur in the mouth, where they rupture and become painful, but the eruption is more common in the pharynx, where it constitutes *herpetic angina*. The eruption on the mucous membranes often coexists with cutaneous herpes, proving the identity of these different manifestations.

The vesicles go through the usual evolution; on the skin, they dry up and form crusts, which soon fall off; on the mucous membrane, instead of crusts there is a pseudo-membranous exudation covering an ulcerated surface, which heals after the membrane is detached. In fact, herpetic fever is a benign affection.

ETIOLOGY.—This is still very obscure. It has been supposed to be an infective disease developing after fatigue, excesses, etc. The liquid of the vesicles always contains micro-organisms (staphylococci), but no specific one has yet been found. Auto-inoculation is sometimes successful. Certain forms of herpes may be contagious; the contagiousness of herpetic angina, although slight, is undoubted.

DIAGNOSIS.—Herpetic fever must not be mistaken for *varicella*, the vesicles of which are isolated and not grouped in patches. The eruption of *scabies* is polymorphous, not exclusively vesicular. Before the eruption, herpetic fever may be mistaken for enteric fever, but not for long. However, the herpetic eruption may be unnoticed if it is situated on the genital organs, especially in women, and the diagnosis may be erroneous for some time.

TREATMENT.—This consists in the administration of sulphate of quinine and a purgative, and the local application of simple powders.

Zona, or Herpes Zoster.

Zona, or herpes zoster, is characterised by groups of herpetic vesicles arranged in linear series along the course of the cutaneous nerves.

PATHOGENY.—Parrot regarded zona as a trophic disorder of the skin. The histological researches of Danielsen and others have shown that nerve changes are constant in zona. Baerensprung, in a case of intercostal zona, found that the intercostal nerves of the region were affected with interstitial neuritis, and that the corresponding spinal ganglia showed interstitial inflammation.

In ophthalmic zona, Wyss and Horner have found interstitial inflammation of the Gasserian ganglion.

ETIOLOGY.—The nerve lesion is only intermediary between the dermatosis and the true cause of zona; which clinical observation shows to be multiple.

First of all, zona often appears in a quasi-epidemic form, especially in the spring; in this case it is accompanied by slight general symptoms, and appears to confer immunity, although there are several authentic cases of recurrence. It seems, therefore, that in such cases the cause of zona may be an infection which acts on the nerves, and, as the result of the nerve lesion, gives rise to a cutaneous eruption. This idea is due to Landouzy, who regards zona as a true zosterian fever. I have myself seen a case in which zona was accompanied by an eruption of febrile herpes affecting the whole body. Some cases appear to have been due to contagion, but, on the other hand, inoculations have always been negative. In other cases zona appears due to the action of cold. Zona may also be symptomatic of affections of the posterior columns of the spinal cord; for example, in *tabes*, it sometimes occurs on the limbs affected with lightning pains. Zona has also been observed in connection with neuritis caused by wounds or contusions of nerves, or secondary to periostitis or pleurisy. The eruption occurs not only after lesions of the large nerve trunks, but also after changes in the ends of the peripheral nerves. A superficial cutaneous lesion or a simple contusion, affecting the cutaneous nerves, may be followed by an eruption of zona on the area supplied by the injured nerve.

Zona has also been observed in connection with nerve lesions caused by carbonic oxide poisoning, and in arsenical peripheral neuritis.

Symptomatology.—Whatever its etiology, zona always presents the same characters; but the general symptoms which mark the onset of zosterian fever are absent in zona which is secondary to affections of the spinal cord or to primary lesions of the nerves. These general symptoms are generally mild, and are often unnoticed; they consist in slight fever, preceded or not by a slight rigor, accompanied by some gastric disturbance, headache and malaise. They cease with the disappearance of the eruption; sometimes as soon as the eruption appears. The eruption is often preceded by neuralgic pains, which may persist during the whole course of the disease, and even afterwards; frequently there are only pricking sensations. Zona is strictly limited to one side of the body. On the trunk, it takes the form of a semi-girdle; on the limbs, it forms bands parallel to the axis of the limbs. Very rarely it is bilateral. Multiple eruptions have been observed affecting the trunk and lower limbs (direct or crossed hemiplegic zona).

The eruption consists of red patches, on which are situated vesicles, usually small but sometimes very large (bullous zona). The patches occur along the course of a nerve, and appear successively in four to eight days. The first groups which appear are usually situated at the two extremities of the affected region: in thoracic

zona the two first groups appear, one anteriorly by the side of the median line, the other posteriorly by the side of the spinous processes.

When they are not excoriated, the vesicles dry up without breaking, forming brownish crusts which fall off in eight or ten days, leaving pigmented spots, which sometimes disappear but are more often replaced by a permanent, colourless cicatrix.

The duration of zona is generally from two to three weeks.

Complications.—Sometimes the vesicles rupture and form small ulcers, which increase the pain of the eruptions and leave depressed scars; these ulcers may be surrounded by a red inflammatory zone. The lymphatic glands corresponding to the affected region are sometimes enlarged, but rarely suppurate.

In old people and debilitated subjects, zona often becomes *hæmorrhagic.* All zonas may present some hæmorrhagic vesicles, but in the hæmorrhagic form all the vesicles are full of blood; these rupture instead of drying up, giving place to ulcers which suppurate for a long time, sometimes two or three months, and leave cicatrices in proportion to the depth of the lesion.

In old people and diabetic subjects, zona may be *gangrenous.* In this form necrotic areas are formed underneath the groups of vesicles; these are sometimes of long duration, according to the extent of the lesions and the condition of the subject.

Besides these severe forms, there is a benign or *abortive* form of zona, in which there are only a few groups of vesicles, sometimes only one or two. Sometimes the vesicles abort, remaining in the state of papules. But this form may be as painful as ordinary zona.

The *neuralgic pains* (which are not constant in zona) are sometimes intense. They may persist long after the eruption, and are sometimes accompanied by disorders of sensation—anæsthesia and hyperæsthesia—affecting the area of distribution of the nerve underlying the eruption. But these patches of anæsthesia and hyperæsthesia have no direct relation to the groups of vesicles. Zona is sometimes accompanied by atrophy of certain muscles, loss of hair, loss of teeth (in zona of the trigeminal nerve), partial paresis, and paralysis, especially paralysis of the oculo-motor muscles and paralysis of the arm; also by vaso-motor and secretory disorders. Facial hemiplegia is occasionally seen, especially in zona of the neck; this may be a proof of the central origin of zona.

Regional Varieties.—Zona may occur along the course of any nerve, but certain situations are more common and more important than others. The seat of predilection is the thorax; then, in order of frequency, the abdomen, lower limbs, face, neck, upper limbs and perineum.

Thoracic zona.—This occupies one of the intercostal spaces,

sometimes two spaces. It lies along the course of an intercostal nerve, usually the third, fourth, fifth, sixth or seventh. It is strictly limited to one-half of the body, from the vertebral column to the anterior median line, without ever overstepping these two lines, except by a few millimetres. When it is accompanied by neuralgia, this may present the three painful points which are characteristic of intercostal neuralgia. Sometimes the eruption is preceded by pain in the side, which may, for two or three days, raise a suspicion of some chest affection.

Abdominal zona.—This has two sub-varieties. In the first, the eruption affects the lower part of the back, from the eighth dorsal to the first lumbar vertebræ, extending forwards to the linea alba (lumbo-abdominal zona); it occupies the course of the eighth, ninth, tenth, eleventh and twelfth thoracic nerves. In the other variety, the eruption follows the course of the upper lumbar nerves, descends anteriorly to the pubis in the inguino-scrotal or inguino-vulvar regions, and posteriorly to the gluteal region (lumbo-inguinal zona).

Lumbo-femoral zona.—This affects the lower limbs along the course of the cutaneous branches of the second, third and fourth lumbar nerves. It thus presents a very extensive eruption along the course of the femoro-cutaneous, genito-crural, obturator and crural nerves. It occupies the buttock, the anterior, external and internal surfaces of the thigh, and the calf, extending forwards to the scrotum or labium majus. Sometimes it is limited to the thigh or buttock.

Sacro-ischiatic zona.—This follows the cutaneous distribution of the branches of the sacral plexus, and occupies the buttock and sacrum, perineum, posterior surface of the scrotum, one side of the penis or one labium majus, the external semi-circumference of the leg and dorsal surface of the foot (external popliteal nerve) or the posterior surface of the leg and plantar region (internal popliteal nerve). It may be limited to the pudic nerve, constituting genital zona.

Facial zona.—Facial or trigeminal zona may be total, or limited to one branch of the trigeminal nerve. When total, the eruption affects the whole face, especially the points of exit of the nerves; the supra-orbital nerve at the supra-orbital notch, the infra-orbital nerve at the infra-orbital foramen, the temporo-malar branch of the superior maxillary nerve at the malar foramen, the inferior dental nerve at the mental foramen. Sometimes a unilateral, herpetic eruption is seen in the pharynx, characterised by vesicles and false membranes, situated on one side of the soft palate and anterior pillar of the fauces at the termination of the palatine nerves derived from Meckel's ganglion.

When the eruption is limited to the course of the ophthalmic

nerve it constitutes *ophthalmic zona.* This affects the upper eyelid, temple, forehead and scalp as far as the lambdoid suture, either at the same time or separately. It descends as far as the corresponding half of the nose, along the nasal nerve; the nasal mucosa may be swollen by affection of the internal nasal branch.

The external membranes of the eye are sometimes affected; the conjunctiva is red, and presents a vesicular eruption, and the iris is inflamed. These trophic ocular lesions, described by Hutchinson and others, are due to extension of neuritis from the ophthalmic nerve to the sensory root of the ophthalmic ganglion and ciliary nerves. Sometimes there is ulceration and even perforation of the cornea. Functional disorders consist in intense photophobia and severe neuralgic pains, which often persist long after the eruption.

Zona may also follow the branches of the *superior maxillary nerve.* The eruption then appears on the cheek and lower eyelid, the side of the nose, and upper lip. There are sometimes groups of vesicles on one side of the soft palate and pharynx. The neuritis may also extend to the dental nerves, giving rise to obstinate neuralgia, and sometimes even to loss of teeth.

When zona follows the branches of the *inferior maxillary nerve,* the eruption appears on the chin and lower lip, sometimes on the lobule of the ear, in the external auditory meatus, and on the temporal region. It may extend to the buccal mucous membrane along the branches of the buccal nerve, and even affect the corresponding half of the tongue (*lingual zona*).

FIG. 10.—Zona of the superficial cervical plexus.

Zona of the scalp.—This is rare, and the vesicles are difficult to see except in the bald. This form of zona is never isolated. It results from extension of zona of the forehead along the supra-orbital nerve, from extension of zona of the inferior maxillary nerve along the cranial branches of the auriculo-temporal nerve, from extension of zona of the nape of the neck along the great occipital nerve, or from zona of the superficial cervical plexus.

Zona of the neck.—This corresponds to the branches of the

superficial cervical plexus, and presents two varieties: ascending and descending zona. *Ascending zona* follows the auricular, mastoid and transverse cervical branches of the plexus; the eruption thus appears on the side of the neck, the ear, and mastoid region: it may extend to the angle of the jaw and the supra-hyoid region, following the course of the descending branch of the transverse cervical nerve. *Descending zona* follows the descending branches of the superficial cervical plexus, the supra-clavicular and supra-acromial. The eruption appears on the lower part of the side of the neck, and on the shoulder; it may descend as far as the thorax.

Brachial zona.—This follows the cutaneous branches of the brachial plexus; it occupies the lower and posterior part of the shoulder, descends more or less along the arm, anteriorly or posteriorly, as far as the forearm and hand. When it affects the region of the first four or five pairs of dorsal nerves, it forms a half girdle in the intercostal spaces, and a few groups of vesicles appear on the inner surface of the arm, owing to the anastomosis of the perforating branches of the first intercostal nerves with the accessory branch of the internal cutaneous nerve and with this nerve itself.

Diagnosis.—As a rule, zona is easy to diagnose. Zona of the limbs may sometimes be mistaken for *vesicular polymorphous erythema*, but the latter is most often mixed with bullæ and erythematous spots, and is always symmetrical, never limited to a single limb. Zona must not be confused with other zoniform eruptions, such as zoniform syphilides or zoniform nævi. It must also be determined whether zona is symptomatic of other diseases.

Treatment.—This is very simple. The vesicles, which should not be ruptured, may be covered with collodion or with zinc and starch powder. If the vesicles are ruptured and painful, zinc ointment containing a little opium or morphine may be applied. Besnier recommends linimentum calcis, covered with wool.

For neuralgic pains we may prescribe subcutaneous injections of morphine, if external applications, such as chloroform liniment, fail to relieve the pain. Electricity, in the form of the induced or continuous current, sometimes gives good results. The application of blisters along the course of the nerves has also been recommended.

Herpes of External Origin.

Herpes may originate in another way than in the preceding forms; it appears to be due to external causes, and to be sometimes of microbial origin (cases of contagion have been reported). But the external cause only acts on subjects who are predisposed (arthritics).

Herpes of external origin affects regions where the epidermis is thin and delicate, and the mucous membranes, especially the borders

of the lips, the tongue, and the genital organs. The most important variety is genital herpes, preputial or vulvar.

Genital Herpes.—In man, this occurs on the prepuce and balano-preputial furrow, more rarely on the glans and penis. In women, it occurs on the internal and external surfaces of the labia majora and minora and on the fourchette: it may extend to the anal region and inguino-vulvar fold, and even spread to the mucous membrane of the vagina and cervix uteri.

It begins with a burning sensation which lasts two or three days. The eruption consists of one or more groups of vesicles situated on an inflamed base. On the external surface of the prepuce and labia majora, herpes runs its usual course, the vesicles drying up and forming crusts; but on the inner surface of the perpuce and labia majora and at the vulvar orifice the vesicles are always excoriated, and form ulcers covered by a whitish exudation. These ulcers are circular, sometimes isolated, sometimes grouped in patches with polycyclic borders. The base of the ulcers is sometimes tumefied, but never indurated. Genital herpes may be accompanied by inguinal adenitis, but the glands hardly ever suppurate. The ulcers heal in two or three days, without leaving cicatrices. However, in confluent herpes of the vulva in uncleanly women, they may last for a fortnight; sometimes the skin undergoes a kind of hypertrophy during cicatrisation, forming raised patches similar to condylomata, but these herpetic patches are not so vegetating as syphilides, and seldom last more than a week.

FIG. 11.—Herpes preputialis. (St Louis Hospital Museum.)

An eruption of herpes often accompanies syphilitic chancre; more rarely it occurs with gonorrhœa.

In some cases herpes is accompanied by neuralgic pain, which not only affects the seat of eruption, but is also felt along the nerves which supply the affected region. This *neuralgic herpes* is perhaps a form of zona, distinct from herpes of external origin.

ETIOLOGY.—In men, genital herpes appears to be provoked by coitus; certain individuals have an attack of herpes after each coitus; others whenever they have connection with a new woman. This is essentially a *recurrent herpes*, and is often mistaken by patients for venereal disease.

In women, genital herpes also occurs after coitus, especially after excessive venery; some women develop herpes after connection with a man affected with the same. It sometimes occurs in young married women after their first sexual intercourse, or during leucorrhœa.

DIAGNOSIS.—Herpes must not be confused with traumatic fissures produced during coitus; these occur on the frænum or balano-preputial furrow in men, the vulvar orifice in women. They differ from herpes by their linear form and the absence of vesicles.

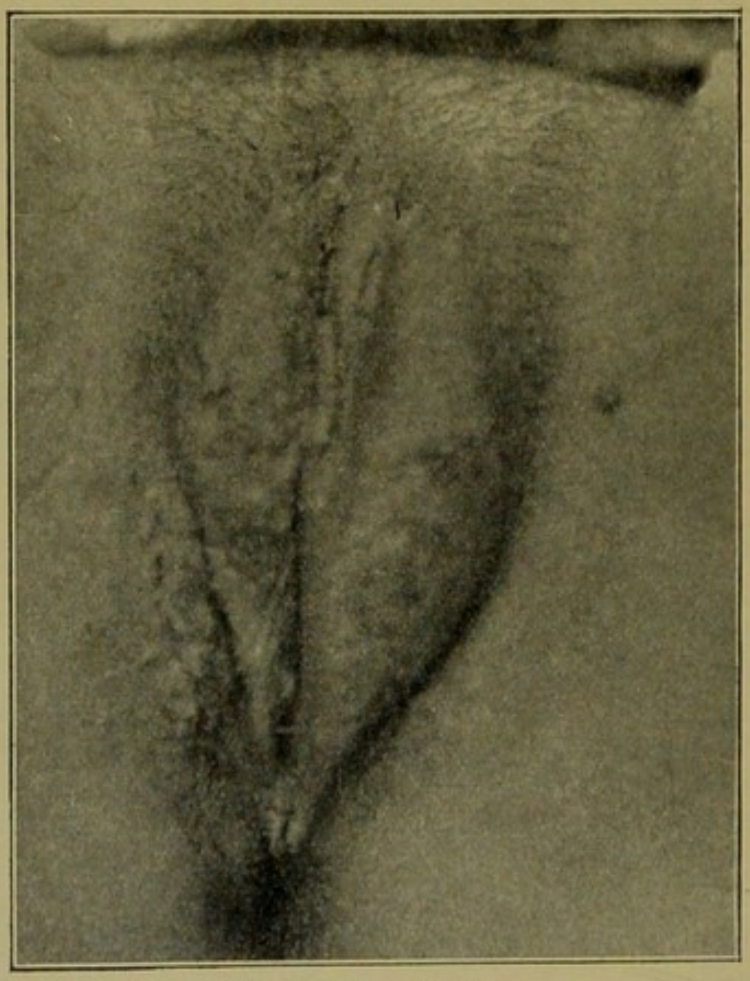


FIG. 12.—Vulvar herpes.

The erosions of *erosive balano-posthitis* are larger, more superficial and less regular than herpetic ulcers, and are accompanied by general redness of the affected part.

Simple chancre causes a deeper and wider ulceration than that of herpes; its borders are undermined, and there is abundant suppuration; the herpetic ulcer has a microcyclic and polycyclic border. Adenopathy is insignificant in herpes, while suppurating bubo is the rule in soft chancre. Lastly, inoculation is always positive in the case of soft chancre.

Herpes is sometimes difficult to distinguish from *syphilitic chancre*,

as the two lesions sometimes coexist. A patient is affected with recurrent herpes; one day a chancre appears, concealed among the vesicles; the herpes heals, but syphilis develops. The diagnosis is also difficult between syphilitic chancre and solitary herpes (Ricord's chancriform herpes); the latter consists of a single ulceration, which may be distinguished from chancre by the following characters: herpes is more superficial, more painful, and does not last so long as chancre; chancre is painless, has a longer evolution, is indurated at its base, and is accompanied by adenopathy.

Syphilitic mucous patches of the vulva are flat or vegetating erosions, instead of punched-out ulcers, as in herpes; they are painless, have a peculiar odour, and are accompanied by multiple adenopathy.

TREATMENT.—The affected part should be dusted with a mixture of equal parts of powdered starch and alum. If this fails, a lotion of nitrate of silver (2 to 5 per cent.) may be used. In herpes of the vulva, compresses soaked in saturated boric acid lotion should be applied, followed by starch and alum powder. In neuralgic herpes an ointment containing 10 per cent. of zinc oxide and 1 per cent. cocaine or morphine is useful. Sulphur waters may be of use in recurrent herpes.

Buccal Herpes.—Herpes of external origin is not uncommon on the lips, the sides and tip of the tongue, and on the rest of the buccal mucous membrane. It is due to contact with irritating substances, such as strongly spiced foods; but, in predisposed persons, herpes may occur after contact with the mildest substances.

Herpes is common in syphilitics in the first years of the disease (Fournier): it occurs chiefly on the borders of the tongue in the form of small painful erosions, which are generally mistaken for mucous patches; but the herpetic ulcerations, while recurring during long periods, have not the same individual persistency as syphilitic lesions.

This herpes of syphilitics is a *recurrent herpes*, which recurs with great obstinacy and is not affected by antisyphilitic treatment. It seems to be provoked by irritations of all kinds.

PEMPHIGUS.

The name *pemphigus* is applied to a group of cutaneous affections characterised by an eruption of bullæ, generally rather large, globular, and distended by an opaline liquid, usually preceded by red spots, on which the epidermis is raised by serous exudation. These bullæ resemble the blisters of burns.

Pemphigus may develop on the mucous membrane of the mouth,

pharynx, conjunctiva and vagina, as well as on the skin. The lesions then at first resemble cutaneous bullæ, but are soon replaced by a whitish exudation, similar to that occurring after the rupture of herpetic vesicles developed on mucous surfaces.

CLASSIFICATION.—There are several kinds of pemphigus: (1) *acute febrile pemphigus* of adults; (2) *epidemic pemphigus* of the newly born, which are perhaps two forms of the same disease; (3) *chronic pemphigus*, which is subdivided into: (*a*) benign chronic bullous pemphigus; (*b*) malignant chronic bullous pemphigus; (*c*) pemphigus foliaceus; (*d*) pemphigus vegetans; (*e*) pruriginous pemphigus, or dermatitis herpetiformis; (4) *congenital pemphigus*, a rare dermatosis.

These forms of pemphigus must be distinguished from pemphigoid lesions symptomatic of other diseases. Acute pemphigus of adults must be differentiated from the pemphigoid bullæ of bullous polymorphous erythema and bullous urticaria. Chronic pemphigus must be distinguished from bullous eruptions, due to iodides or bromides, from the bullous syphilide and the bullous eruption of leprosy (incorrectly termed syphilitic pemphigus and leprous pemphigus respectively). It is also necessary to separate from pemphigus the pemphigoid eruptions of nervous origin, including: (1) bullæ occurring along the course of nerves after neuritis or nerve injuries; (2) the bullous eruptions, sometimes observed in connection with certain diseases of the nervous system; (3) hysterical pemphigus, which occurs in women affected with hysteria.

Acute Pemphigus of Adults.

This name should be reserved for a febrile bullous affection, in which the bullæ are not mixed with erythematous spots, vesicles or papules. It has also been called *pemphigoid fever*. There are two forms—benign and malignant.

Benign Acute Pemphigus.—This begins with general symptoms; moderate fever and malaise, lasting twenty-four or forty-eight hours, and disappearing when the eruption appears. The eruption commences in the form of red spots, on which bullæ rapidly develop; these are larger than a pea and sometimes larger than a nut, and are often surrounded by a red areola. The bullæ vary in number, and may be disseminated over the whole cutaneous surface or confined to certain regions. They appear in successive crops. At the end of a week they subside, and are replaced by large squames or thin crusts. The eruption may extend to the mucous membrane of the mouth and pharynx, or to the conjunctiva. The disease lasts from one to four weeks, according to the number of eruptive outbreaks. The prognosis is benign.

Malignant Acute Pemphigus.—There is a malignant form of acute pemphigus in which microbes have been found. This form is preceded by severe general symptoms—high fever, rigors, vomiting, intense thirst and dry tongue, insomnia and delirium. These symptoms persist during the whole course of the eruption. The latter is generalised and sometimes confluent, and consists of numerous bullæ with sero-sanious or purulent contents. The patient soon sinks into an adynamic condition, and may suffer from epistaxis, intestinal hæmorrhage, or pleuro-pneumonia. Death takes place in a week or a fortnight, from progressive infection. There are intermediate forms between benign and malignant acute pemphigus.

Etiology.—Acute pemphigus, in both its forms, is an *infective exanthematous fever*, which appears to have occurred in epidemics in former times, according to the older writers. The pathogenic microbe, in two cases of malignant acute pemphigus which I examined, appeared to be a streptococcus. This malignant form of pemphigus would, therefore, appear to be the cutaneous manifestation of a streptococcal septicæmia.

Diagnosis.—Acute benign pemphigus must not be mistaken for *herpetic fever*, nor for *varicella*, the eruptive elements of which are smaller. *Bullous polymorphous erythema* differs by the presence of erythematous spots. In *bullous urticaria*, the bulla is a superadded lesion. Malignant acute pemphigus cannot be mistaken for any other disease.

Treatment.—The bullæ should be pricked with a sterilised needle, to evacuate their contents and prevent them rupturing; the parts may then be dusted with simple aseptic powders, or covered with carbolised (1 in 200) linimentum calcis and wool. In the malignant form large doses of sulphate of quinine should be given.

Pemphigus Neonatorum.

Epidemic pemphigus of the newly born generally appears from the second to the fifteenth day after birth, sometimes later; but it is never present at birth, like palmar and plantar congenital bullous syphilides. The eruption, which is preceded by fever lasting from twelve to twenty-four hours, consists of red patches, in the centre of which develops a vesicle, which at the end of twenty-four hours becomes a bulla as large as a lentil, sometimes larger. It may affect all parts of the body, but chiefly occurs on the neck, face, trunk, or roots of the limbs. It rarely affects the palms and soles. The bullæ vary from three or four to a considerable number. The eruption appears in successive crops, each bulla lasting a week; but, on account of successive outbreaks, the disease may persist for four weeks.

ETIOLOGY.—Epidemic pemphigus of the newly born is more common than that of adults. Isolated cases are sometimes observed, but it more often occurs in an epidemic form in maternities and children's hospitals. It is contagious, not only from one infant to another, but also from infant to adult. The disease is inoculable, and its pathogenic microbe is a streptococcus.

DIAGNOSIS.—It is especially with *congenital syphilis* that this form of pemphigus may be confused. The so-called "syphilitic pemphigus," which is better called *palmar and plantar papulo-bullous syphilide*, is always present at birth, and is confined to the palms and soles; it is very malignant, and usually fatal. Epidemic pemphigus, on the other hand, does not appear till two days after birth, affects the whole body, and is of benign prognosis.

In an infant recently vaccinated, a more or less generalised eruption of small bullæ sometimes appears; this "vaccinal pemphigus" appears to be nothing more than an acute pemphigus appearing at the time of vaccination.

TREATMENT.—This consists in pricking the bullæ, and applying aseptic powdered talc. Later on starch baths may be given.

Chronic Pemphigus.

Benign Chronic Pemphigus. — In this form the bullæ are identical in evolution and morphological characters with those of the malignant form; but the eruption is limited to a few bullæ, which nearly always appear in the same region, most often on the legs, and recur at irregular intervals without pain or itching. Occasionally there is only one very large bulla, which usually appears during the night; after this a second bulla appears, and so on (*pemphigus solitarius*). The absence of itching distinguishes this form from pruriginous pemphigus (*dermatitis herpetiformis*).

This affection is incurable and of indefinite duration, but the general health is not necessarily affected. Cures have been reported, but I have never seen one; however, it is essentially a benign pemphigus.

Malignant Chronic Pemphigus.—This form (*pemphigus diutinus*) begins by red spots, pruriginous or painless, which appear without any prodromal symptoms. In the centre of each spot develops a bulla, the size of a hazel nut or walnut, or even larger. The bulla is filled with clear or opalescent liquid, which, in a few days, generally becomes sero-purulent. Some bullæ subside without rupturing, but most of them rupture spontaneously or from scratching. They then produce painful ulcers covered with brown crusts; the latter fall off and leave red spots which disappear slowly. The eruption usually appears simultaneously or successively on all the affected regions;

the bullæ then appear in crops at irregular intervals, so that recent bullæ occur along with excoriations, crusts and red spots. At the period of cachexia, a certain number of bullæ may become hæmorrhagic, and the consequent ulcers gangrenous.

Chronic pemphigus often spreads to the mucous membrane of the lips, mouth, pharynx and conjunctiva; it may even begin on the bucco-pharyngeal mucous membrane. More rarely, bullæ develop in the vagina and urethra. Pemphigus of the conjunctiva sometimes gives rise to grave lesions; such as adhesion of the conjunctiva to the globe of the eye, constriction of the palpebral aperture, and sometimes even atrophy of the eye. Pemphigus of the urethra causes difficulty in micturition, and a sound introduced into the urethra brings forth pieces of ruptured bullæ. Pemphigus of the pharynx may spread to the œsophagus and cause dysphagia, necessitating catheterism.

Each outbreak of bullæ may also be accompanied by visceral complications, such as bronchitis, broncho-pneumonia, vomiting and hæmorrhagic diarrhœa.

This affection is sometimes accompanied by general symptoms: complete loss of appetite, progressive wasting, and gradual loss of strength. The patient sinks into a cachectic condition accompanied by fever. Sometimes pulmonary tuberculosis or albuminuria supervene.

PROGNOSIS.—The duration of the disease is indefinite, sometimes very prolonged; but chronic generalised pemphigus is always fatal, either from cachexia or from some complication.

PATHOLOGICAL ANATOMY.—Fatty or amyloid degeneration of the liver, spleen and kidneys has been observed, and in two cases gastric and intestinal ulceration.

DIAGNOSIS.—Chronic pemphigus is easily distinguished from the *bullous syphilide* (wrongly called syphilitic pemphigus of adults), a rare eruption, characterised by small bullæ with purulent contents, surrounded by a copper-coloured areola, and followed by peculiar cicatrices. This bullous syphilide is, moreover, only a variety of the pustular syphilide.

In *leprosy*, bullæ occur on the extremities and around the joints. The bullæ rupture and form ulcers which leave cicatrices; but these cicatrices are white and insensitive, like all the manifestations of leprosy.

The *bullous eruptions* observed in lesions of the spinal cord, after injuries to nerves, or peripheral neuritis, can be recognised by the concomitant signs of the initial disease; they are situated at the extremities, or along the course of the nerves.

Chronic pemphigus must not be confused with *hysterical pemphigus*, which coincides or alternates with hysterical seizures,

and only lasts for a few days; nor with *simulated pemphigus*, produced by the application of cantharides, liquid ammonia, etc.

ETIOLOGY.—The cause of chronic pemphigus is obscure; it has been attributed to general debility, but pemphigus is sometimes seen in persons living under excellent hygienic conditions. Some cases may be due to arthritism, for a history of rheumatism, gout, or biliary lithiasis is often obtained, either in the patients or their family.

Some authors, on the basis of changes found in the spinal cord and peripheral nerves in subjects who have died of pemphigus, regard the disease as of nervous origin; but they have perhaps confounded certain pemphigoid eruptions, in which degeneration of nerves occurs, with true pemphigus. Moreover, Kaposi and Weiss carefully examined the spinal cord in nine fatal cases, and only found diffuse sclerosis in a single case. However, it must be admitted that the theory of the nervous origin of pemphigus is still the most satisfactory.

But this nervous pathogeny does not completely explain the etiology. The primary cause of *pemphigus diutinus* appears to me to be an infection. Gibier found bacteria, arranged in chains, in the contents of the bullæ and in the blood, and Demme found diplococci, which he succeeded in cultivating. In two cases of chronic generalised pemphigus which ended fatally in a year, and both of which commenced in the mouth and pharynx, I found streptococci in the contents of intact bullæ. In these two cases the buccal and pharyngeal manifestations were exclusive, or at least predominant, for several months; the cutaneous eruption being only secondary. These researches are not absolutely conclusive, and require to be repeated.

Pemphigus Foliaceus.—This is a form of chronic pemphigus in which the eruption consists of bullæ which subside as soon as they are formed, so that the bullous period of the eruption is unnoticed and the skin becomes quickly covered with abundant, thin, large squames. The name pemphigus foliaceus is due to Cazenave.

Some authors have confounded this affection with pityriasis rubra, or exfoliating dermatitis; but these affections are primarily and exclusively squamous, while in pemphigus foliaceus the soft moist squames result from slight elevation of the epidermis by serous exudation.

ETIOLOGY.—Pemphigus foliaceus is sometimes primary, sometimes secondary. The *primary* form presents from the first the characters described above, while the *secondary* form is always secondary to chronic bullous pemphigus or to chronic eczema. Although this distinction is of little importance with regard to the evolution of the disease, I have retained it, but I shall describe the two varieties

together, for, at their period of maturity, the two forms are identical. The etiology of primary pemphigus foliaceus is unknown.

SYMPTOMATOLOGY.—In primary pemphigus foliaceus the bullæ abort, so to speak, and are replaced by squames; the eruption spreads in a few days to the whole cutaneous surface. In secondary pemphigus foliaceus the bullæ are also incompletely formed, but from time to time it is possible to find them in different places.

At its period of maturity the whole body is covered with squames, even the scalp and the palms and soles. These squames, thin, superposed, semi-detached, and rolled at their edges, fall off and are reproduced incessantly. They are sometimes so abundant that they fill the patient's bed. Moist at the onset of the disease, they become dry after a time; but here and there some soft and moist ones can always be found. Underneath the squames the skin is slightly red and eroded. Owing to irritation of the skin, especially over the elbows, buttocks and trochanters, the erosions become covered with yellow crusts, formed by the dried exudation. A peculiar characteristic is the fœtid odour. After a time, the hair, eyebrows, eyelashes and nails are shed. Quinquaud has observed cases of generalised papillomatosis of the skin in the course of this disease. Pemphigus foliaceus may affect the mucous membranes, like ordinary pemphigus; and, like the latter, it may have gastro-intestinal or broncho-pulmonary complications. The disease is not painful, but patients suffer from cold when they are exposed.

FIG. 13.—Pemphigus foliaceus. (Audry.)

PROGNOSIS.—The disease is always of long duration. After a time the patient becomes debilitated, develops anasarca, and dies either from cachexia or from some complication. According to Hardy, recovery has been observed, but this is quite exceptional.

DIAGNOSIS.—Pemphigus foliaceus must be distinguished from *chronic eczema* and *psoriasis*. When eczema becomes generalised and takes the form of secondary pemphigus foliaceus, there is no diagnosis to be made; but the characters of the former eruption, or the remains thereof on some part of the body, may reveal the origin of the disease. *Pityriasis rubra* and *exfoliating dermatitis* are exclusively squamous and always dry; thus differing from pemphigus foliaceus.

Pemphigus Vegetans.—In this form, first described by

Neumann, rupture of the vesicles is followed by the formation of fungous vegetations on the denuded surface, which secrete a fœtid liquid and become covered with thin crusts. The lesions occur chiefly in the articular folds and in the inguino-scrotal and inguino-vulvar regions. In several cases published by Köbner, the disease began on the pharyngeal or buccal mucous membrane, and did not affect the skin till some time afterwards. I have also observed two cases in which the eruption began in the mouth and pharynx, in the form of post-bullous ulcerations, which were mistaken for some months for syphilitic lesions, especially as the first cutaneous lesions following the bucco-pharyngeal eruption appeared on the genital organs. This mode of onset must, therefore, be borne in mind in order to avoid errors in diagnosis. After a few months the lesions become generalised, predominating in the groins and axillæ, and the disease assumes its usual aspect.

In two cases streptococci have been found in the liquid of intact bullæ.

Pemphigus vegetans, like chronic bullous pemphigus and pemphigus foliaceus, leads to death from cachexia. It is chiefly mistaken for syphilis.

TREATMENT.—This applies to all the preceding forms of chronic pemphigus. Arsenic is still the best remedy, although there is not much to be expected from internal medication. Sulphate of strychnine improves the general condition and digestion; iron and quinine are also beneficial. Milk diet should be prescribed when there are gastric or renal complications. Purgatives should be avoided, as they may produce obstinate diarrhœa.

Locally, linimentum calcis may be applied, carbolised or not, according to the intensity of the itching, and the parts covered with wool. I have had good results from a 2 per cent. tannin ointment. Carbolised ointments should not be applied to extensive surfaces, on account of the danger of carbolic poisoning. The bullæ should be punctured beforehand, to avoid getting torn. In some cases much relief is obtained by prolonged or continuous baths. Ulcers should be dressed with a saturated solution of boric acid, or dusted with powdered starch or talc, mixed with boric acid (10 per cent.).

Pemphigus of the mucous membranes should be treated by frequent applications of boric lotion, or oxygenated water diluted with three to five parts of boiled water. In pemphigus of the conjunctiva, warm boric compresses should be applied, and a drop of borax lotion (1 in 300) instilled into the eye night and morning.

In pemphigus foliaceus, continuous baths are especially indicated. In pemphigus vegetans, the ulcers should be dressed with boracic ointment, linimentum calcis or aristol. Köbner recommends scrap-

ing the vegetations under an anæsthetic, followed by the application of tincture of iodine.

Pruriginous Pemphigus, or Dermatitis Herpetiformis.—This dermatosis corresponds to the benign chronic pemphigus of some of the older authors (Gibert), and to the pruriginous pemphigus with small bullæ of Cazenave and Hardy. Recently it has been more

Fig. 14.—Dermatitis herpetiformis.

completely studied by Duhring, who gave it the name of *dermatitis herpetiformis.* Brocq names it *chronic painful polymorphous dermatitis* with successive outbreaks. There are, in fact, cases in which the elements are very small and resemble the vesicles of herpes, and others in which the bullæ abort and the eruption consists of various forms of erythematous elements.

Etiology.—The causes of this dermatitis are somewhat obscure. It occurs especially in nervous individuals and after violent emotion.

Winfield has found glycosuria and polyuria in some cases of dermatitis herpetiformis, and considers that these symptoms point to the nervous origin of this disease. Moreover, the cutaneous eruption may be complicated by other nervous or trophic disorders. I have seen a patient who, several years after the onset of the dermatosis, developed arthropathies, sclerodactylia and muscular atrophy, and

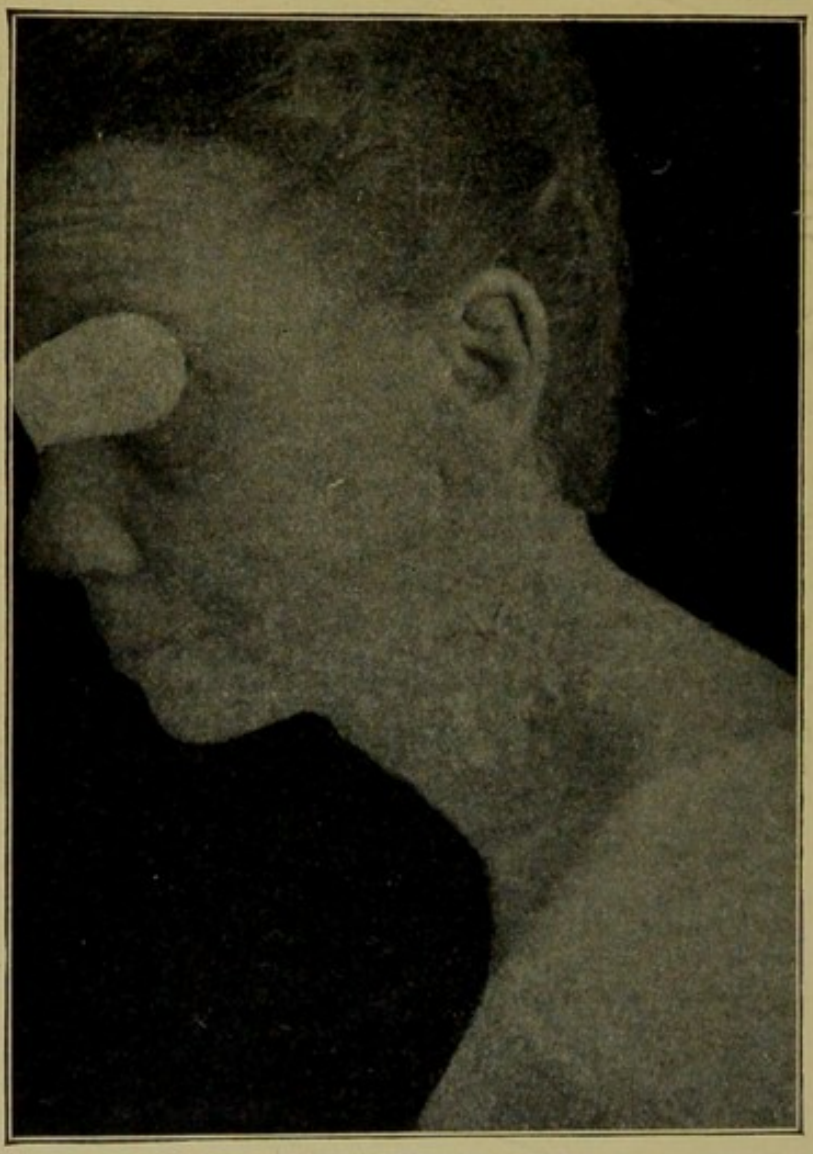


Fig. 15.—Dermatitis herpetiformis.

died after all the symptoms of a spinal affection. The nervous origin of dermatitis herpetiformis is, therefore, very probable. The hypothesis of an auto-intoxication is not in accord with the fact that the urinary excretion is usually normal. Numerous cultures and experimental inoculations with liquid from the bullæ have shown that the disease is not of a microbial nature.

In some cases an increase in the number of eosinophile cells has been found in the blood and in liquid from the bullæ; but it has

been proved by numerous observations that eosinophilia is a common phenomenon in a great number of dermatoses, and is not peculiar to dermatitis herpetiformis.

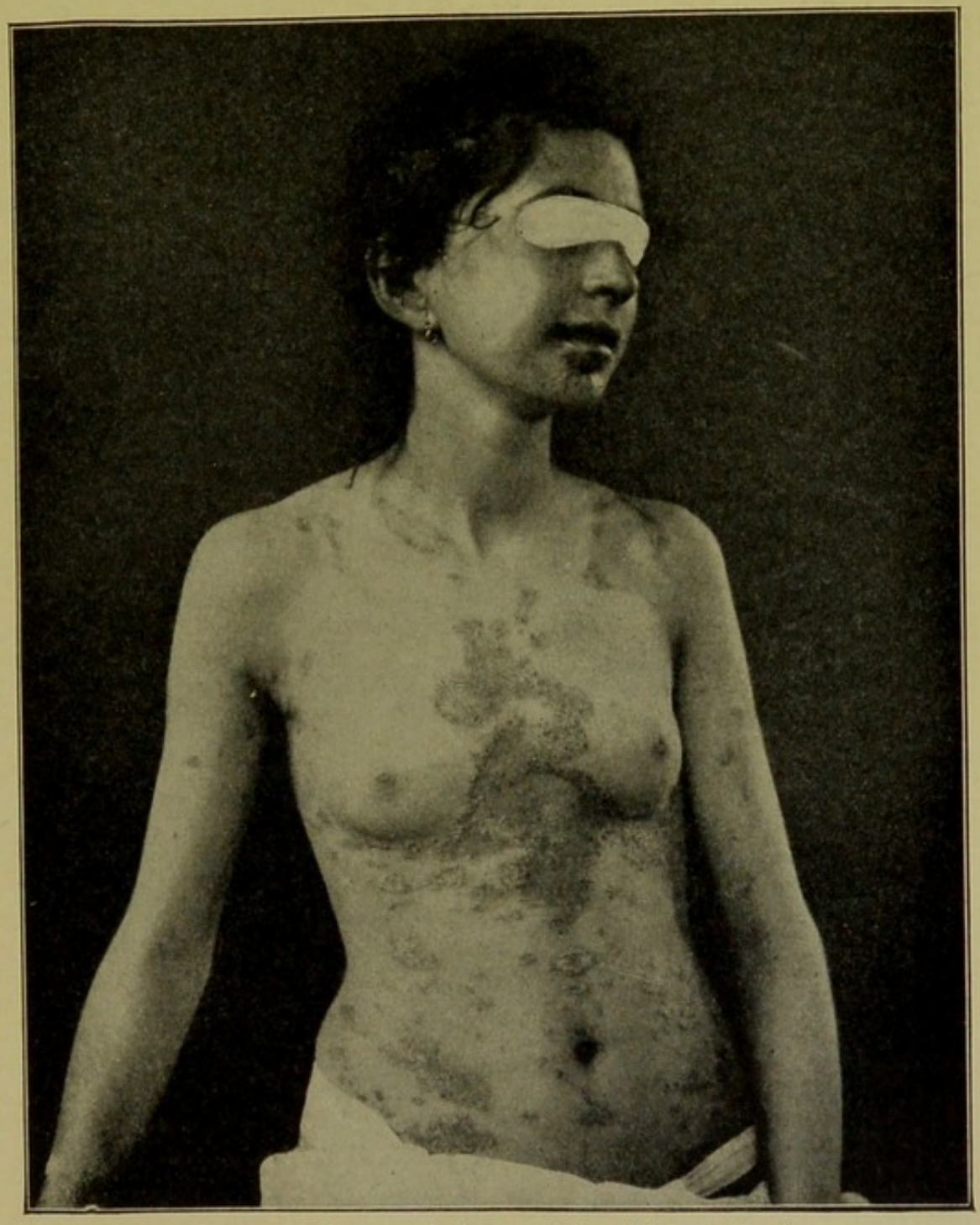


Fig. 16.—Erythemato-vesicular form of dermatitis herpetiformis.

Symptomatology.—The eruption is generally preceded by more or less intense itching, and also by tingling and burning sensations, sometimes by painful tension of the skin; these symptoms accom-

pany the eruption and persist after it. The eruption begins on the arms and legs, and gradually extends to the rest of the body symmetrically. It consists of vesicles and bullæ, varying in size and number, situated on erythematous patches, or appearing on healthy skin. These bullæ are mixed with erythematous spots on which there is no elevation of the epidermis; this is a characteristic feature. They are also accompanied, either by urticarial eruptions or by papules, which are only aborted vesicles or bullæ. Some of the vesicles and bullæ become purulent by secondary infection, others hæmorrhagic. Sometimes hæmorrhagic spots occur, chiefly on the lower limbs.

The vesicles and bullæ may rupture and leave excoriations, which become covered with crusts, or they may subside without rupturing and give place to squames. Sometimes vegetations or papillomatous excrescences develop on the excoriations, accompanied by thickening of the horny layer, especially on the feet. The eruptive elements never leave cicatrices; but when they have disappeared there is pigmentation of the skin, which persists for a long time.

According as one or other eruptive element predominates, special varieties of dermatitis herpetiformis have been distinguished, especially an erythematous variety, and a circinate erythemato-vesicular variety which simulates herpes iris.

The eruption may affect the mucous membranes, as in malignant chronic bullous pemphigus.

Evolution.—The eruption appears in successive crops at irregular intervals, and is of variable duration. During the intervals the pruritus may persist. The disease lasts for many years. During the eruption the general health usually remains good, and there is only slight fever in confluent and generalised cases. However, in some cases there may be diarrhœa, albuminuria or diabetes, and endocarditis has been reported. The disease may terminate fatally, either from some intercurrent complication, or by transformation into pemphigus foliaceus.

Prognosis.—Apart from these exceptional cases, the prognosis is not grave; the disease is seldom fatal, but it is usually incurable. However, cure is possible, and has been observed in rare cases.

Diagnosis.—Dermatitis herpetiformis differs from *polymorphous erythema* by its long duration, by its painful symptoms and itching, and by its tendency to recur. Common *urticaria* must not be confused with the urticarial eruptions which are sometimes observed in dermatitis herpetiformis; these eruptions, which are often bullous, are accompanied by other symptoms of the dermatitis. Dermatitis herpetiformis differs from *acute pemphigus* by its long duration and by the intensity of its painful phenomena; from *pemphigus diutinus*,

by its polymorphism, its successive crops, its intense itching, and by the preservation of the general health.

TREATMENT.—Milk should be prescribed; tea, coffee, and alcohol forbidden. Attention should be paid to the digestive functions. The nervous system may be calmed by valerian or other antispasmodics. Continuous warm baths and hydrotherapy are useful. Duhring recommends the administration of *arsenic* in increasing doses; but this often fails to cause any improvement. Sulphate of quinine is indicated if there is fever. Locally, to relieve the intense itching, antipruriginous lotions, sedative powders, vaseline containing 1 per cent. of carbolic acid, menthol or guaiacol may be applied, and the affected parts covered with wool.

When the inflammatory phenomena have subsided, the best application, according to Duhring, is *sulphur ointment*. This is especially useful in the vesicular, bullous, and pustular forms, but usually causes irritation in the erythematous variety. It should be rubbed into the skin with sufficient force to rupture the vesicles, and should be used as soon as the lesions begin to appear. An ointment containing 1 or 2 per cent. tannin also gives good results. The disease is favourably influenced by change of air, and by moral and hygenic treatment.

Herpes Gestationis.—This affection is identical with dermatitis herpetiformis. It is the dermatitis herpetiformis of pregnancy. As a rule, the eruption appears in the last six months of pregnancy, and disappears soon after the end of the puerperal state. After several successive attacks, coinciding with repeated pregnancies, it may persist long after accouchement. It has a great tendency to recur and to become aggravated at each new pregnancy; but occasionally a pregnancy occurs without any eruption. After several attacks the affection sometimes becomes permanent, and then persists, in the absence of pregnancies, like ordinary dermatitis herpetiformis.

The eruption begins on the limbs, sometimes in the umbilical region. It is polymorphous, and consists either of erythematous lesions, sometimes figured (erythema iris), or of papules, or of vesicles and bullæ. The evolution of herpes gestationis is the same as that of dermatitis herpetiformis; the itching is as intense and the general health as good, except for a little prostration.

In patients previously affected with dermatitis herpetiformis, the eruption has, curiously enough, been known to disappear when they became pregnant.

Herpes gestationis can only be confounded with Hebra's *impetigo herpetiformis*; but the latter is an infective, pustular disease, which always ends fatally.

The treatment is the same as in dermatitis herpetiformis.

Infantile Recurrent Dermatitis Herpetiformis.—According to

Vidal and Leloir, dermatitis herpetiformis may also occur in young children, presenting the following characters: early onset during the first years of life; persistent recurrences; maximum of attacks during the warm seasons; an eruption consisting of erythema, papules, vesicles and pustules; pruritus and painful sensations; changes in the general health, even before the eruption; progressive attenuation of the attacks at puberty; spontaneous and total disappearance, or attenuation of the eruption at adult age.

Congenital Pemphigus, or Epidermolysis Bullosa.

Congenital pemphigus includes two forms, which have by some been regarded as two distinct diseases: (1) simple hereditary traumatic pemphigus; (2) congenital pemphigus with epidermic cysts.

Simple traumatic pemphigus is exclusively bullous. It is a hereditary and familial affection, in which bullæ are produced on any part of the body after slight friction. The bullæ occur chiefly on the hands and feet, as these parts are most exposed to external irritation. The bullæ heal without leaving cicatrices, but are reproduced with great facility, especially during warm weather. This bullous process persists during life, but becomes attenuated in old age. It is an incurable affection, but does not affect the general health. The only treatment consists in attention to the hygiene of the skin.

This hereditary pemphigus is probably a trophic disorder of the skin. It is due to special fragility of the epidermis, characterised by want of adhesion between the horny and mucous layers. The Germans give it the name of *epidermolysis bullosa hereditaria.*

Congenital pemphigus with *epidermic cysts*, or *dystrophic epidermolysis bullosa*, is a rare affection, probably due to a congenital trophic disorder of the skin. The affected subjects suffer from birth from successive eruptions of discrete bullæ, few in number, transparent, sometimes hæmorrhagic, situated on the face, arms, forearms and legs. The skin is red and smooth and studded with small white points the size of a pin's head, resembling pearls. These white points are sebaceous cysts formed by obliteration of the ducts of the glands. The bullæ appear without pain or fever; after a time they disappear, leaving indelible cicatrices. They may occur on the buccal and nasal mucous membrane. The epidermic cysts may occur on the scalp. The skin of these subjects is dry and affected with keratosis pilaris; in one of Besnier's cases it was ichthyotic. The nails may also be affected.

The duration of this disease is indefinite. Kaposi states that the cysts sometimes become detached after a few months.

CHRONIC ECCENTRIC PUSTULAR DERMATITIS
(Hallopeau's Disease).

Under this name Hallopeau has described a rare dermatosis, very few cases of which have been published. I have observed one case.

The affection is characterised by the production of foci of suppuration, which commence as pustules, spread eccentrically, and

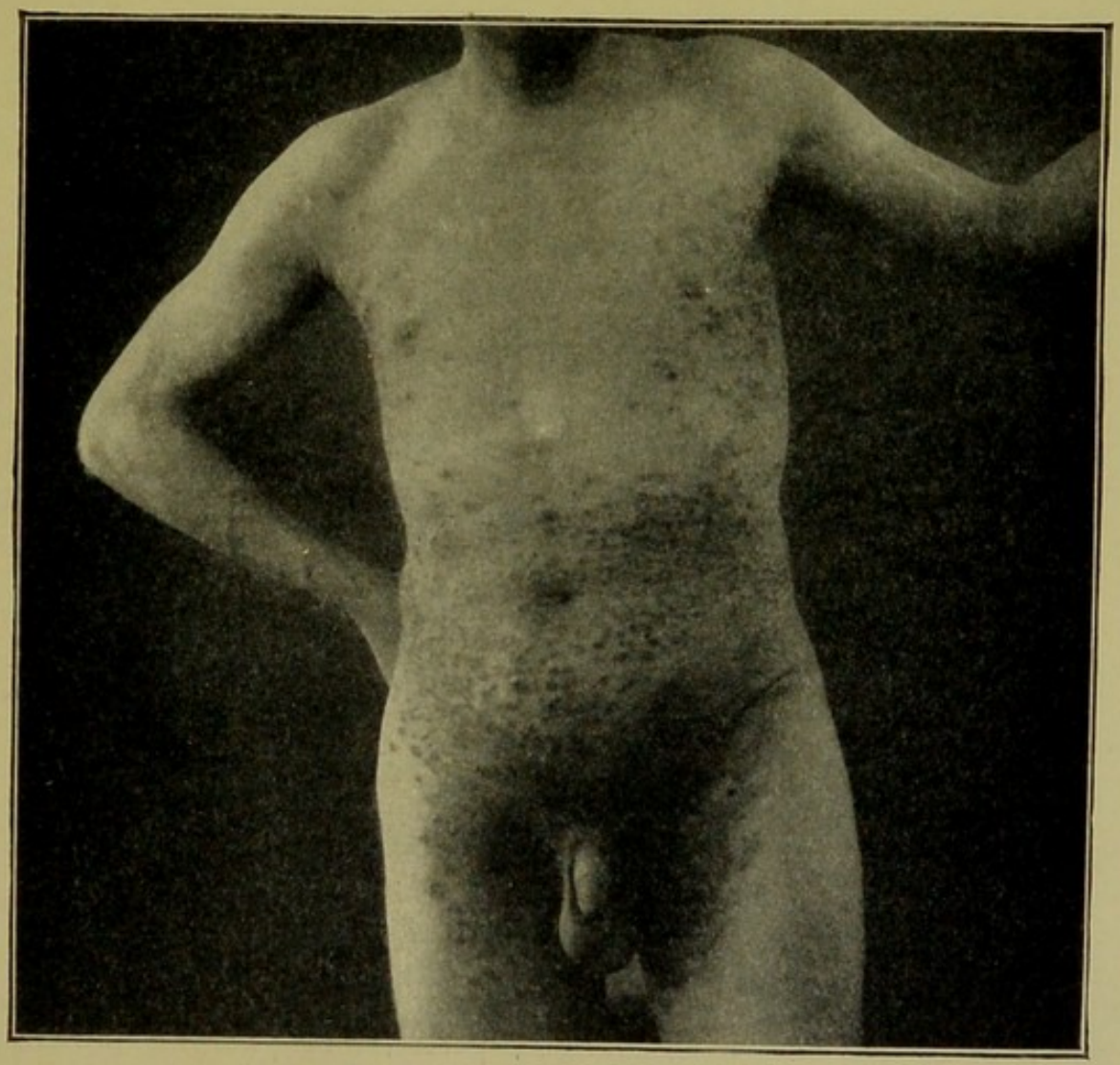


Fig. 17.—Chronic pustular dermatitis.

become united so as to form groups, the centres of which may undergo resolution. The lesions are chiefly situated on the pubis, axillæ and scalp, but they also occur on the trunk, thighs and hands, and even on the buccal mucous membrane. After the pustules have opened, the subjacent dermis undergoes vegetating

proliferation, or at any rate papular elevation. The general health is not affected.

According to Hallopeau, the disease is a pyodermatitis due to pyogenic infection. It presents analogies with the pustular form of dermatitis herpetiformis, but must be considered as a distinct disease. In the case which I observed, the cutaneous suppuration originated by successive auto-inoculations from an open suppurating gland in the groin which was consecutive to a furuncle near the anus.

TREATMENT.—The affection can be cured by sublimate dressings, the application of iodoform, iodol or aristol, and the galvano-cautery. A one per cent. solution of resorcin also gives good results.

I have described Hallopeau's disease after chronic pemphigus, because, apart from certain analogies with purulent dermatitis herpetiformis, it also presents many points of resemblance with pemphigus vegetans, especially as regards its distribution and pyogenic origin. However, it cannot be regarded as an attenuated form of pemphigus vegetans, for in the intact pustules of Hallopeau's disease, staphylococci alone are found, while pemphigus vegetans appears to be produced by a streptococcus. In my own case a culture of the *staphylococcus aureus* was obtained.

IMPETIGO HERPETIFORMIS.

This name was given by Kaposi to a disease which differs from common impetigo. It occurs usually in pregnant women, but not exclusively, for Köbner has observed cases apart from pregnancy, and Dubreuilh saw a case in a male.

This dermatosis is formed by very small yellow pustules the size of a pin's head, arranged in circinate groups and situated on a red and inflamed base. The pustules soon break, and are replaced by brown crusts. Around these develop successive and subinvolutive eruptions, concentric with one another. The disease eventually covers a large surface of the body. The eruption occurs chiefly in the groins, around the umbilicus, on the breasts, and in the axillæ. When the crusts fall off, the subjacent dermis is red, moist and infiltrated. Sometimes the tongue, palate, and pharynx present gray, circumscribed patches, depressed in the centre and surrounded by pustules.

The *prognosis* of this affection is grave. There is high fever and a bad general condition, and generally a fatal termination in a few months, either at the first attack or during a relapse. It appears to be a special infective disease.

DIAGNOSIS.—It is absolutely impossible to mistake it for *common*

impetigo. It differs from *dermatitis herpetiformis*, which is characterised by a polymorphous eruption with intense itching, while the general health is not much affected. However, according to Besnier, there are transitional forms in which the diagnosis is very difficult.

TREATMENT.—For the general symptoms, quinine and alcohol are indicated; locally, antiseptic applications, sedative powders and continuous baths.

ECZEMA.

Eczema is an inflammatory dermatosis, characterised by congestive redness and tumefaction, on which develop vesicles, small, acuminate and confluent, of very short duration, the rupture of which gives rise to the exudation of a transparent viscid liquid which coagulates in the form of crusts, followed by furfuraceous squames, and then by progressive induration of the dermis, constituting chronic eczema.

Eczema may be acute or chronic; the acute form, apart from eczematiform eruptions of external origin, is usually only an acute outbreak in the course of a chronic eczema, or the acute onset of a chronic eczema.

Acute Eczema.—This presents three periods: (1) congestion and vesiculation; (2) exudation and formation of crusts; (3) desquamation.

First period.—This lasts from twenty-four to forty-eight hours. The skin becomes red and swollen; the redness, which is accompanied by painful tension of the skin and sometimes by intense itching, becomes covered with acuminate, confluent vesicles full of clear liquid. These vesicles are sometimes large, sometimes so small that they can only be seen by a lens. Sometimes the vesicles rupture as soon as they are formed, and give rise to abundant exudation; in other cases they become absorbed without causing exudation, and desquamation appears from the first.

Second period.—The vesicles persist for a few hours or a day, then rupture and give rise to a transparent viscid liquid, which dries up and, together with epidermic debris, forms crusts, usually gray, sometimes yellow when the vesicles have suppurated, or brown when the exudation is mixed with blood as the result of scratching or excoriation.

Third period.—After a variable time, the exudation subsides and the crusts fall off, leaving a red, smooth skin. The skin then becomes covered with more or less abundant desquamated squames, which are repeatedly renewed. The redness of the skin persists for a time after the desquamation has ceased. When the vesicles do

not rupture, and their contents are simply absorbed, desquamation is finer and less prolonged.

These three periods occur together in the same subject, so that eruptive elements of different ages, consisting of vesicles, crusts, and squames, are seen at the same time.

Concomitant symptoms.—There is sometimes slight fever and gastric disturbance, but these symptoms may be absent. In some cases there is enlargement of the lymphatic glands corresponding to the eczematous regions. Sometimes furuncles appear after the eruption, due to secondary infection of the skin; these are very obstinate, and liable to recur for a long time.

Chronic Eczema.—This may follow the acute form, in which case the eruption persists and is followed by induration of the skin, or the disease may be chronic from the first. There is less redness of the skin than in acute eczema, but the itching is more intense, and is aggravated by changes of temperature, the warmth of the bed, the ingestion of spiced foods or alcoholic drinks. Chronic eczema is characterised by successive outbreaks, during which new vesicles of short duration appear. Vesicles are often absent, the exudation being produced directly, less abundant but thicker than in acute eczema, and drying in the form of crusts. Another characteristic sign is thickening of the skin, which is hard to the touch and presents fissures and superficial ulcerations, giving rise to serous exudation, or sero-purulent from secondary infection.

Sometimes chronic eczema is dry and without crusts; the skin is then rough and thickened, and covered with a mixture of papules, aborted vesicles and epidermic exfoliation. Itching, in this dry form, is even more intense than in moist eczema. This *chronic dry eczema* is allied to chronic *lichen simplex;* hence its name, *lichenoid eczema.*

Acute eczema may evolve rapidly, but as there are often repeated outbreaks, the duration of the disease may be prolonged. The duration of *chronic eczema* is indefinite, and the thickening of the skin persists for a long time, and is often followed by permanent pigmentation. From time to time recurrences are liable to occur.

VARIETIES.—Eczema presents varieties according to the form of the eruption, or according to its situation. It may be localised or generalised, but in the latter case there are always intervals of healthy skin.

Varieties according to the form of the eruption.—**Acute Eczema Rubrum.**—This may begin as such, or it may originate from a chronic localised eczema. Its onset is accompanied by fever and gastric disturbance, sometimes by restlessness and even delirium. It is preceded by general itching over the whole body. The eruption differs from that of ordinary eczema; instead of a diffuse redness,

raised red patches appear on several parts of the body, which spread and sometimes coalesce, but always leave areas which are free from eruption. These red patches are situated in the articular folds and axillæ, on the loins, elbows, inner surface of the thighs, scrotum, wrists, neck, and even on the face. On the red patches appear vesicles, which become confluent and sometimes very large; these soon rupture and discharge serous exudation, which forms crusts as in ordinary eczema. Eczema rubrum begins as a pseudo-exanthem, which evolves in two or three weeks, and is very liable to recurrences.

Nummular Eczema.—This is formed by round patches the size of a five-shilling piece, dry or slightly moist, with well-defined borders, scattered on the trunk and limbs, chiefly on the upper limbs and anterior surface of the wrists. These patches last for a long time, and are liable to recur. Nummular eczema is essentially chronic.

Channelled Eczema.—This is a simple form of nummular eczema. According to Brocq, it is formed by small round or oval patches, of a pale red colour, with well-defined edges, situated on the dorsal surface of the hands and on the limbs. Its characteristic feature is the presence of concentric grooves, visible only with a lens.

Fissured Eczema.—This may be associated with ordinary vesicular eczema, or it may occur by itself. It is constituted by superficial fissures of variable depth, zigzag in form, and crossing each other so as to enclose areas of healthy skin. There is serous exudation similar to that of ordinary eczema. As the fissures heal, others form, thus prolonging the affection. This form of eczema affects chiefly the legs, thighs, forearms and axillæ, and the lips and anus, where it is very painful.

Crackled Eczema.—This is a variety of fissured eczema, characterised by shallower fissures, arranged in a regular manner in the form of network of red meshes enclosing areas of healthy skin, instead of the irregular zigzag arrangement of fissured eczema.

The above forms, in which vesicles are entirely or almost absent, serve as arguments for those who consider eczema as a polymorphous dermatosis, and not as an exclusively vesicular affection.

Impetiginous Eczema.—This results from secondary infection, and is due to the transformation of eczematous vesicles into pustules by inoculation with the pyogenic microbes of impetigo. Like impetigo, it occurs chiefly in young lymphatic subjects. The crusts are not so yellow as those of impetigo. It is less pruriginous than ordinary eczema, and more easily cured.

Lichenoid Eczema.—This variety is constituted by a mixture of vesicles and papules; most of the vesicles are arrested in their development and remain as papules. This form occurs in patches on the outer surface of the limbs. (Vide *Lichen simplex.*)

Psoriasiform Eczema.—In this form the eczematous patches are covered with white epidermic scales, not so well defined as those of psoriasis, and presenting a slight exudation, which never occurs in psoriasis. The patches are very pruriginous, while those of psoriasis cause little or no itching. Psoriasiform eczema occurs chiefly on the legs and forearms.

The *eczema marginatum* of Hebra is not an eczema; it is sometimes a variety of cutaneous trichophytosis, sometimes a dermatitis caused by the *microsporon minutissimum* (*Erythrasma*).

The *eczema folliculorum* of Malcolm Morris is an affection the nature of which is not determined.

Varieties according to situation.—**Eczema of the Scalp.**—The scalp is often the seat of eczema. Sometimes it is ordinary eczema with abundant exudation, which glues the hair together and causes intense itching; this form is generally an extension of eczema of the face; but occasionally it develops *in situ*, in which case there are nearly always patches of eczema behind the ears. Sometimes the eczema is impetiginous, generally in young children, and may cause loss of hair and even patches of alopecia. Lastly, it may be dry from the first; the vesicles abort, and their contents are absorbed, after which there is desquamation. Eczema is diagnosed from *pityriasis* of the scalp by the presence of eczematous patches behind the ears; also, the squames of pityriasis are finer and less adherent than those of eczema. In *psoriasis* of the scalp the squames are thicker and stratified, and have a characteristic pearly appearance; psoriasis is limited to the scalp, while eczema generally extends to the forehead or behind the ears. *Ringworm* is distinguished from dry eczema of the scalp by the broken hairs, and the presence of spores on microscopic examination.

Eczema of the Beard and Eyebrows.—This has also been called *eczema pilaris*. It is characterised by bright redness, scanty exudation, and abundant desquamation, the squames of which surround the base of the hairs. If the inflammation extends to the hair follicle it constitutes *sycosis*, which we shall study later. Eczema pilaris must not be confounded with parasitic *pityriasis alba*, which is a second stage of ringworm; in this the skin is not so red, and the microscope shows the presence of spores. *Lupus* is distinguished by its purple colour and by the formation of cicatrices.

Eczema of the Lips.—This may result from the extension of eczema of the face, or it may occur by itself. It occurs in several forms. There is first of all an *orbicular eczema*, limited to the cutaneous part of the lips; this is dry and radiating, with transverse fissures following the folds of skin; it affects both lips, which become painful, indurated, cracked and bleeding, and are covered with squames. The other forms are *elephantiasic eczema* of the

upper lip and eczema of the red part of the lips. Elephantiasic eczema is seen in young lymphatic subjects, and is generally associated with chronic coryza, of which it is the consequence. The upper lip is swollen and hard; the skin is red, moist and covered with crusts. The second form (described by Bateman and Rayer as psoriasis and pityriasis of the lips) begins in the form of red patches, followed by general redness and continual desquamation of the epithelium. The latter becomes yellow, thickened and cracked, and is detached in large flakes, under which new epithelium forms, which is detached in its turn. This form of eczema, which is always rebellious, is quite distinct from the transitory inflammation of the lips produced by cold, or that occurring in acute diseases. It must also be distinguished from *mucous patches* of the lips, which are opaline, and from *perlèche*, which affects the commissures of the lips, especially in children.

Eczema of the Nostrils.—In lymphatic subjects, chronic eczema may occur on the edges of the nostrils and in the nasal cavity, often in conjunction with elephantiasic eczema of the upper lip. The nasal mucous membrane is swollen and covered with crusts.

Eczema of the Eyelids.—This also occurs in lymphatic subjects and is generally bilateral, affecting the external and internal surfaces and free border of the eyelids (eczematous blepharo-conjunctivitis); at the external commissures there are fissures. The eyelids are red, swollen, painful and indurated, and deformed by ectropion. The ocular conjunctiva is red, and covered with small vesicles. The inflammation causes disturbance in the nutrition of the cornea, and by extension to the lachrymal canals produces epiphora. The free border of the eyelid is red, and covered with crusts and vesico-pustules, which rupture and give rise to a discharge which glues the eyelids together, especially during the night.

Eczema of the Ears.—This may be acute or chronic. Acute eczema is generally impetiginous, and occurs in children and adolescents. The ear is red, hot, swollen and painful; the corresponding lymphatic glands are usually enlarged. Chronic eczema is dry, with abundant desquamation; it affects the concha and external auditory canal (eczematous otitis). The squames accumulate in the canal and on the tympanic membrane, causing deafness, which is increased when the integument becomes thickened so as to cause constriction of the passage.

Eczema of the Breast.—This is often observed during pregnancy and lactation, and in lymphatic subjects. The eczematous patches are round and moist; painful fissures occur on the nipple and areola. This form is very tenacious, and leaves pigmentation behind it which lasts for a long time. Eczema of the breast must not be mistaken for *scabies*, which is common in this region, nor for

Paget's disease, in which the surface is dark red and granular, and free from fissures or crusts.

Eczema of the Umbilicus.—This is common in young lymphatic girls; in adults, obesity and uncleanliness predispose to it. It is obstinate, and gives rise to abundant fœtid exudation with the formation of crusts and painful fissures. It may be mistaken for *mucous patches*, but the latter are free from squames or crusts, and have a characteristic odour.

Eczema of the Anus.—This affects the anus and its periphery. It is bright red, with scanty exudation. It is accompanied by very intense itching, sometimes intolerable, and aggravated by walking or by lying in bed, and is generally complicated by lesions produced by scratching. There are also painful fissures in the folds of skin, which cause acute pain during defæcation. Eczema of the anus must not be confused with *simple pruritus*, in which there are no skin lesions. *Mucous patches*, which are common about the anus, are of an opaline tint, and are never covered with crusts; they tend to become vegetating, and have a peculiar odour.

Eczema of the Scrotum and Penis.—This occurs in adults and in old men. It is sometimes limited to the scrotum, but may extend to the penis, perineum, internal surface of the thighs, anus and intergluteal fold. The skin is red, moist and swollen, covered with crusts and later on with squames; itching is intense. When the glans and prepuce are affected, they become swollen and œdematous; this occurs especially in diabetes, and is due to irritation caused by the sugar in the urine.

Eczema of the Vulva.—This affects the labia majora and labia minora, the urethral meatus, and the vagina as far as the cervix uteri. It may extend to the perineum and pubis. It is characterised by redness, swelling, exudation, crusts, fissures, and scratch marks due to the intense itching. If the vagina is affected, there is a discharge which must be distinguished from leucorrhœal and gonorrhœal discharges. The eczematous discharge is of a gray colour, while leucorrhœal discharge is white, and gonorrhœal discharge yellow. Eczema of the vulva is common in diabetes.

Eczema of the Articular Folds.—This occurs in the axilla, groin, fold of the elbow, and popliteal space. It is very obstinate, on account of constant movement of the joint. In the axilla, it is often complicated by inflammation of the sudoriparous glands and abscess (*hydrosadenitis*).

Eczema of the Legs —Varicose Eczema.—Eczema of the legs is most often associated with varicose veins. It forms dark red patches, sometimes moist and covered with crusts, sometimes dry and scaly. It is very rebellious on account of the varices, which retard the circulation. The patches are followed by pigmentation,

which is often permanent. Sometimes the skin undergoes papillomatous transformation: it then becomes covered with small elevations, the epidermis becomes thickened and fissured, and its striæ more marked than in the normal state. Varicose eczema often ends in ulcer of the leg.

Eczema of the Hands and Feet.—Eczema of the dorsum of the hand and foot is accompanied by small moist fissures in the interdigital spaces. On the palmar and plantar surfaces the thick epidermis impedes the production of vesicles, so that squames only are present, underneath which the epidermis is red and shiny.

FIG. 18.—Eczema of the back of the hand. (St Louis Hospital Museum.)

FIG. 19.—Horny eczema of the palm. (St Louis Hospital Museum.)

Chronic palmar and plantar eczema is characterised by thickening of the skin, which becomes hard and horny, with painful fissures in the cutaneous folds. This form of eczema may be mistaken for palmar psoriasis (which is very rare), with the palmar syphilide, or with essential keratosis. In *psoriasis*, the patches have more definite borders, and the fissures are dry, while those of eczema are moist; the squames of psoriasis are thick and white, while those of eczema are thinner and of a gray or yellowish colour. In the *palmar syphilide*, the patches are of a coppery-red colour with well-defined borders, and do not cause itching. In *essential keratosis*, the epidermis alone is affected, and there is no eczematous eruption.

Eczema of the Nails.—This is often associated with eczema of

the hands and feet or other regions, but sometimes it occurs alone. The nails are dry, thickened and brittle, sometimes more or less regularly striated, sometimes pitted, sometimes rough and irregular. A thick layer of horny substance develops underneath the nail. The skin around the nail is often red, swollen, and painful, and covered with vesicles (*eczematous perionyxis*), a condition which may lead to shedding of the nail. This perionyxis may also exist by itself; it is a very obstinate affection, and very painful.

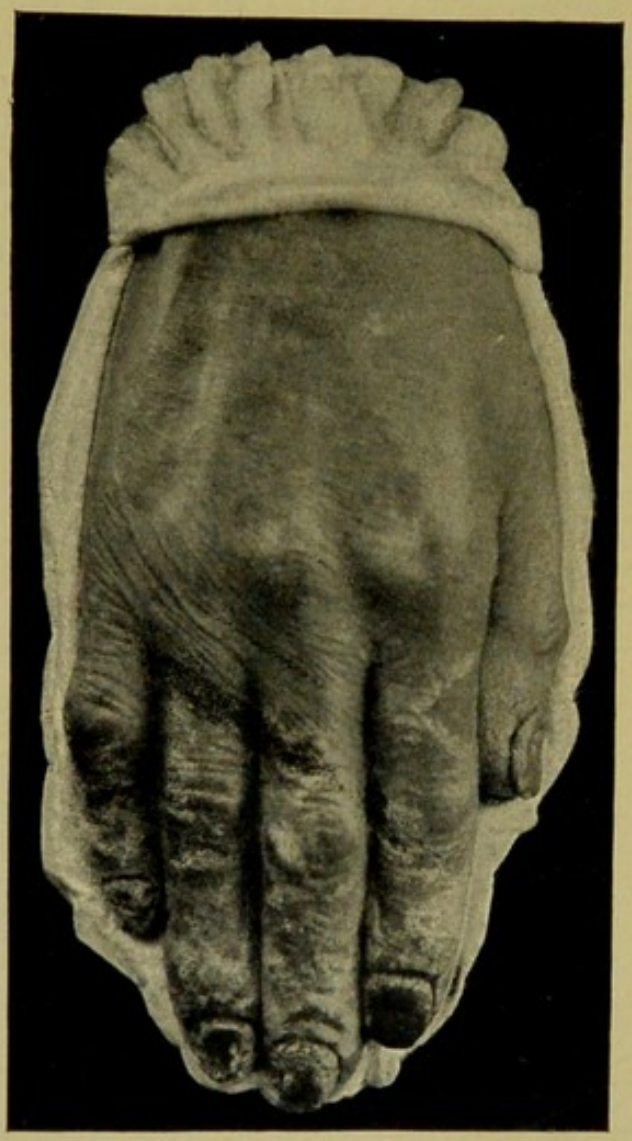


FIG. 20.—Eczema of the nails. (St Louis Hospital Museum.)

FIG. 21.—Horny plantar eczema. (St Louis Hospital Museum.)

Eczema of the nails, when it occurs alone, is difficult to diagnose, especially from psoriasis of the nails. Some dermatologists consider pitting of the nails as characteristic of psoriasis. Bazin held that transverse striation occurred in psoriasis, longitudinal striation in eczema. But these lesions occur equally in psoriasis and eczema.

Eczema of Mucous Membranes. — Apart from eczema of the mucous membranes already studied (nasal, vaginal, ocular), eczema may affect the buccal and pharyngeal mucous membrane, which

becomes red, swollen, painful, eroded, and covered with soft, moist squames. The diagnosis is difficult in the absence of eczema on some other part of the body.

Some authors regard *exfoliating marginate glossitis* as a form of eczema; but this affection is a quaternary manifestation of congenital syphilis (Gaucher).

DIAGNOSIS.—We have already discussed the diagnosis of eczema affecting certain regions; we have now to consider the diagnosis of eczema in general.

Acute eczema of the face may at first be mistaken for *erysipelas*, but the presence of vesicles and the absence of fever and glandular enlargement will settle the diagnosis. Acute eczema rubrum may simulate *scarlatiniform erythema*, but the patches of eczema are isolated, tumefied, covered with exudation and crusts, and there are intervals of healthy skin. In *exfoliating dermatitis* the squames are larger and more abundant than in eczema rubrum; the eruption is generalised, while it is never so in eczema. *Herpes* is distinguished from eczema by the form of the vesicles, which are larger, isolated or grouped on a red surface, and generally situated near the mucous orifices; herpes does not cause exudation, but forms brown crusts, which are never followed by the prolonged desquamation observed in eczema. *Sudamina* and *miliaria* are two affections connected with diaphoresis, and differ from eczema in the absence of itching and crusts. *Dysidrosis* is also a sudoral affection, affecting chiefly the hands, and characterised by the absence of crusts. Eczema is easily distinguished from *dermatitis herpetiformis*, in which the vesicles are isolated and much larger than those of eczema. The lesions of *scabies* are somewhat similar to those of eczema, but they chiefly occur in the interdigital spaces, on the genital organs and breasts, and form characteristic burrows. Eczema may sometimes be mistaken for *impetigo*, especially when the eczematous vesicles become pustular from secondary infection. The crusts of eczema are easily distinguished from those of *seborrhœa*, which are soft, fatty, and easily detached, leaving no trace of inflammation. *Pityriasis simplex* is characterised by fine squames, which are not preceded by exudation or crusts; we have already mentioned the differential characters of pityriasis capitis and eczema of the scalp. It is impossible to confuse eczema with *pityriasis rosea*, which consists of rose-coloured finely squamous spots. *Trichophytic herpes* is distinguished from nummular eczema by the presence of the characteristic spores; it consists of a few red patches, which extend eccentrically and cause but little itching. In *psoriasis* there is never exudation; the squames are thick, white and dry, and are situated on well-defined patches, while the patches of eczema are more diffuse. Acute *lichen simplex* has several points of resemblance with eczema, but the

papules are never transformed into vesicles, and there is no exudation, the crusts sometimes observed being due to scratching. *Lupus erythematosus*, when it is covered with squames, may simulate chronic eczema, but differs in its deeper infiltration of the skin, in the absence of vesicles, and in the presence of cicatrices, which never occur in eczema. In *mycosis fungoides* the tumours are often preceded by an eczematous eruption, which must be distinguished from true eczema.

Pathological Anatomy.—The lesions of eczema affect the dermis and the epidermis.

In the dermis there is congestion of the papillary vessels, which explains the redness of the skin and abundant serous exudation, which is characteristic of eczema. The papillæ are also infiltrated by proliferating connective tissue cells: in acute eczema, these consist only of embryonic cells; in chronic eczema, connective tissue cells and fibres predominate, and produce induration and thickening of the skin. This infiltration of the papillæ increases their dimensions, and becomes the point of origin of the papillomatous formations which occur especially in eczema of the lower limbs.

In the epidermis the Malpighian layer is studded with shining vesicles, resulting from cavitary transformation of the cells due to vesicular alteration of the nucleus. At a more advanced stage, in the centre of some of the interpapillary columns, are seen broken-down cells opening into one another and forming a cavity containing a sort of fine reticulum formed by the walls of the broken-down cells, in the meshes of which accumulates serous liquid exuded from the papillary vessels. This cavity constitutes the vesicle of eczema. The cavitary changes in the Malpighian cells cause disturbance in the nutrition of the epidermis and in its keratinisation; the cells of the stratum corneum are nucleated; the stratum granulosum and stratum lucidum have disappeared.

Another change, which I described some time ago, consists in complete detachment of the epidermis. In the normal state, intimate union between the epidermis and the papillary layer is maintained by a hyaline layer or basement membrane, formed by condensation of amorphous matter from the papillæ. In eczema this union no longer exists, and the epidermis is separated from the papillary layer.

The histological changes in the mucous membranes are the same as in the skin, viz., vascular congestion, small-celled infiltration, and vesicular transformation of the cells. Ulceration occurs more rapidly on mucous membranes than on the skin.

Visceral changes are generally absent; but, in some cases of generalised eczema, visceral congestions (cerebral or pulmonary) may occur, either spontaneously or as the result of bad treatment, and even end fatally, as occurs after extensive burns.

ETIOLOGY.—There are two etiological doctrines: the French, according to which eczema is of internal origin; and the German, which regards eczema as always of external origin; the latter theory is that of Hebra and his school.

According to the French school, eczema is a diathetic eruption due to a general disposition of the organism, a humoral alteration, which Bazin called arthritism, and which modern researches attribute to a disturbance of nutrition (Bouchard). In fact, eczema is an *autogenous toxidermia* due to auto-intoxication of the organism by nitrogenous extractive matters (Gaucher). Analysis of the urine of eczematous patients shows a diminution in urea, an increase in extractive matters, and a constant diminution in the proportion of nitrogen. Most French dermatologists hold that external causes only play a secondary part, acting on predisposed subjects.

We have seen (*eruptions of external origin*) that the so-called artificial eczemas are in reality *eczematiform dermatoses*, and not true eczemas. These eruptions disappear under simple treatment, or even without treatment, as soon as the irritant cause is removed. When the eruption persists after removal of the cause, it is because the patient has the eczematous diathesis. If this condition, depending on defective nutrition, was not present at the origin of all forms of eczema, the same irritant cause would produce the same eruption in all subjects, which is not the case.

Among the *exciting causes* of eczema are spiced or easily fermentable foods, meat broths, fish, game, pork, alcoholic drinks, tea and coffee; but these only produce eczema in eczematous or arthritic subjects. Other causes act only by creating conditions which are favourable to the development of eczema. The *eczema of dentition*, which occurs in some children at the first dentition, is a diathetic eczema which develops because the children are constitutionally eczematous; it affects the forehead, cheeks, and external border of the back of the hand and wrist. Pregnancy, the menopause, and violent emotions are also exciting causes of eczema. We have already seen that nerve lesions may produce eczematous eruptions, but these must be distinguished from true eczema.

We need not dwell on the theory of the *microbial origin* of eczema; this idea does not rest on any sound basis.

PROGNOSIS.—Eczema, even in its acute form, is a disease which is difficult to cure and liable to recur. Chronic eczema is a protracted, tenacious, painful affection, subject to acute exacerbations; but, with rare exceptions, it is not a fatal disease. In some cases eczema may become the point of origin of secondary pemphigus foliaceus, or secondary pityriasis rubra, which is of grave prognosis.

TREATMENT.—In eczematous patients the elimination of toxic matters should be assisted by laxatives and diuretics, and an attempt

made to improve the nutrition. Local treatment should only be commenced when general treatment has been instituted.

General Treatment.—This must be studied separately in acute eczema and chronic eczema.

Acute Eczema.—First of all, frequent laxatives should be prescribed. Diuretics are also indicated; the best is milk. Milk and milk foods should form the basis of the diet in eczematous patients. Alkaline waters, such as Vichy, should be prescribed, to modify nutrition. Arsenic should be avoided in acute eczema and in all acute exacerbations of chronic eczema. All foods containing fermented or fermentable substances should be forbidden; such as game, pork, duck, tripe, fermented cheese, fish, crustacea and molluscs: also, spiced meats, strong sauces, meat soups which are rich in extractive matters, wine and liqueurs, coffee and tea. The following may be allowed: well-cooked white or red meat, roast, grilled or boiled; fresh cheese; all vegetables except cabbage, sauerkraut, and asparagus; all fruits except strawberries and raspberries.

Chronic Eczema.—Laxatives should be prescribed, and digestive fermentation checked by charcoal cachets and benzonaphthol. The diet should be the same as in acute eczema, milk being the basis, especially in children. Nutrition should be modified by lithia, or alkaline waters such as Vichy. In gouty subjects the diuretic waters of Contréxéville are useful. In lymphatic subjects iodide of iron, sulphur waters, and cod-liver oil are indicated. At the squamous stage of the disease, in the absence of acute exacerbations, *arsenic* may be prescribed in the form of Fowler's solution (3 to 8 drops at each meal), or arseniate of sodium (1 to 4 milligrammes daily). When there is much itching, tincture of musk, or preparations of valerian or asafœtida may be given.

Local Treatment.—This differs entirely in acute eczema and chronic eczema; in the former, it should be simply sedative; in the latter, it should cause modifications in the epidermis. In the acute exacerbations of chronic eczema the treatment is the same as in acute eczema.

Acute Eczema.—In the acute stage of vesiculation and exudation, compresses of aseptic gauze soaked in boiled water should be applied; ointments and cataplasms are contra-indicated, as they only increase the exudation. The treatment of acute eczema by boiled water, which I have employed for many years, undoubtedly gives the best results.

In some cases of acute eczema limited to small patches, the latter may be painted with a solution of picric acid (1 per 100), and then covered with wool.

In the second stage the crusts may be removed by starch poultices.

The starch should be mixed with tepid water; boiling water, or, better, boracic lotion, is then added. The starch grains burst, and form a jelly on cooling. Hardy and Colson recommend sheets of gutta-percha instead of poultices; but these sometimes cause pustules and excoriations; they should only be used in cases where the application of poultices is difficult; for instance, in eczema of the scalp and face, when a cap or mask of gutta-percha may be worn. Gauze compresses soaked in boiled water and covered with gutta-percha are preferable to poultices, and still more to applications of gutta-percha.

In the third or squamous stage, ointments of oxide of zinc or subnitrate of bismuth (1 in 10) should be used, made with vaseline, fresh or benzoated lard, neutral glycerole of starch, or a mixture of one part lanolin with two of vaseline. When there is much itching, menthol may be added to the ointment (1 or 2 per 100). Starch or bran baths are useful in this stage, but rather harmful in the stage of exudation.

When acute eczema shows signs of passing into the chronic state, astringent ointments may be prescribed; calomel (10 per 100), tannin (6 per 100), subacetate of lead (4 per 100), salicylic acid (1 per 100) with oxide of zinc or starch, oil of cade (10 per 100) with glycerole of starch.

Chronic Eczema.—To loosen the crusts and reduce inflammation, moist compresses or starch poultices may be applied. Alkaline baths, and especially starch baths, are very useful; also daily spraying with boiled water. When the skin has been well cleansed, substances should be applied which cause alterative changes in the epidermis and dermis. The best alterative is *oil of cade;* but this must be pure juniper tar and not coal tar. Oil of cade has a disagreeable odour and stains the linen, but it is one of the best curative agents for chronic eczema. It may be mixed with glycerole of starch, vaseline, or lard (from 10 to 50 per cent.). In chronic eczema I generally use an ointment which combines the reducing properties of oil of cade, sulphur, and salicylic acid with the anti-pruriginous property of camphor, and which is thick enough to serve as a protective covering. This is the formula:—

Powdered camphor, sifted Powdered salicylic acid Precipitated sulphur, sifted	aa 1 part
Oil of cade	10 parts
Oxide of zinc	20 „
White vaseline	30 „

This ointment has the advantage over other preparations of oil of cade, in not causing tar acne.

In chronic eczema with much induration, pure oil of cade may be

applied. Vidal recommends styrax ointment mixed with olive oil (1 part in 2 or 3). Naphthol ointment (2 to 5 per 100) is useful in certain chronic localised forms. When ointments cannot be used, astringent lotions may be prescribed: aqueous solutions of tannin (3 to 10 per 100), sulphate of zinc (1 to 3 per 100), nitrate of silver (2 per 100), perchloride of mercury (1 in 200); the latter should not be continued for more than three or four days, and should only be used in cases of limited area, on account of the possibility of mercurial intoxication. Natural sulphur and mineral waters are also useful.

Treatment of the Different Varieties of Eczema.—In *acute eczema rubrum* baths should not be prescribed, still less ointments. Simple powders only are indicated.

In *fissured* and *crackled eczema*, in the exudative stage, simple powders should be used, and when the exudation has ceased, starch poultices to allay the inflammation. Later on, ointments of oxide of zinc or tannin may be used.

In *impetiginous eczema*, boracic compresses should be applied till the crusts are got rid of; sprays may then be prescribed, especially when the face is affected, followed by boracic ointment.

In *eczema of the scalp*, the crusts may be removed by means of starch poultices, or, better still, a gutta-percha cap, changed day and night. In men the hair can be cut short; but in women who object to this sacrifice, the crusts may be removed by almond oil or by tepid spraying. After this sulphur ointment should be used (5 to 10 per 100), or mild preparations of oil of cade. The scalp tolerates oil of cade fairly well, but it can only be used in persons with brown hair, on account of the staining.

In *eczema of the beard*, zinc or calomel ointments should be applied, after the crusts have been removed by means of moist compresses.

In *eczema of the lips*, crusts can be removed by strips of gutta-percha; after this, ointments of oxide of zinc, tannin, yellow oxide of mercury (1 per 100), salicylic acid (1 in 60 to 1 in 30), may be applied. On the borders of the lips, cold cream should first be applied; afterwards one of the above ointments.

In *eczema of the nostrils*, nasal irrigation with boric acid lotion is indicated; if this cannot be tolerated, boracic, tannin, or calomel ointments may be introduced into the nostrils on wool tampons.

In *eczema of the eyelids*, moist compresses should be applied, followed by yellow oxide of mercury ointment (1 per 100).

In *eczema of the auditory canal*, injections of boiled water, followed by wool tampons soaked in boracic ointment, or mild applications of oil of cade, should be prescribed.

In *anal, vulvar* and *scrotal eczema*, after the inflammation has

been reduced by lotions, warm sitz baths or moist compresses, ointments of tannin or extract of rhatany (4 in 30), or calomel should be applied. Fissures may be cauterised with sulphate of copper, and when these have healed, the compound oil of cade ointment previously mentioned, or even pure oil of cade, may be applied. Rectal pruritus can be treated with suppositories of morphia ($\frac{1}{3}$ grain) or belladonna ($\frac{1}{6}$ grain); external itching by Gowland's lotion:—

Hydrarg. perchlor. } Ammonii chlor. }	. . .	aa 1 part
Aqua laurocerasi	. . . .	40 parts
Aqua	. . . . .	1000 „

or menthol ointment (1 per 100).

In *vaginal eczema*, the inflammation should first be reduced by poultices introduced through a speculum: after this, boracic or tannin ointment.

In *eczema of the articular folds*, which resembles erythema intertrigo, a lotion of perchloride of mercury (1 in 500) may be applied; after this the surfaces should be dusted with starch and separated by fine lint.

Palmar and plantar eczema require energetic treatment, after the inflammation has been reduced by poultices or moist dressings. Hebra's method consists in applying an alcoholic solution of soft soap spread on flannel, which is kept on during the night and washed off in the morning. This is continued for several nights; if it sets up inflammation, it must be replaced by glycerole of starch.

Eczema of the nails should be treated by salicylic ointment (5 per 100) or pure oil of cade.

Eczema of the mucous membranes requires treatment by gargles of boiled water or Vichy water. Spiced foods, strong alcoholic drinks, and tobacco should be avoided.

SEBORRHŒIC ECZEMA.

Seborrhœic eczema differs from simple seborrhœa in the presence of inflammatory phenomena beneath the seborrhœic matter; from ordinary eczema in the presence of seborrhœic matter, and absence of serous exudation. Bazin called it *acneic eczema;* Erasmus Wilson, *lichen annulatus serpiginosus;* and Payne, *seborrhœa circinaria.* Unna (1887) gave it the name of *seborrhœic eczema,* which has since been generally adopted.

It may occur on all parts of the body, but is chiefly observed on the sternal and interscapular regions of the thorax, from which it extends laterally. It appears in the form of small, well-defined,

circular patches, of a yellowish red colour, covered with seborrhœic crusts; the centre of the patches is pale red and slightly squamous, sometimes squamous enough to give a certain resemblance to psoriasis. The patches are pruriginous, and may present excoriations produced by scratching. They extend more or less regularly at one part of their circumference, while they heal at another part, uniting with neighbouring patches to form various figures. The name *lichen annulatus serpiginosus*, although incorrect as regards the term *lichen*, is very descriptive of the appearance of the patches, especially when confluent. The centre of these large patches is pale red to normal in colour, and is covered with squames and seborrhœic crusts; the borders present the same appearance as those of the small patches. There is no formation of vesicles and no exudation, so that the term eczema is not quite appropriate.

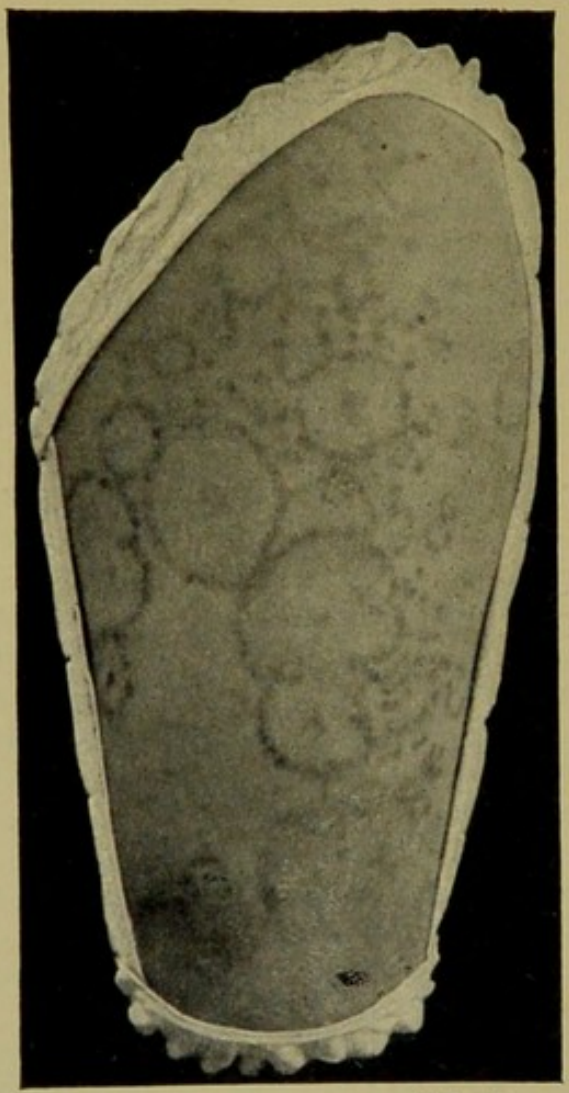


FIG. 22.—Seborrhœic eczema. (St Louis Hospital Museum.)

On the face and on smooth parts, seborrhœic eczema presents the same aspect as on the trunk, sometimes with a more inflammatory character. It occurs on the forehead, on the sides of the nose and cheeks, on the border of the eyelids, and in the external auditory canal, where it is very tenacious. In the beard and hairy parts of the face, it occurs in the form of reddish patches covered with crusts and squames; but there is no shedding of the hairs.

Unna has extended the term seborrhœic eczema to all exudative and crustate eczemas of the scalp, but I cannot accept this view. It is true that pityriasis of the scalp, as the result of scratching and cutaneous irritation, may be complicated by eczema, but this is a true eczema with exudation and crusts—a *post-seborrhœic* ordinary eczema, not a seborrhœic eczema. True seborrhœic eczema of the scalp is characterised by circular patches covered with seborrhœic

crusts and squames, resting on inflamed skin, but without exudation. It remains for a long time unnoticed; then, after a few months, the crusts and squames increase, the hair falls off, and similar patches appear on the forehead and temples, and often on the trunk.

Seborrhœic eczema may occur in the axillæ, in the form of extensive patches with red borders, and a bistre-coloured centre, slightly squamous and very pruriginous; also in the groins, inguino-scrotal and inguino-vulvar folds, and about the anus. These patches are moist, but form few crusts.

Seborrhœic eczema may also affect the palms and soles, in spite of the absence of sebaceous glands. This localisation tends to confirm Unna's view that the sweat glands play a certain part in the production of seborrhœa. In these parts the eruption appears in the form of small squamous spots, resembling isolated squamous papules of psoriasis; but the squames are fatty instead of dry and pearly white.

Seborrhœic eczema may affect the free border of the lips, where it assumes a crustate form and produces squames and fissures. It has also been observed on the genital organs.

Cases of generalised seborrhœic eczema followed by death have been reported; but these cases, if they exist, are certainly post-seborrhœic eczemas. Simple seborrhœic eczema is generally cured in a few weeks, but is very liable to recur.

Pathological Anatomy.—According to Unna, seborrhœic eczema is a chronic inflammation of the skin, characterised by elongation of the papillæ, cell infiltration in the superficial layers of the dermis, especially around the papillary and subpapillary vessels, dilatation of the lymphatic spaces around the vessels, and proliferation of the epithelium of the pilo-sebaceous and sudoriparous glands. In the epidermis there is considerable karyokinetic proliferation of the layer of prickle cells, disappearance of the granular layer, and a peculiar œdematous condition of the basal corneal layer. By the aid of osmic acid, Vidal and Leloir have shown fatty infiltration of the lower parts of the Malpighian layer, papillæ, sudoriparous glands, and some parts of the dermis.

Etiology.—This is still very obscure. Is it a special disease quite independent of ordinary eczema, or is it an eczema modified by the production of seborrhœa? These questions are at present undecided. Seborrhœic eczema affects specially the male sex, and subjects with a greasy skin. The use of flannel next the skin favours its development.

According to Unna, seborrhœic eczema is a microbic disease due to a *morococcus*, but the results of inoculation with these morococci are not convincing. Seborrhœa is a constitutional affection; seborrhœic eczema is the result of inflammation of the *seborrhœic*

skin, and may be produced by external irritation, but its microbic nature has not been proved. In reality, seborrhœa and seborrhœic eczema are diathetic affections.

DIAGNOSIS.—Seborrhœic eczema is easily distinguished from *true eczema*, in which there is exudation and the crusts are not fatty. In *pityriasis rosea* there are red spots covered with squames, but never fatty crusts; also the eruption is more generalised. However, in some cases of seborrhœic eczema dry squames predominate so much that the diagnosis from pityriasis rosea is very difficult; in fact, these may be transitional forms between the two affections.

Psoriasis, when the squames are small, may be confused with seborrhœic eczema; but the squames of psoriasis are dry and not fatty, and when scratched leave a white mark; also, there may be typical patches of psoriasis on other parts of the body. The diagnosis is more difficult when psoriasis occurs in a seborrhœic subject, and the patches are covered with fatty crusts; also, when seborrhœic eczema becomes so squamous as to simulate psoriasis. Moreover, there may be transitional forms between seborrhœic eczema and psoriasis, which, no doubt, represent what Devergie called *eczematous psoriasis*.

Acne rosacea and *impetigo* are easily distinguished from seborrhœic eczema; acne rosacea may coincide with it. Seborrhœic eczema differs from the *papulo-squamous syphilide* in the presence of seborrhœa of the scalp and the absence of infiltration of the dermis underneath the patches.

TREATMENT.—The scabs and crusts should first be removed with soap or soft soap. If there is much irritation after this, ointments of zinc oxide or calomel should be applied. But, as soon as possible, sulphur (1 in 30) or yellow oxide of mercury (1 in 60) ointments should be used. In some cases an alcoholic solution of sublimate (1 in 500) may be used instead of ointments. The patient should wear fine linen instead of flannel next the skin.

PITYRIASIS SIMPLEX.

This is characterised by more or less abundant furfuraceous desquamation, preceded or not by pale redness, but without papular elevation of the skin. It was regarded by Hardy and others as a form of eczema (*squamous eczema*), but is now generally connected with seborrhœa, and may be called *pityriasic dry seborrhœa*.

Pityriasis Simplex of the Body.

On the body, and especially on the face, it forms irregular patches, varying in size from a sixpence to a five-shilling piece,

sometimes scattered over the whole body, but more commonly limited to the cheeks, forehead and beard. It is covered with furfuraceous squames which are easily removed by scratching; hence the name *dandruff*. The squames are white, situated on a white or pink surface, and are constantly renewed. There are no vesicles nor crusts. There is sometimes some itching.

DIAGNOSIS.—By the above characters pityriasis is easily distinguished from *eczema*. In *psoriasis*, the squames are thick, pearly white, and situated on a red base. *Ichthyosis*, a congenital disease of indefinite duration, is either general or affects large surfaces; the squames are large, very adherent, and imbricated. *Pityriasis versicolor* forms large patches, chiefly situated on the chest; the squames are of café-au-lait colour, and contain spores of the *Microsporon furfur*. Another affection which may be confused with pityriasis simplex is *trichophytic pityriasis alba*, which is a phase in the evolution of ringworm of the beard; but in pityriasis simplex the hairs do not break off when pulled; in trichophytic pityriasis the hairs break easily, and are surrounded by a powdery sheath formed by spores and squames; also, ringworm may be present elsewhere.

TREATMENT.—The diet should be the same as in eczema. Local irritation by shaving, etc., should be avoided. Tepid water should be used, instead of cold, for washing. A lotion of salicylic acid may be prescribed:

Salicylic acid	1 part
Alcohol	10 parts
Water	90 „

Pityriasis, or Pityriasic Dry Seborrhœa of the Scalp.

This is a very common affection, generally known as scurf or dandruff. It remains for a long time unnoticed by the patient, and manifests itself by the formation of small squames, which fall spontaneously or after scratching, and are replaced by others. The affection is exclusively squamous and the hairs are dry. Seborrhœa of the scalp may be present at the same time, constituting an intermediate form between pityriasis and seborrhœa; in this form there are small yellow crusts, squames, and excoriations from scratching, the itching being sometimes intense. The skin underneath the squames and seborrhœic crusts is normal or slightly reddened.

This *pityriasis capitis* is an affection of long duration, which becomes progressively worse if not checked by early treatment. It extends to the hair follicles, and causes loss of hair, at first on the vertex and above the forehead, leading to more or less complete alopecia; in advanced cases there is no hair left except at the back and sides of the head (*alopecia pityrodes* of Pincus).

There is a benign form of pityriasis capitis which occurs after severe diseases such as enteric and other infective fevers. This is easily cured by stimulating lotions.

DIAGNOSIS.—Pityriasis capitis must be distinguished from *squamous eczema* of the scalp; in the latter, eczema is nearly always present elsewhere, especially behind the ears, and the squames are preceded by exudation and crusts; the squames are more adherent, and the subjacent skin is slightly thickened; lastly, there is never alopecia. In *psoriasis* of the scalp the squames are thicker and more adherent, forming patches underneath which the skin is red; scratching often causes slight bleeding; the patches extend beyond the scalp and form a characteristic border along the forehead; alopecia is absent, and psoriasis can be found in other parts of the body. *Ringworm* differs from pityriasis in being circumscribed instead of diffuse, forming more or less circular patches; the hairs are full of spores, and broken, or break easily when pulled upon.

ETIOLOGY.—Some authors regard pityriasis as a manifestation of arthritism; others consider it a microbic disease (Unna, Melassez, Sabouraud). Melassez described spores, but Vidal claims to have found identical spores in other dermatoses. However, Lassar and Bishop produced alopecia in rabbits by covering them with a paste containing squames from pityriasis capitis. If these facts are confirmed, prophylactic measures should be taken against pityriasis capitis, as against alopecia areata. Nevertheless, there is no proof that pityriasis is contagious, and I continue to regard it as a diathetic affection.

TREATMENT.—The general and dietetic treatment are the same as in eczema.

Local treatment consists first of all in cleansing the scalp, removing the squames and fatty matter. In men the hair should be cut short, but in women such a sacrifice is not so easy to obtain, and the treatment is consequently more difficult. The scalp may be washed with a tepid decoction of soap bark once a week. In brunettes, tar soap may be used. Every day or every other day the scalp may be treated with solution of bicarbonate of soda (5 to 10 per 100), or the following lotion:—

Borax	6 parts
Sulphuric ether	6 „
Distilled water	100 „

When the scalp has been thoroughly cleansed by the above measures, *sulphur preparations* are indicated in the form of ointments, powders, or lotions. Sulphur ointment (30 to 40 grains to the ounce of vaseline) should be applied to the scalp every night; or powdered sulphur mixed with simple dusting powder (5 to 10 per

100). In the morning the scalp should be washed with an alkaline lotion. Sulphur lotions may be used instead of ointments; the latter have the disadvantage of increasing the greasiness of the scalp, which is often affected with fatty seborrhœa at the same time as pityriasis. The following lotion is useful:—

Precipitated sulphur (sifted) . .	6 parts
Camphorated alcohol . . .	5 „
Water	100 „

Other lotions which may be used are perchloride of mercury (1 in 1000 in water or alcohol) or formol (40 per 100). If there is much itching, chloral may be added to the perchloride lotion (2 per 100).

LICHEN SIMPLEX.

Lichen simplex is characterised by an eruption of acuminate papules, closely applied to each other, and covering a varied extent of surface. Lichen simplex is closely allied to eczema, especially to lichenoid eczema (*eczema papulosum seu lichenoides* of Hebra), so much so that Hardy and Hebra considered it as a papular form of eczema. Moreover, lichen has the same etiology, and the lesion results from a morbid process which is almost identical with that of eczema; but in lichen the process does not go as far as exudation and vesicular elevation of the epidermis.

Lichen simplex may occur on all parts of the body, but it chiefly affects the forearms and dorsal surface of the hands, the neck and shoulders, the external and posterior surfaces of the legs, and the internal surface of the thighs. In some cases it is generalised.

Symptomatology.—Lichen simplex may be acute or chronic, the latter form being more common.

Acute lichen simplex.—This is formed by small, red, hard papules, juxtaposed on a varied extent of surface, and accompanied by intense itching and a burning sensation, which is aggravated at night by the heat of the bed. The eruption generally appears in the spring or summer, usually without any general symptoms. The apex of each papule is often excoriated and covered with a small brown crust. After three or four days the papules subside, and are followed by slight desquamation; but as there are successive crops of papules, the disease may last for two or three weeks, or even longer. In aged and debilitated subjects the papules are sometimes ecchymotic (*lichen lividus*). Acute lichen simplex presents great analogies with the *strophulus* of infants.

Chronic lichen simplex.—The papules, instead of lasting three or four days, persist indefinitely. They are pale red or colourless, and

form more or less extensive patches. The largest papules are in the centre of the patches, the periphery consisting of a kind of diffuse infiltration, causing thickening, induration and dryness of the skin, which persists after cure, rendering the natural folds and furrows more apparent. The eruption appears in successive crops during several months or even years. Itching is intense, but still more so in a variety of the disease known as *lichen agrius*. This variety, sometimes of long duration, is characterised by bright red papules situated on an erythematous base. The itching is aggravated by warmth, especially by the warmth of the bed, and causes the patients to scratch themselves furiously with the nails or with any hard object that is handy, the papules thus becoming excoriated, bleeding, and covered with brown crusts. Painful fissures also occur in the articular folds.

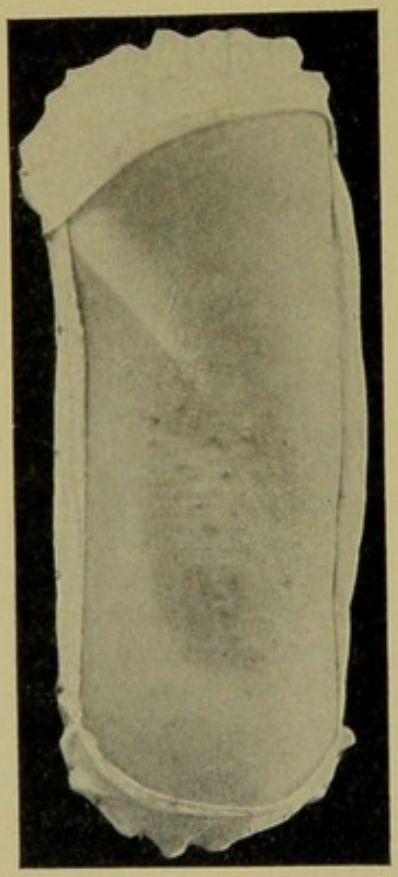


FIG. 23.—Chronic lichen simplex. (St Louis Hospital Museum.)

ETIOLOGY.—Lichen simplex is a manifestation of arthritism, and has the same diathetic cause as eczema. Arthritic subjects with a nervous temperament are more liable to lichen than to eczema. Lichen may be provoked by excesses at table, alcoholic excess, etc., but these causes only act in predisposed subjects. Papular eruptions caused by drugs and those of exclusively external origin are lichenoid eruptions, not true lichen.

DIAGNOSIS. — It is unnecessary to dwell upon the diagnosis between lichen and eczema; the one is a papular affection, the other vesicular. Lichen simplex may be mistaken for *scabies*, but the latter is generally polymorphous, seldom exclusively papular, and the papules are associated with burrows in the interdigital spaces, etc., containing the acarus. *Chronic prurigo* is distinguished from lichen by its isolated papules, which are excoriated by scratching, and covered with blood crusts. The *papular syphilide*, consisting of small acuminate papules, resembles lichen simplex, but the syphilitic papules are coppery red, and not accompanied by itching or inflammation.

PROGNOSIS.—Acute lichen is a mild affection, while chronic lichen is as obstinate as eczema.

TREATMENT.—In *acute lichen*, laxatives, starch baths, and the application of simple powders are sufficient.

In *chronic lichen* the internal treatment is the same as in eczema.

With regard to arsenic, it should not be given in acute lichen, but may be prescribed in inveterate forms of chronic lichen, except during acute exacerbations. To relieve itching and nervous irritability, tepid douches applied to the spine are useful; internally, antispasmodics, such as valerian, etc., may be given. Externally, the itching may be relieved by a warm solution of perchloride of

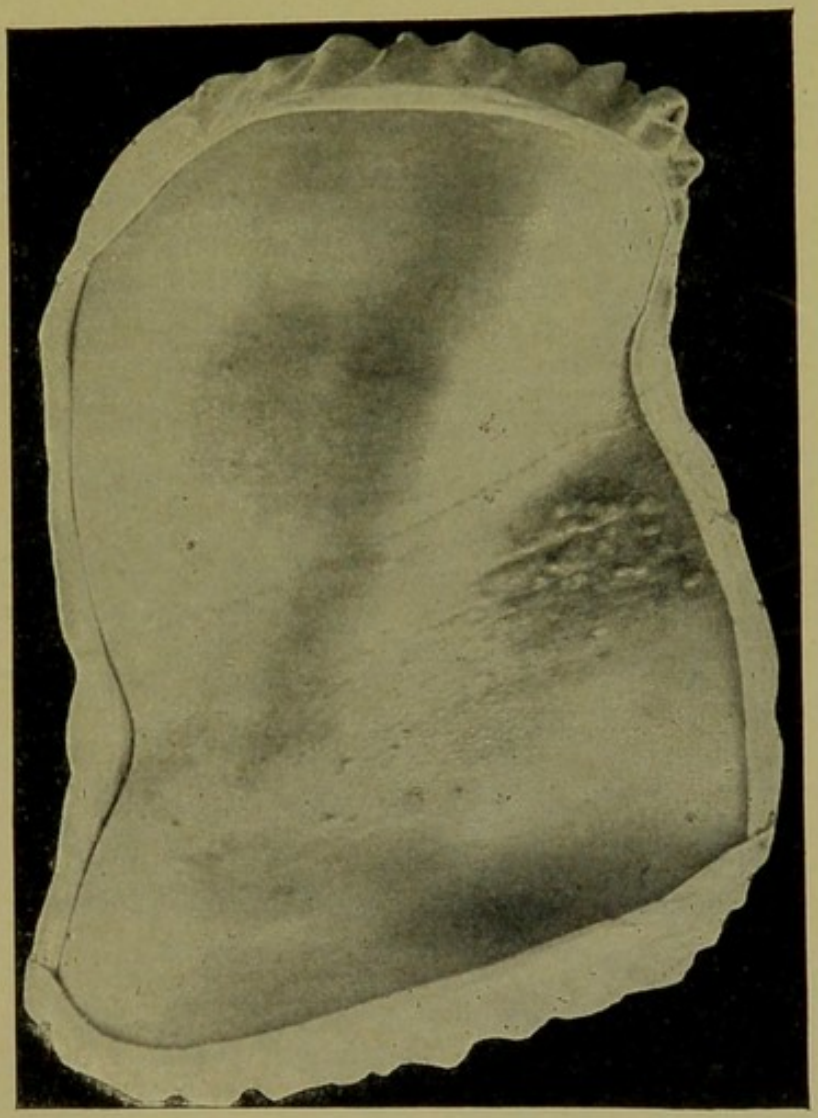


Fig. 24.—Chronic lichen simplex. (St Louis Hospital Museum.)

mercury (1 in 1000), followed by dusting powders. Menthol ointment (1 per 100) is also useful. As soon as the inflammation has subsided, salicylic ointment (1 or 2 per 100), or oil of cade, pure or diluted according to the extent of surface to be treated, are indicated. High frequency currents are also useful in chronic lichen simplex, as in all lichenoid thickenings of the skin. Sedative mineral waters are also of some service.

LICHEN PLANUS, OR LICHEN RUBER PLANUS.

Lichen planus, individualised by Erasmus Wilson, is characterised by hard, dry papules of a bright red or lilac colour, generally polygonal, flat, shiny, and slightly depressed in the centre; sometimes isolated, sometimes grouped in patches.

SYMPTOMATOLOGY.—The eruption is generally preceded by more or less itching. At first the papules resemble pin points; they increase to the size of a millet seed and then remain stationary;

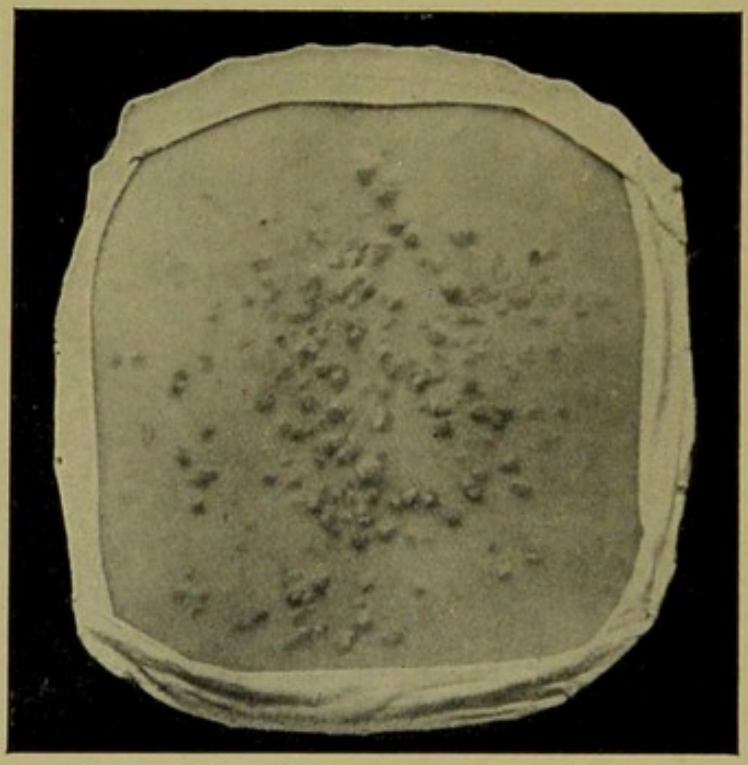


FIG. 25.—Lichen planus. (St Louis Hospital Museum.)

some papules preserve their original minute dimensions. They are always solid, and never undergo vesicular or pustular transformation; they are polygonal in form, sometimes irregularly rounded, flat, smooth and shiny on the surface. They are often umbilicated in the centre, owing to the presence of a hair follicle the hair of which has disappeared, or a glandular orifice. Their colour varies from deep red to yellowish red, often with white points or ramifying streaks in the centre. The surrounding skin is generally normal in colour, sometimes congested and thickened; sometimes it has an urticarial tendency. The papules do not desquamate, except at an advanced stage in their evolution, and then only slightly. In rare cases, Caspary and Kaposi have observed vesicles and bullæ in lichen planus. Itching is seldom absent; sometimes it is severe enough to

disturb sleep (*lichen pruriginosus*). The degree of itching is generally proportional to the nervous irritability of the subject. Scratching gives rise to fresh developments of the eruption, and papules can be seen to form along the lines produced by the nails on the skin.

Lichen planus may occur on any part of the body, but its seats of predilection are the anterior surface of the forearms and wrists, the antero-external surface of the legs, the thighs, loins, hips, abdomen, neck and genital organs. It is less common on the hands and feet. In one of my cases the nails showed trophic changes (pitting and roughness), no doubt of the same nervous origin as the cutaneous eruptions. In rare cases it has been observed on the forehead and scalp.

The papules may be scanty (*discrete lichen*), or numerous and even generalised (*diffuse lichen*). Sometimes they form complete or incomplete rings with healthy skin in the centre (*circinate lichen*). In other cases they form more or less regular patches with thickening of the skin, which in their turn may coalesce to form larger patches, but are nearly always surrounded by isolated papules with typical characters. The appearance of these patches sometimes resembles a mosaic.

Evolution. — Lichen planus has a slow evolution, and may remain localised for a long time in one region. Usually, it appears in successive crops, the old patches healing in the centre while new papules appear at the periphery. Sometimes there is no new peripheral crop of papules, and the patches heal through their whole extent, leaving pigmented spots which gradually fade. This disappearance is spontaneous, and independent of treatment. In some cases the papules, after they have disappeared, leave a brown, depressed cicatricial macule which gradually fades. To this form I have given the name of *pigmentary atrophic lichen planus*.

Lichen planus is not accompanied by any severe general symptoms. Patients are sometimes affected with dyspepsia, neuralgia, migraine, or excessive nervous irritability, but these manifestations are due to the same cause as the dermatosis itself, and are not symptoms produced by the eruption.

Regional Varieties. — **Palmar and Plantar Lichen.** — On the palms and soles the eruption is sometimes constituted by isolated papules, covered with a thick layer of horny epidermis, which becomes detached and leaves a depression in the place of each papule; these depressions give a sieve-like appearance to the affected region. In other cases the papules are confluent and the epidermis is detached in large flakes, underneath which the surface is red and painful. The diagnosis is then very difficult, unless some characteristic papules are found around the patch.

Lichen of Mucous Membranes. — Lichen planus affects the dermo-papillary mucous membranes. On the mucous membrane of the cheeks, between the rows of teeth, the eruption somewhat resembles a white streak produced by nitrate of silver; this raised streak is covered with small papules which result from proliferation of the corium. On the tongue, lichen planus affects the upper surface and sides, in the form of opaline patches resembling leucoplakia, but covered with indurated papules. Lichen planus may also occur on the glans penis, vulva and anus. I have observed it on the conjunctiva and tonsil.

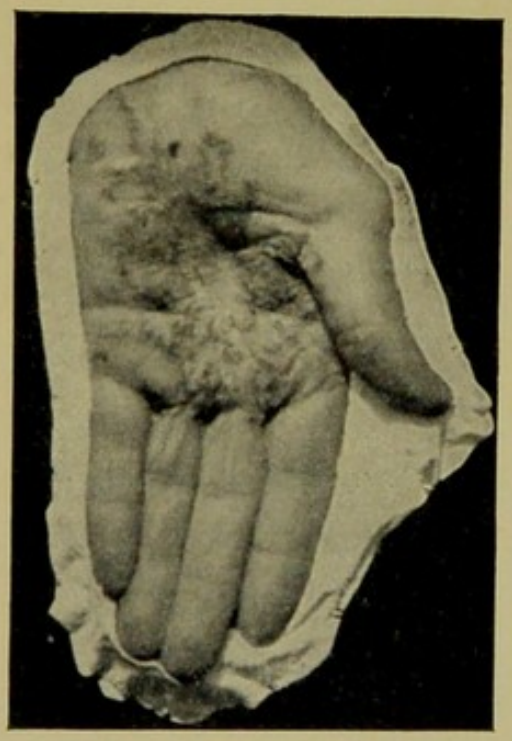


Fig. 26.—Lichen planus of the palm. (St Louis Hospital Museum.)

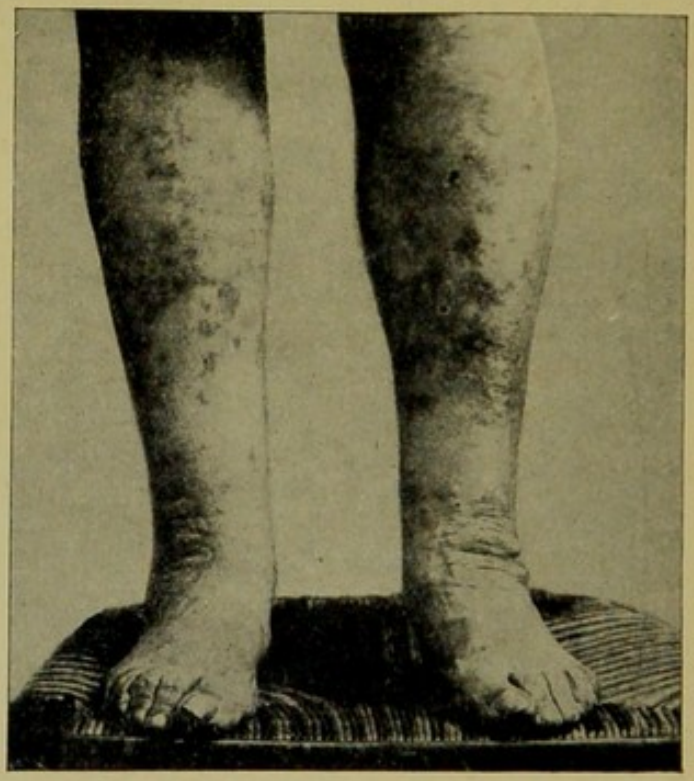


Fig. 27.—Lichen planus of the legs. (Audry.)

Varieties. — **Acute Lichen Planus.**—Instead of being chronic from the first, lichen planus may have an acute onset. The evolution of this form is rapid, and nearly the whole of the body is affected. The papules are red, sometimes much larger and more raised than in chronic lichen, and form patches by confluence. Itching is intense. Between the patches the skin is congested. After this acute onset, the affection usually pursues the chronic course of ordinary lichen.

Horny Lichen Planus. — This is always circumscribed, and usually affects the antero-external surface of the leg, where it forms thick patches of indefinite duration. The patches are irregularly rounded, rough, and studded with small depressions; they have a darker colour than ordinary lichen, and present interlacing lines on

the surface. The papules which enter into their formation can hardly be distinguished, except at the periphery, where characteristic isolated papules may often be found. There is nearly always intense itching.

Lichen Verrucosus is a more pronounced form of the preceding variety.

Hypertrophic or Tuberous Lichen (*Lichen obtusus* of Unna). —In this form the papules are large and smooth, sometimes round, sometimes flat, of a red or brown colour, and cause but little itching. This eruption is rare, usually circumscribed, and always of nervous origin. Kaposi has described cases in which the papules were large and arranged in lines (*moniliform* lichen). Lastly, there is a form described by Kaposi as *lichen planus atrophicus*, and by Hallopeau as *sclerous lichen*, characterised by faintly coloured patches, the centres of which have a cicatricial appearance, and which are situated chiefly on the anterior surface of the forearms. According to Hallopeau, this appearance is due to atrophy of the papillæ with sclerosis of the dermis.

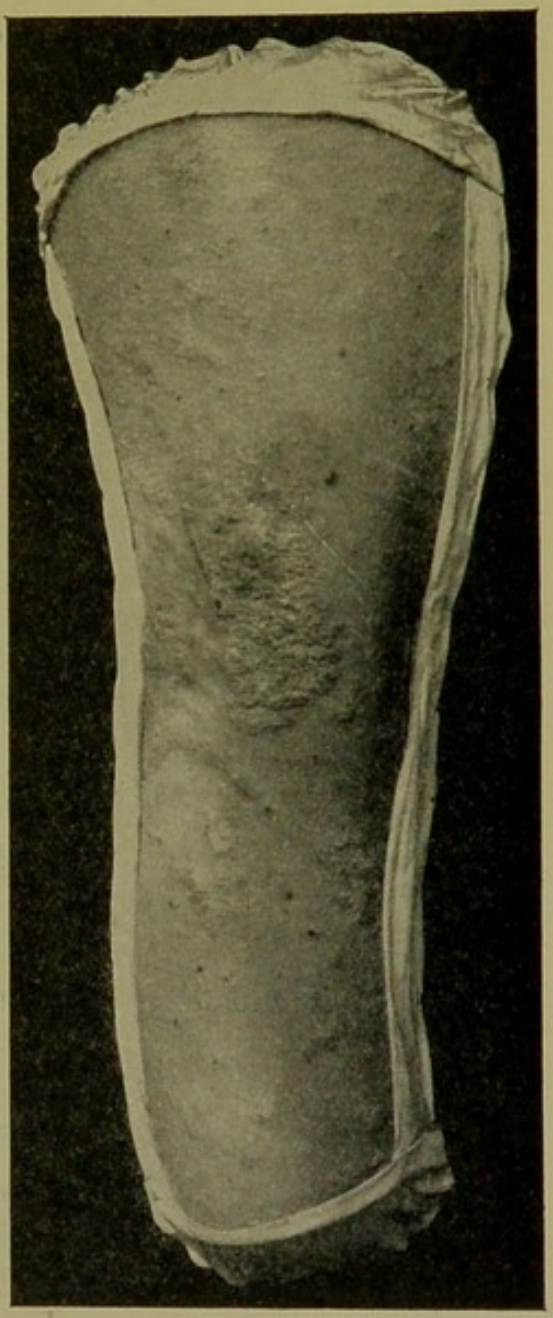


Fig. 28.—Lichen verrucosus. (St Louis Hospital Museum.)

Prognosis.—Lichen planus is an obstinate disease, but is nearly always cured; but horny lichen often resists all kinds of treatment.

Pathological Anatomy. —The histological lesions consist essentially in embryonic proliferation of the superficial layer of the dermis, especially around the blood-vessels, hair follicles, and sweat glands. The lesions have always a perivascular origin (Balzer). This proliferation leads to atrophy of the hair follicles and sweat glands by compression. The papillæ are also infiltrated with embryonic tissue, especially at the periphery. According to Torok, the umbilication of the papules is due

to the presence of a gland duct, generally that of a sweat gland, which prevents the elevation of the skin at this point by the subjacent proliferating cells; according to others, the central depression is only the remains of a hair follicle. The epidermis is thickened,

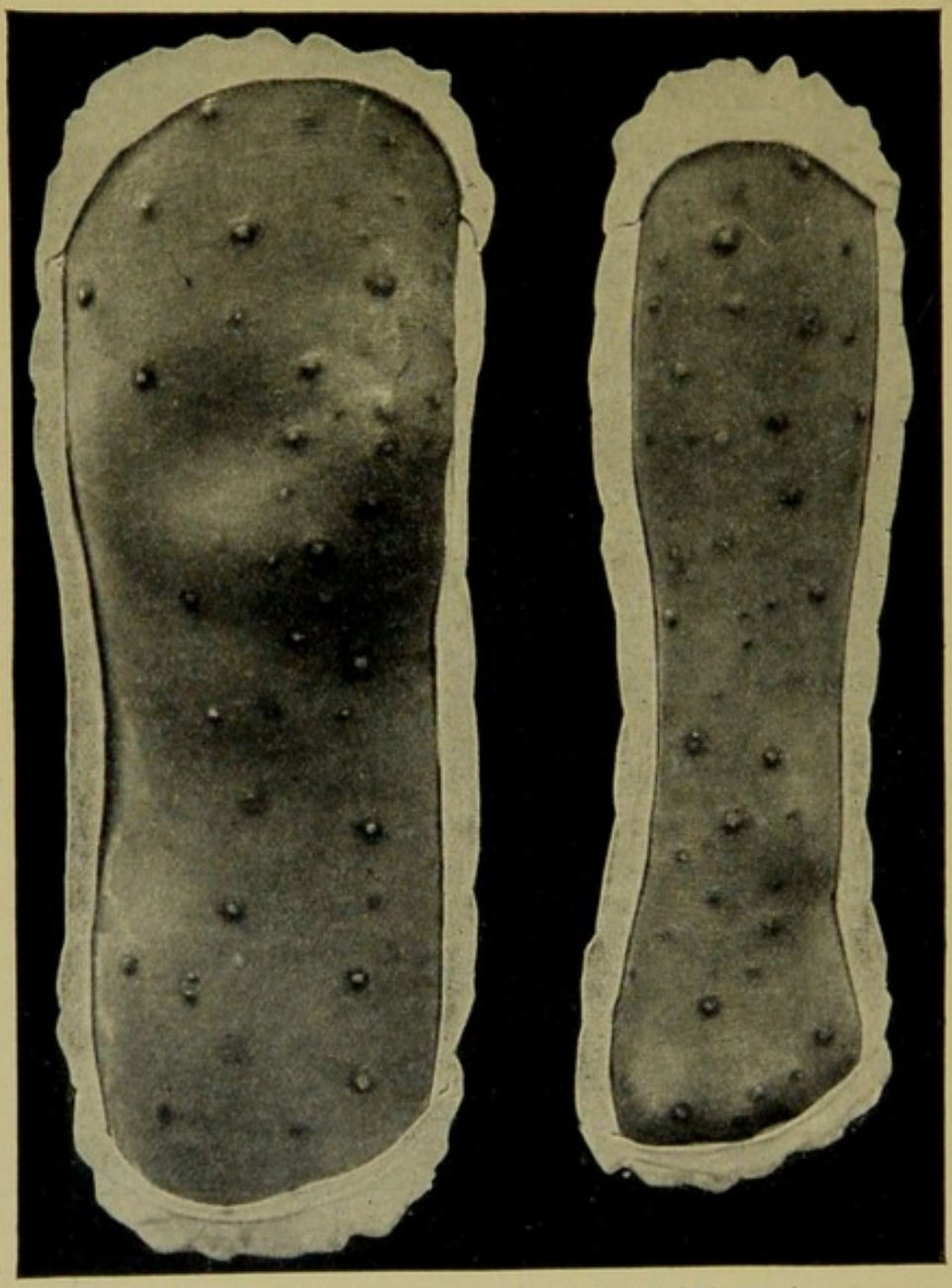


Fig. 29.—Tuberous lichen. (St Louis Hospital Museum.)

owing to active proliferation of its cells, especially those of the stratum granulosum, which form several rows of cells charged with eleidine. In horny lichen the stratum granulosum is still more thickened, and its cells more infiltrated with eleidine.

Etiology.— This is imperfectly known. The disease occurs

very often in arthritic subjects, especially in nervous arthritics. It is not contagious, and does not appear to be hereditary.

DIAGNOSIS.—Lichen planus must be distinguished from tuberculous pilo-sebaceous folliculitis (*lichen scrofulosorum*), in which the papules are miliary, pale red, non-pruriginous, and destitute of central umbilication; this dermatosis occurs in tuberculous subjects. It is impossible to confuse the characteristic papules of lichen planus with the round or acuminate elements of *keratosis pilaris*. Lichen planus is easily distinguished from *lichen simplex*, in which the papules differ in their form and mode of grouping, and in their confluence; from *prurigo*, in which the papules are excoriated and cause intense itching; from *pityriasis rubra pilaris*, in which the epidermic cones surrounding the base of the hairs in no way resemble the papules of lichen planus, and in which there are large red and squamous patches with abundant desquamation on the scalp, face, palms and soles, and changes in the nails. Sometimes old patches of lichen planus resemble those of *psoriasis*, when desquamation in the latter is finer than usual, and does not give rise to the characteristic squames. In the small *papular syphilide* (incorrectly called syphilitic lichen) the papules always have a slightly convex surface, not flat as in lichen planus; they have a copper-red colour, and differ from the papules of lichen planus in the absence of central umbilication and polygonal outlines; lastly, they are not pruriginous. The *lichen ruber acuminatus* of Hebra is a rare dermatosis the nature of which is not determined.

TREATMENT.—*Internal treatment* consists chiefly in the administration of *arsenic* in progressively increasing doses for several consecutive months, in the form of Fowler's solution, from four up to twenty drops or even more, increasing the dose every day by one or two drops. This drug should be stopped if gastric irritation occurs. Arseniate of sodium may also be given, also in gradually increasing doses (from $\frac{1}{16}$ to $\frac{1}{6}$ grain daily). Alkalis may be prescribed, and the same diet as in eczema. For *local treatment*, oil of cade may be applied, or one of the following ointments: pyrogallic acid, 5 or 10 per 100; glycerole of starch containing 3 to 5 per cent. of tartaric acid; salicylic acid, 2 or 3 per cent., with zinc ointment. The itching may be relieved by carbolic lotion (1 per 100); menthol, or guaiacol ointment (1 per cent.), with vaseline and zinc oxide. Tepid douches applied to the spine or tepid shower-baths are useful in pruritus and nervous conditions. Cases have been reported in which lichen planus was cured by tepid hydrotherapy alone, without any other treatment. In acute lichen planus and in acute exacerbations of ordinary lichen planus, sedative treatment by moist compresses is indicated. In horny lichen, the patches should first be treated with soft soap applied on flannel, after which salicylic or pyrogallic

ointments should be used. High frequency currents give good results in all forms of lichen planus, except in the acute form.

PRURIGO.

Prurigo is characterised by two symptoms—a papular cutaneous eruption, and more or less intense pruritus. These two symptoms are to a certain extent independent of each other; the eruption is not the cause of the pruritus, for this may exist without any eruption; on the other hand, the eruption is not caused by the scratching provoked by pruritus, for the itching is often greatest in cases where papules are almost entirely absent. (Devergie's *prurigo without papules.*) On account of the usual association of these two symptoms, prurigo and pruritus must be studied both independently and together.

Acute Prurigo.

This affection has also been called acute lichen simplex. It is constituted by isolated disseminated papules. From the morphological point of view, it is rather a prurigo than a lichen.

Etiology.—This is still little understood. It generally occurs in nervous subjects, who sometimes attribute the eruption to profuse sweating or to errors in diet. Barbe observed an eruption of acute prurigo in a woman who was acutely affected by an operation performed on her daughter. Acute prurigo may occur at all ages, but is most common in children.

Symptomatology.—The eruption usually appears rather suddenly without any general symptoms. In some cases it is preceded by urticaria, or the characteristic papules may develop on urticarial patches. It generally begins on the upper limbs, sometimes on the neck, lower limbs, or on the whole of the body. The characteristic lesion is a small papule the size of a pin's head, pink at first, afterwards bright red. According to Vidal, the apex of most of the papules is whitish in tint, and on puncturing this point a little transparent liquid escapes. Each papule becomes covered by a brown crust, which is eventually detached. When the papule disappears, it leaves a slight pigmentation which gradually fades. The papules may become excoriated by scratching and covered by blood crusts. The duration of each papule varies from three to eight days. The eruption occurs especially on the internal surface of the limbs, on the neck, buttocks and trunk; it is rare on the face and in the articular folds. The elements are always disseminated and quite distinct from each other.

The affection is accompanied by itching, pricking and sometimes

burning sensations, generally intense, but intermittent, and more severe during the night. The eruption appears in successive crops; the total duration of the disease being from two to eight weeks. The papules disappear without leaving any cicatrices or thickening of the skin, as occurs in chronic prurigo. Recurrences are frequent. Besides the acute form, there are subacute forms lasting several months, and constituting transitional stages between acute and chronic prurigo.

DIAGNOSIS.—Acute prurigo is very different from *acute lichen simplex* or papular eczema, the elements of which are agglomerated instead of being distinct like those of prurigo, and which so much resemble ordinary eczema that many authors describe acute eczema and acute lichen together. Acute idiopathic prurigo must not be confused with *symptomatic prurigo.*

TREATMENT.—Externally, antipruriginous lotions or an ointment of zinc oxide containing menthol or guaiacol should be prescribed; internally, sedatives and antispasmodics. In rebellious cases tepid douches may be tried.

Chronic Prurigo.

Already described by Rayer, Cazenave, Gibert and Bazin, this dermatosis was studied afresh by Hebra, and is sometimes known as the *prurigo of Hebra.*

SYMPTOMATOLOGY.—It begins in infancy, sometimes in childhood. It is generally preceded by repeated outbreaks of urticaria and the appearance of red papules, rather large and very pruriginous, resting sometimes on a red base and resembling the papules of strophulus. Hardy described this phase of the disease as *strophulus pruriginosus.*

After a time the true papules of prurigo appear, and replace the urticarial eruptions and strophulus present at first. The papules are very minute, sometimes difficult to see by the naked eye; in this case a sensation of roughness only can be felt by the finger; but they are sometimes of larger size. They are always isolated and quite distinct, and pale pink in colour. Itching is sometimes tolerable (*prurigo mitis*), sometimes intolerable (*prurigo formicans* or *ferox*). In the latter case the patient suffers from pricking and burning sensations, which lead to incessant and violent scratching with the nails or any object that is at hand. The itching is exacerbated by warmth in bed and by contact with the clothes.

The skin soon becomes covered with blood crusts, due to excoriation of the papules by scratching. These excoriated papules may be scattered over the whole body or confined to certain regions; the extensor surface of the limbs, especially the external surface of the thighs and legs, and the posterior surface of the forearms. The hands,

feet and articular folds are not affected. The eruption is rare on the face, but may occur on the buttocks, loins and back.

After the disease has lasted some time the skin becomes rough, thickened and indurated (lichenisation), especially on the lower limbs. It also shows pigmentary spots, which sometimes last long after the prurigo has disappeared. In some severe cases, the eruption becomes more general, itching is more intense, and the prurigo is complicated by secondary lesions due to continued irritation of the skin. The skin then presents, here and there, irregular red patches of various sizes, generally symmetrical, and covered with vesicles, pustules and crusts. The vesicles arise from vesicular transformation of the papules by increased serous exudation; the pustules result from secondary infection of the vesicles by pus cocci, and produce yellow crusts like those of impetigo, or brown like those of ecthyma. These lesions are especially marked on the lower limbs, where prurigo attains its greatest intensity. The infection of the skin may be still more profound, giving rise to furuncles and even subcutaneous abscesses, and the inflammation, spreading to the lymphatics, may cause glandular swellings, especially in the groins (Hebra's *buboes of prurigo*).

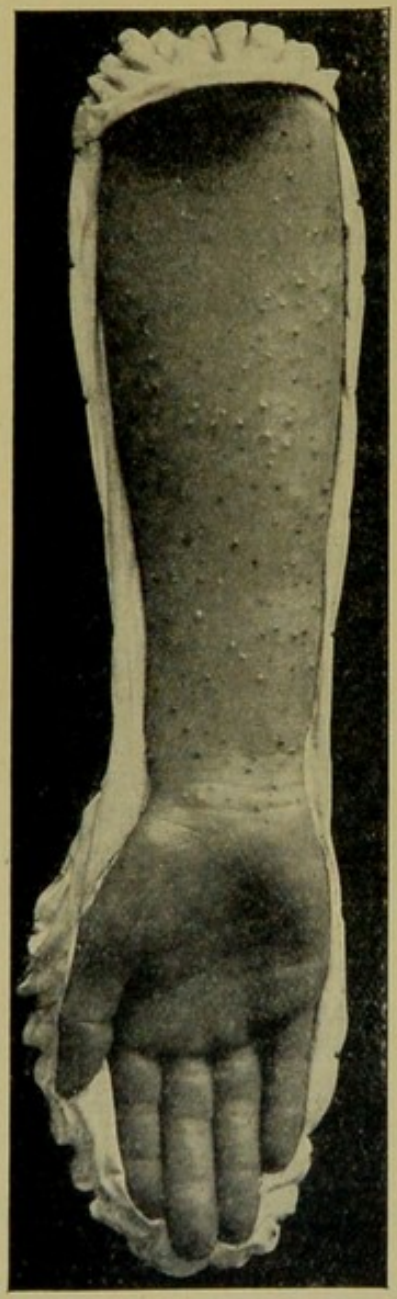


Fig. 30.—Prurigo. (St Louis Hospital Museum.)

When the affection reaches this degree of intensity, the general health is much affected; patients suffer from insomnia owing to the itching, and from loss of appetite; fever may be caused by the secondary suppuration, and life becomes a veritable torment.

Prognosis.—Chronic prurigo is in most cases a very obstinate affection, which lasts nearly for life; even if it becomes cured for a time, it is always liable to recur. Exacerbations occur after fatigue, alcoholic or dietetic excesses, or after exposure to cold. The attacks are especially frequent during spring and winter, but become milder in summer owing to the increased secretion of sweat, which moistens the skin and relieves the

itching. The prognosis is better in *prurigo mitis*, which is sometimes curable.

PATHOLOGICAL ANATOMY.—The histological changes do not account for the intensity of the pruritus which is characteristic of this dermatosis, and no changes have been found in the cutaneous nerves. Leloir and Tavernier have shown that the lesions occur chiefly in the Malpighian body, and consist in small cystic cavities, underneath which the superficial part of the dermis presents dilated blood-vessels surrounded by embyronic cells. The cavities contain altered epithelial cells, white globules and granular detritus.

ETIOLOGY.—Chronic prurigo is most certainly a dermatosis of diathetic origin. This is proved by the alternation so frequently observed between eruptions of prurigo and bronchitis, as well as by the hereditary nature of the disease, which often affects several members of the same family. Patients affected with prurigo are not only arthritic or lymphatic, but also essentially nervous subjects.

DIAGNOSIS.—Chronic prurigo must be distinguished from *lichen simplex*, *lichen planus* and *eczema*. In eczema there is always a history of a vesicular eruption and exudation preceding the crusts, and there are no characteristic papules of prurigo around the patches. Prurigo must also be distinguished from urticaria and strophulus, but it must be borne in mind that recurrent urticaria and strophulus in children are often the prelude to chronic prurigo.

Prurigo must be diagnosed from *parasitic prurigo*, which is symptomatic of *scabies* and *pediculosis*. *Scabies* affects the interdigital spaces, genital organs, breasts, etc., where the characteristic burrows can be found; it is a polymorphous eruption, in which vesicles and pustules are nearly always present as well as papules. In *pediculosis* the lesions occur chiefly on the back and shoulders, where traces of scratching can be found; the discovery of the parasites will remove all doubt. The diagnosis between prurigo and the different forms of *pruritus* is given in the next article.

TREATMENT.—In lymphatic subjects arsenic and cod-liver oil should be prescribed, or if the latter is not well tolerated, iodo-tannic syrup. The diet should be the same as in eczema. Itching may be relieved by bromides, valerian, or aqua laurocerasi. Opium, chloral, sulphonal, etc., may be given to procure sleep. Nervous subjects obtain benefit from tepid hydrotherapy.

Locally, the cutaneous irritation may be relieved by permanent aseptic gauze compresses, soaked in boiled water, or by starch poultices. Prolonged starch baths are useful, dusting powders, zinc ointment or linimentum calcis being applied during the intervals.

After the inflammation has subsided the body should be rubbed daily with cod-liver oil. Later on, ointments are indicated: glycerole of oil of cade or naphthol ointment (5 per 100, or 2 per 100 for

children), or salicylic acid (1 or 2 per 100). Hebra recommended sulphur, and prescribed the following ointment:

Sulphur Oil of cade	. . . .	aa 15 parts
Soft soap Lard	. . . . .	aa 30 „
Prepared chalk	. . . .	10 „

This is a good prescription, but is irritating and must be used with caution.

Mineral waters containing sulphur or sodium chloride may be prescribed.

PRURITUS.

By this name I mean *primary pruritus*, not symptomatic of any eruption, in which the itching is the only symptom, but is even more intense than in prurigo.

SYMPTOMATOLOGY.—Pruritus may be general or local.

General Pruritus.—This is manifested by various degrees of itching, sometimes by pricking or burning sensations. These symptoms generally occur intermittently, with intervals during which there is hardly any pruritus; but sometimes they are permanent, and so severe that patients cannot bear the contact of clothes. These attacks occur several times a day; sometimes they are periodic, occurring at the same hour every day. They may be caused by emotion, anger, hard brain work, or by variations of temperature. They are especially liable to occur at bedtime, and are aggravated by the warmth of the bed.

During the attack, the itching begins in one place and then spreads over the whole body. Patients scratch themselves furiously, and seek solitude in order to scratch more comfortably. They are usually relieved by cold. This violent itching leads to insomnia and melancholia, and sometimes drives patients to suicide. The skin is dry and rough and may present a very few papules; it may even show no signs of scratching. In other cases there are excoriations, which may leave pigmented spots.

There are two forms of general pruritus: (1) senile pruritus; (2) winter pruritus, described by Duhring.

Senile pruritus generally appears after the sixtieth year, and gives rise to intolerable itching. The skin is only a little rough and pigmented.

Winter pruritus appears in the autumn and lasts during the winter till the spring, appearing again in the following autumn. It is at first confined to the lower limbs, but may extend to the whole body. The skin presents papules and scratch-marks. The

attacks occur especially at bedtime. A *summer pruritus* might also be described, for there are some patients who are only affected during the warm season.

These two forms of pruritus show that the skin reacts differently in different subjects; in senile pruritus, there are few cutaneous lesions, in spite of the violence of the itching; in winter pruritus, the skin is altered, although the itching is less intense.

Crocker has described *pruritus mentis* as a form of mental aberration. I have also observed nervous individuals, on the borderland of neurasthenia and mental aberration, who imagined they had parasites in the skin, and, possessed with this idea, suffered from incessant itching which nothing would relieve, so that life became a veritable torment.

Local Pruritus.—This is more common than general pruritus, but also occurs in arthritic and nervous subjects. It affects chiefly the anus, genital organs, palms and soles.

Pruritus ani affects the anus, but may extend on the one hand to the lower part of the rectum, and on the other hand to the perineum and along the intergluteal fold as far as the coccyx. There is no eruption, but the itching is very intense and is aggravated by walking, sitting, rest in bed and defæcation. The skin of the anus is sometimes normal, sometimes rough, swollen, and excoriated. Pruritus ani is sometimes due to hæmorrhoids, but more often primary and essential. It is not uncommon in phthisical patients.

Pruritus of the genital organs in women (*prurigo pudendi muliebris*) affects the upper part of the labia majora, clitoris and pubis. It causes violent itching, which, from repeated scratching, sometimes leads to onanism and nymphomania. Pain is sometimes intolerable, and may provoke attacks of hysteria. The skin of the vulva is rough and excoriated by scratching; the labia majora are swollen and pigmented. The pruritus may extend to the vaginal mucous membrane, which becomes red and swollen and gives rise to a muco-purulent discharge. This condition is often associated with pregnancy and disappears afterwards. Sometimes it occurs at the menopause, and is then very obstinate.

Genital pruritus is less common in men, and affects the scrotum and perineum. The skin of the scrotum is red, rough, and covered with papules and excoriations.

Palmar and plantar pruritus is symmetrical; it is rather rare, but causes intense itching.

ETIOLOGY.—Pruritus is always a manifestation of arthritism and nervous instability. In winter pruritus, cold is only an exciting cause. The affection is aggravated by errors in diet and alcoholic excess.

Apart from this diathetic pruritus, *symptomatic pruritus* is

observed in cases of jaundice, diabetes and Bright's disease. Vulvar pruritus is very often due to diabetes. One of the most common forms of symptomatic pruritus is that observed in jaundice, especially in chronic icterus; it causes very intense itching, and sometimes the skin presents papules. This form has been attributed, without proof, to irritation of the papillæ by biliary acids in the blood.

Pruritus, whether it be diathetic, primary or secondary, is always connected with an auto-intoxication.

Senile pruritus is the result of insufficient elimination and disturbance of nutrition, with retention of nitrogenous extractive matter in the blood, either from fibroid degeneration of the kidneys, or from excessive production of extractive matters. In fact, the excretion of nitrogen is always diminished in senile pruritus. The pruritus of Bright's disease is naturally of the same origin.

Icteric and diabetic pruritus are due to intoxication by bile or sugar, and also by extractive matters, which are always produced in excess in diabetes and in affections of the liver. Local pruritus, like general pruritus, is also diathetic and auto-toxic.

Prognosis.—This is unfavourable, all forms of pruritus being very obstinate.

Diagnosis.—In *general pruritus* the nervous irritability may be relieved by prolonged starch or alkaline baths or by tepid hydrotherapy. Sedative lotions are often useful, such as: vinegar (1 part to 2 of warm water); chloral (2 per 100); carbolic acid (1 per 100, with a quarter of glycerine); perchloride of mercury (1 in 1000, with 4 per cent. aqua laurocerasi). In the intervals between lotions and baths the skin should be dusted with powdered talc and oxide of zinc, powdered camphor, or menthol (1 or 2 per 100.) A 1 per cent. ointment of guaiacol or menthol also gives good results. Internally, antispasmodics and sedatives should be prescribed; valerian, tincture of musk, or aqua laurocerasi.

In *pruritus ani*, suppositories of opium, cocaine or belladonna are useful. In obstinate cases a solution of nitrate of silver (5 per 100) may be applied to the anus.

In *vulvar pruritus* the preceding lotions may be used, or very hot water; also sedative powders and ointments: carbolic, 1 per 100; camphor, 10 per 100; menthol, 1 or 2 per 100; cocaine, 1 or 2 per 100.

In rebellious cases of *local pruritus* linear scarifications, or light application of the actual cautery, have been recommended.

The constant current is sometimes successful in cases of general and local pruritus, the positive pole being placed on the affected part, the negative pole on the neighbouring regions. In vulvar pruritus, high frequency currents may be tried, or simple faradisation.

Mineral waters, such as Vichy, are useful in pruritus.

It is important to regulate the diet in patients suffering from pruritus, in accordance with the etiology which I have already explained. This diet is the same as in eczema, and its object is to avoid foods which are rich in extractive matters, and fermented or easily fermentable substances.

When pruritus is due to diabetes, suitable diet must be ordered.

STROPHULUS.

This is a papular affection, commonly known by the name of teething rash.

Some authors deny its independent existence, and connect it with acute lichen simplex, urticaria or prurigo. Chronic diathetic prurigo is preceded by an eruption which is similar in all respects to the dermatosis under consideration (*strophulus pruriginosus*). Again, it is not uncommon to see erythema, urticaria, or prurigo coincide with strophulus. In spite of this, the latter has its autonomy and exists independently. It most resembles acute lichen, but the papules differ from those of lichen.

Strophulus is characterised by red or white papules distinct from one another, rather large, rounded, pruriginous and of short duration.

Etiology.—This affection occurs exclusively in young children or sucklings. It is due to gastro-intestinal disorder, caused either by defective or too abundant alimentation, and is often provoked by the first dentition.

Symptomatology.—Strophulus is usually, but not always, preceded and accompanied by gastric disturbance with slight febrile symptoms. The papules, the size of a pin's head or millet seed, first appear on the trunk, then on the limbs and face; they cause intense itching, which leads to scratching and excoriations covered with blood crusts, as in prurigo. The papules are red or white, isolated or grouped in the form of patches. Bateman described: (1) *strophulus intertinctus*, with red prominent scanty papules mixed with erythematous spots; (2) *strophulus confertus* (confluent), with smaller but more numerous papules, confluent, although always distinct, and paler red than the preceding; (3) *strophulus volaticus*, with red papules arranged in circular groups, appearing and disappearing while others appear elsewhere; (4) *strophulus albidus*, with white papules surrounded by an erythematous areola; (5) *strophulus candidus*, with larger white papules, but without any red areola.

Evolution.—All the papules are of short duration and disappear in a few days, but as they occur in successive crops the affection

may last for several weeks. It is a benign affection. However, it is necessary to make some reserves; when strophulus is prolonged by repeated outbreaks, accompanied by recurrent urticarial eruptions, it may develop into chronic diathetic prurigo.

DIAGNOSIS.—*Acute lichen* and strophulus do not appear at the same age. *Papular erythema* is rarely isolated, and is nearly always associated with other characteristic lesions of polymorphous erythema.

Scabies is sometimes difficult to distinguish from strophulus, for, in children, the lesions of scabies do not specially affect the interdigital spaces nor the genital organs; they may occur on any part of the body, especially on the feet. But in scabies, vesicles and pustules are nearly always present in addition to papules, and the characteristic burrows containing the acarus can be found.

TREATMENT.—The diet should consist of milk exclusively. A mild purgative should be given. The papules may be dusted with a mixture of powdered talc and starch. Starch baths and an ointment of oxide of zinc with menthol (1 per 100) are useful.

PSORIASIS.

Psoriasis is a dermatosis characterised by dry, whitish, micacious squames, which are always situated on slight papuliform red elevations, of variable form and extent.

SYMPTOMATOLOGY.—The eruption of psoriasis appears insidiously, without any prodromal symptoms or general phenomena, and without any noticeable itching. At its onset, the lesion consists of a small macule, slightly raised, but already squamous. The congestive macule and the squame often appear at the same time. This macule is at first the size of a pin's head (*psoriasis punctata*), but it grows to variable dimensions. The papulo-squamous eruption, by developing and becoming grouped in different ways, gives rise to the different morphological varieties of psoriasis. If the macules remain isolated, and only a little enlarged, we have *psoriasis guttata*, with white, micacious, elevated, dry squames resembling the droppings of a wax match. If the macules unite to form more or less extensive round patches, we have *nummular*, *orbicular* or *discoid psoriasis*. In other cases the eruption is grouped in the form of large, irregular patches with thick squames, constituting *psoriasis scutata*. The patches may be so extensive as to cover most of the skin (*psoriasis diffusa*). Sometimes the patches form various figures (*psoriasis figurata*), or they may be arranged in sinuous bands (*psoriasis gyrata*). In some cases they form complete rings surrounding healthy skin (*psoriasis circinata*); this was formerly regarded as a

distinct disease and was called "common leprosy." This circinate form may also be produced by a patch of discoid psoriasis which has healed in the centre.

In all these varieties the squames are characteristic; scaly, shining and white, becoming whiter when scratched; sometimes they are yellowish. The squames vary in thickness; sometimes fine, but more commonly thick. They consist almost entirely of flattened epidermic cells, sometimes mixed with a few blood cells and leucocytes produced by scratching. They are rather adherent to the subjacent papule, and when detached reveal a sub-squamous cuticle (Bulkley). The subjacent papule is usually slightly marked; it is red, smooth, and sometimes extends beyond the squame which covers it; it bleeds easily when scratched, showing blood points which are almost characteristic of this disease.

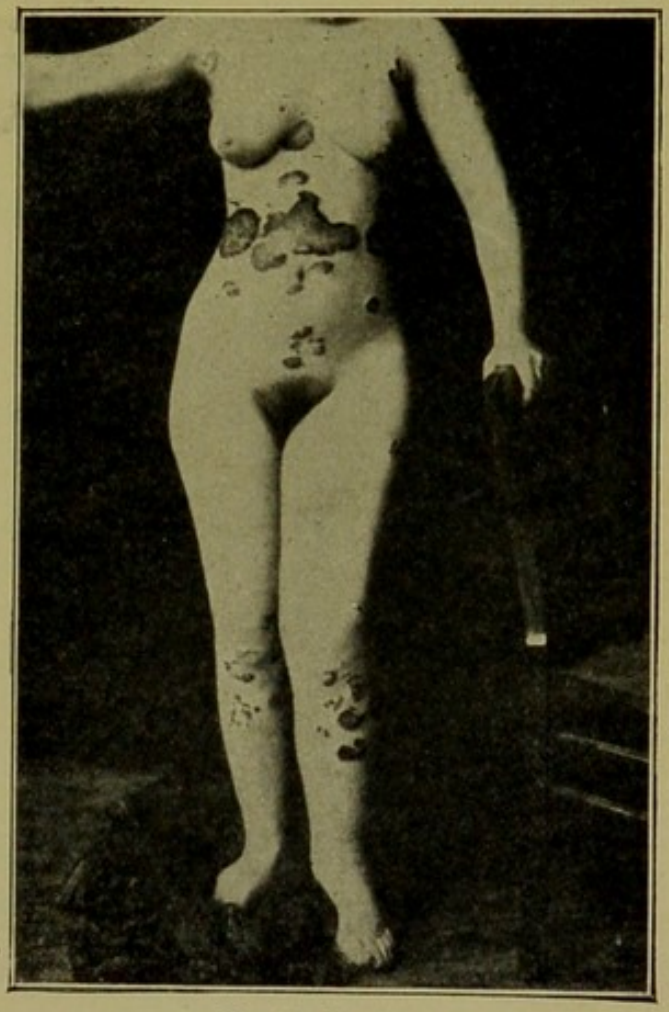


Fig. 31.—Psoriasis.

Itching is usually absent; when present it is slight, and occurs chiefly at the onset of the eruption and at each fresh outbreak. Sometimes painful excoriations are produced by scratching.

Psoriasis is not accompanied by any functional troubles; if patients are affected with dyspepsia, neuralgia, rheumatic or gouty joint affections, these depend on the same cause as the psoriasis itself. The arthropathies of psoriasis are identical with those of chronic rheumatism, as is shown by radiographs (pp. 124, 125).

Evolution.—Psoriasis runs a chronic course. In rare cases it disappears spontaneously; the squames become detached, the papules subside, leaving red spots which are followed by pigmentation of long duration, but no cicatrices.

When left to itself, psoriasis usually forms thicker and thicker

squames, and large patches which extend and coalesce and may be complicated by painful fissures. In chronic cases, papillary hypertrophy sometimes develops, especially on the lower limbs and lower part of the back, giving rise to a warty appearance (*papillomatous psoriasis*).

Acute Psoriasis.—In exceptional cases psoriasis develops, either at its onset or on the occasion of fresh outbreaks in an acute form, due to alcoholic or dietetic excess, or to irritating local treatment.

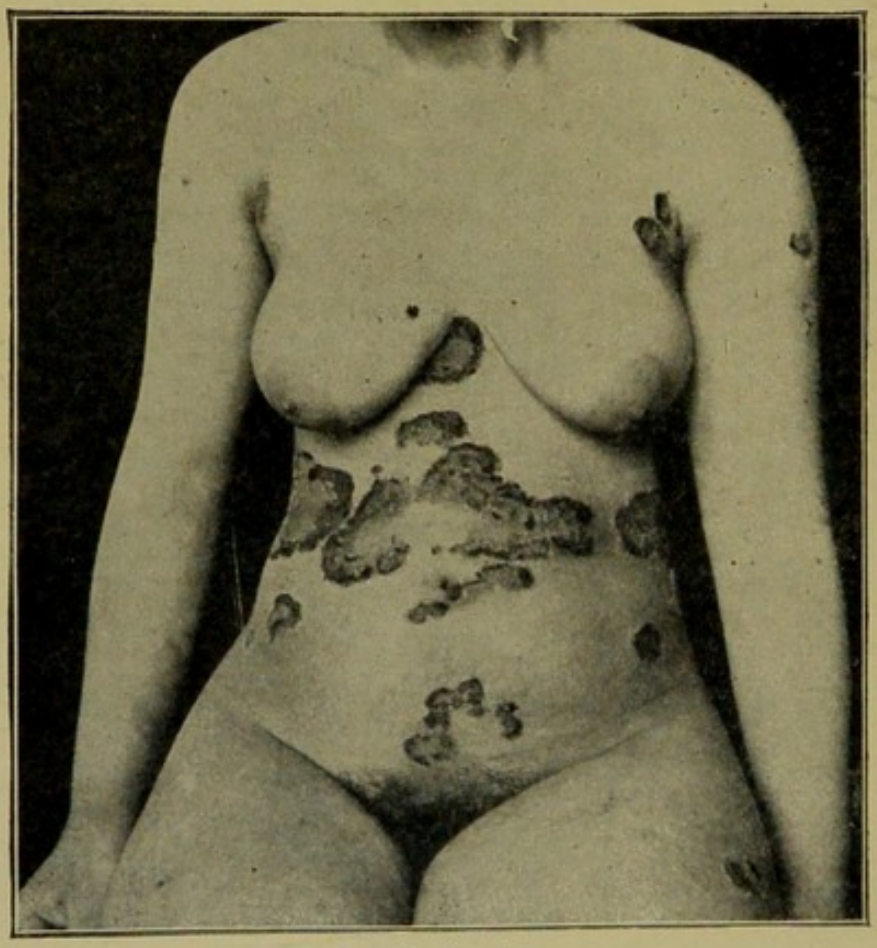


FIG. 32.—Psoriasis.

This acute psoriasis, which sometimes develops spontaneously, is an almost generalised eruption, formed, not by white patches, but by red, confluent, pruriginous patches, accompanied by abundant epidermic exfoliation, with fever, headache and gastric symptoms. This acute form may gradually pass into the chronic state, or the eruption may become aggravated and transformed into a variety of *secondary pityriasis rubra*; but this transformation is rare.

REGIONAL VARIETIES.—Psoriasis occurs chiefly on the limbs, especially on the extensor surfaces; the *knees* and *elbows* are affected so frequently that in any doubtful eruption these parts should

always be examined for typical spots of psoriasis. It is also common on the trunk, back, loins and sacral region, less common on the dorsum of the hands and feet, and rare on the palms and soles.

FIG. 33.—Psoriasis circinata.

On the *scalp*, it occurs sometimes in the form of circumscribed patches, sometimes in a diffuse form; the squames are large, thin or thick, and very adherent to the scalp and hairs; when the eruption is diffuse it often stops at the junction of the forehead and scalp;

there is never consecutive alopecia. On the *face*, psoriasis is somewhat atypical in appearance, and is characterised by red patches covered with fine squames, resembling lupus erythematosus, seborrhœic eczema, or pityriasis simplex. Psoriasis often occurs on the *ears*, when it is usually the result of extension from the scalp. When the eruption affects the external auditory canal it may produce deafness by accumulation of squames. On the *genital organs*—scrotum, prepuce, glans penis and vulva—psoriasis presents a

Fig. 34.—Arthropathies of psoriasis.

slightly different aspect, due to the natural moistness of these regions; the papules are covered with thin, soft squames, resembling syphilitic mucous patches.

Atypical Psoriasis.—This term is given to psoriasis occurring in regions where it is not usually met with, and which does not present the usual characters of common psoriasis. It applies especially to *palmar* and *plantar psoriasis*. This localisation is so exceptional that some authors have considered all squamous palmar eruptions as syphilitic; but this is incorrect. This atypical psoriasis is constituted by epidermic stratifications of variable thickness with

a circinate tendency. It may be mistaken for *eczema*, or for the *papulo-squamous palmar syphilide*. Psoriasis is more circumscribed and drier than eczema, in which vesicles are sometimes present; the squames of psoriasis are whiter and thicker, and are situated on a redder base. The papules of the papulo-squamous syphilide are of a coppery-red colour, and are arranged in a characteristic polycyclic form. In some cases, however, the diagnosis can only be settled by the history of the case or by the presence of psoriasis in other parts of the body; sometimes it is only elucidated by the positive or negative results of anti-syphilitic treatment, especially in cases where psoriasis occurs in subjects who have had syphilis.

FIG. 35.—Arthropathies of psoriasis.

In the *articular folds*, psoriasis is so rare that its occurrence in these situations has been denied by some authors. It occurs in the form of red, smooth patches, sometimes covered with soft squames and accompanied by slight exudation and crust formation (*eczematous psoriasis*). Psoriasis of the articular folds has been confused with seborrhœic eczema (Unna). In fact, there are transitional forms between the two eruptions, but atypical psoriasis of the articular folds exists nevertheless.

Psoriasis of the Nails.—This is fairly common. The nails present a punctate appearance, and the spots are covered by transverse and longitudinal striæ; they are dry, brittle, and thickened by

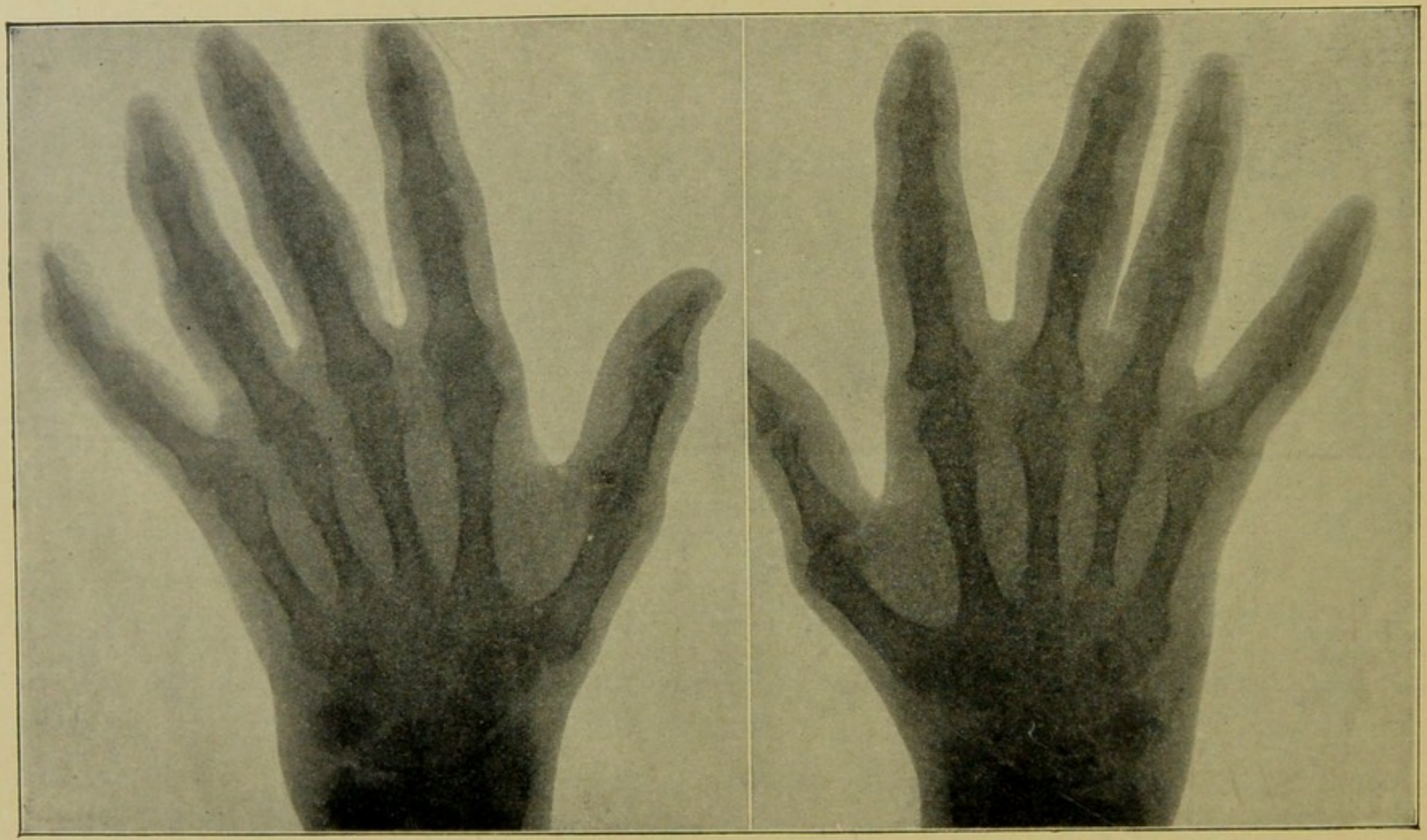


Fig. 36.—Arthropathies of psoriasis.

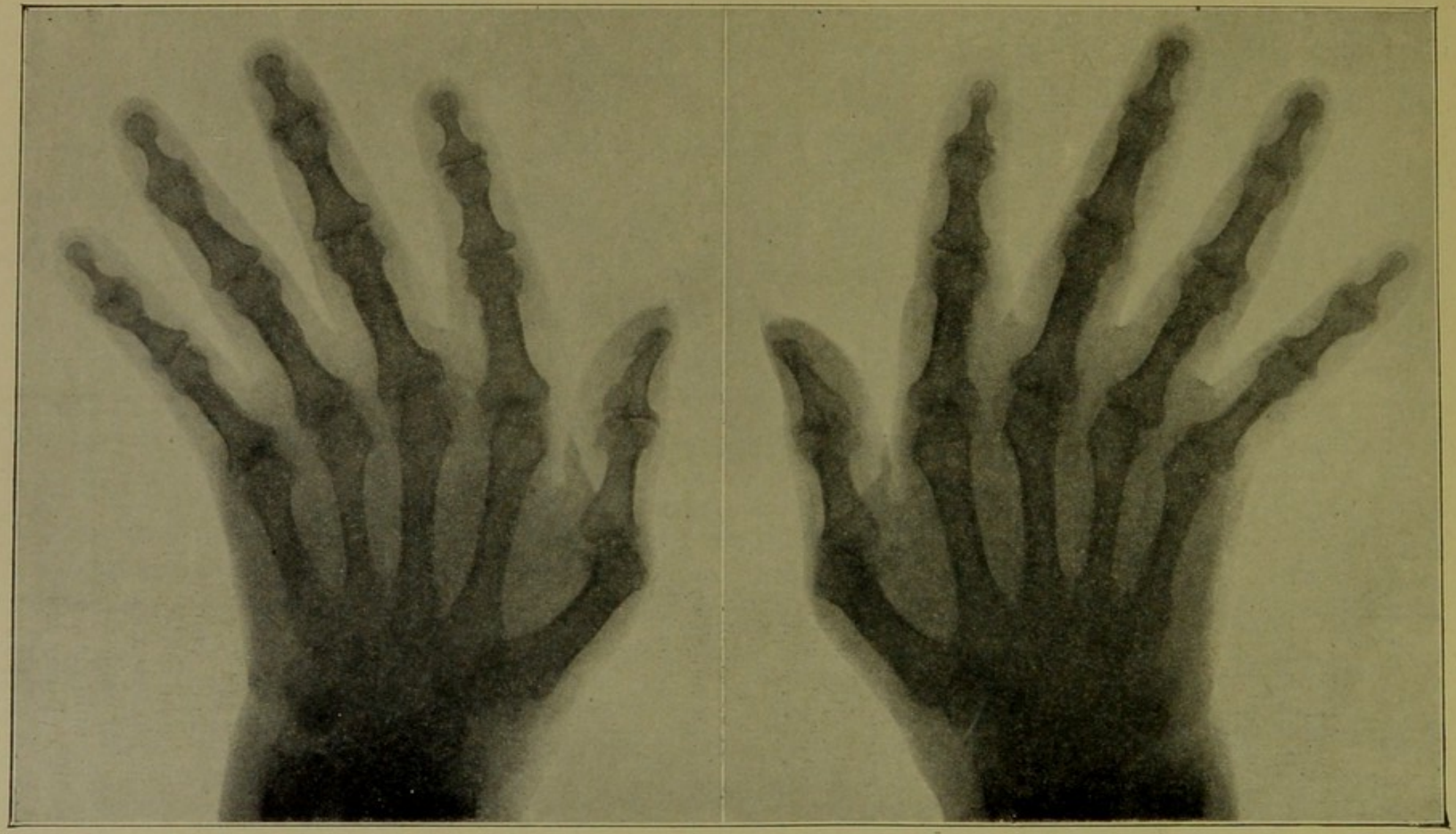


Fig. 37.—Arthropathies of chronic rheumatism.

epidermic stratification. The nails often become detached. The diagnosis is easy when psoriasis is present in other regions; but when the nails only are affected it is impossible to distinguish it from ungual eczema.

Psoriasis never affects the mucous membranes. The affection incorrectly called *buccal psoriasis* is quite different from cutaneous psoriasis, and should be termed *buccal leucoplakia.*

DIAGNOSIS.—Psoriasis can only be mistaken for ordinary *eczema* when the latter is squamous and nummular; but in this case the squames are fine, gray, and preceded by exudation. *Seborrhœic eczema* leads to confusion when its patches are the seat of abundant desquamation; but the different distribution of the two diseases, and the result of scratching the patches, which in psoriasis makes the squames whiter and produces punctiform hæmorrhages, facilitate the diagnosis.

Psoriasis guttata is easily distinguished from *pityriasis rosea;* the latter is a generalised eruption characterised by red spots covered with fine squames; scratching produces no punctiform hæmorrhages as in psoriasis.

The pearly squames of psoriasis cannot be mistaken for the large, abundant squames of *exfoliative dermatitis,* in which there is also general redness of the skin. In the *papulo-squamous syphilide* the papules are coppery red, and the squames are thinner and less pearly than in psoriasis; they are also less raised, and do not bleed when scratched. The diagnosis from *pityriasis rubra pilaris* will be considered with the latter disease.

Psoriasis of the scalp is easily distinguished from *pityriasis capitis,* which is not accompanied by cutaneous inflammation and has finer and usually fatty squames.

Favus cannot be mistaken for psoriasis; the thick, yellow masses are powdery and not squamous, and have a mouse-like odour; moreover, the characteristic fungus can be found by microscopic examination.

On the face, psoriasis is easily diagnosed from *pityriasis simplex* and *trichophytic erythema.* In *lupus erythematosus* the squames are thin and yellow, and there are cicatrices.

ETIOLOGY. — Psoriasis is a diathetic affection which usually appears after puberty, but may occur in children, especially in hereditary cases, which are fairly frequent. It is not uncommon for psoriasis to develop at the onset of menstruation, during pregnancy, or after childbirth. It sometimes appears after errors in diet, alcoholic excess, or violent emotion. In predisposed subjects, it may occur after external irritation or prolonged pressure.

The influence of emotion in predisposed subjects cannot be denied, and has led to a theory which attributes the eruption of

psoriasis to the same tropho-neurotic cause as the arthropathies which are sometimes observed in this dermatosis. But this theory is not exact; the nervous disturbance is only an exciting cause of the eruption.

Lang and Wolff regarded psoriasis as a parasitic affection, but this is neither proven nor probable; first, because the spores described (*lepocolla repens*) are common spores; secondly, because attempts at inoculation have failed (Lassar), and no case of contagion has been proved. However, Destot obtained a positive inoculation on himself, but this single experiment is insufficient.

In fact, the old diathetic doctrine of psoriasis has not been demolished. Diathesis being now understood, as I have already pointed out, to signify auto-intoxication by nitrogenous extractive matters, psoriasis has a humoral origin, analogous to that of eczema.

It has been shown by Desmoulière and myself that the nitrogenous excretion in the urine is constantly diminished in psoriasis, as in eczema. We have also shown that there is considerable demineralisation in all cases of psoriasis, especially an excessive elimination of chloride of sodium, chiefly during resolution of the eruption. Chloride of sodium aids in the elimination of toxic nitrogenous matters, which do not undergo dialysis without it, or are only incompletely dialysed.

But, on the other hand, this excessive demineralisation, especially the considerable loss of chloride of sodium, diminishes the resistance of the organism, and this no doubt explains why psoriasis is so common in tuberculous families. In fact, the sodium salts constitute a means of protection against certain infections, such as tuberculosis, as has been shown by the researches of Charrin, Richet, Robin, etc.

Prognosis.—The prognosis of psoriasis is not grave in itself, but is unfavourable owing to the numerous recurrences, which may occur during the whole life. It may also be aggravated by visceral disorders and chronic arthropathies, depending on the diathesis which causes psoriasis. It may become grave owing to metastases, which have been observed in chronic cases of psoriasis after rapid disappearance of the eruption.

Treatment. — The occurrence of these metastases does not contra-indicate treatment of the eruption; but, as in eczema, treatment should be gradual, and local treatment should always be combined with appropriate general treatment. In old people, active medication should be avoided; the squames should be removed by baths or simple ointments; but, on the face and hands, more active measures may be adopted.

Internal Treatment.—Patients should follow the same regime as in eczema. The diet should consist largely of milk and vegetables, and alkalis should be prescribed. This treatment applies especially to those in whom the disappearance of the eruption is followed by severe dyspepsia and bronchitis.

Arsenic, which is fairly efficacious, should be combined with local treatment. Vidal treated cases of psoriasis with arsenic alone, but the eruption was only cured with large doses, which are liable to cause arsenical poisoning.

Arsenic must not be prescribed in all cases of psoriasis; it is harmful in acute cases, and should only be prescribed in the chronic forms, or in debilitated subjects. It may be given in the form of Fowler's solution, commencing with four drops twice a day, increased by one drop daily up to 20 or 25 drops. It should be stopped when gastric or intestinal symptoms occur. Arsenate of sodium may also be prescribed instead of Fowler's solution ($\frac{1}{30}$ up to $\frac{1}{10}$ grain daily). Arsenical treatment requires strict supervision. This drug has also been given to prevent recurrences, in the same way that mercury is given in syphilis, but it has not given the results that were expected.

Large doses of *iodide of potassium* have been recommended by some dermatologists, and have been tried in France without much result. This treatment is to be condemned on account of the gastric disturbance it produces.

External Treatment.—This differs according as we have to deal with a case of old torpid psoriasis, or with a fresh acute outbreak, or with a case which will not tolerate active treatment. In the two last conditions, starch baths and zinc ointment or simple lard should be prescribed. At the termination of the acute stage, treatment is the same as for chronic psoriasis. In the latter, the squames must first be removed, and afterwards stimulating ointments applied. The squames may be removed by means of starch or alkaline baths, or by vapour baths; if they are very thick, prolonged baths with some form of soap are necessary. Hillairet recommends vapour baths and starch baths on alternate days. Zinc ointment or vaseline may also be used to remove the squames. When the squames have been removed, stimulating or alterative applications are indicated, the best of which is *oil of cade*. Introduced some time ago by Gibert and Devergie in the therapeutics of psoriasis, oil of cade retains an undoubted superiority over all the medicaments which have since been used. But it also has disadvantages: first, a strong odour which is objectionable to some people; secondly, it stains the skin dark brown, and sometimes causes irritation and even inflammation of the sebaceous glands (*cadic acne*).

Oil of cade may be mixed in variable proportions with oil of

sweet almonds, vaseline, lard or glycerole of starch (from 10 to 50 per cent.). The following is a good prescription:—

Oil of cade	15 parts
Glycerole of starch	90 „
Liquid extract of quillaia } Essence of cloves }	Q.S.

The compound oil of cade ointment recommended for chronic eczema is also useful in psoriasis, and has the advantage of not causing cadic acne:—

Precipitated sulphur } Salicylic acid }	aa 1 part
Oil of cade	10 parts
Oxide of zinc	20 „
White vaseline	30 „

In torpid cases, pure oil of cade may be used.

One of the above preparations is applied to the skin every night; after this the patient sleeps in a flannel nightshirt. The next morning the oil of cade is removed by means of olive oil followed by soap and water. If the patient can remain at home, it is advantageous to retain the ointment during the day, a bath being taken every third day.

In cases of discrete psoriasis with small patches, oil of cade may be applied in the form of *collodion*, the latter being made with anhydrous acetone (1 part oil of cade to 2 parts of collodion).

It has been proposed to replace oil of cade by *birch tar*, which has a less disagreeable odour, but it is not so efficacious as oil of cade.

Jarisch, of Vienna, recommends *pyrogallic acid* ointment (5 to 10 per cent., with vaseline). This sometimes causes irritation of the skin, but this easily yields to emollient applications; it also stains the skin black by oxidation from contact with air in the presence of alkalis. It is also toxic. If there are no excoriations of the skin, it may be applied without danger, but if excoriations or fissures are present, it requires to be used very carefully. The urine should always be examined, and if it becomes black, the ointment should be discontinued. In intoxication by pyrogallic acid, severe gastro-intestinal symptoms, anæmia, hæmoglobinuria, and pulmonary congestion have been observed, and even fatal cases have been reported.

Chrysophanic acid, or rather *chrysarobin*, the active principle of goa powder or araroba, was introduced by Balmanno Squire of London. This is used in the form of an ointment with vaseline (5 to 10 per 100), applied daily. Chrysarobin is free from odour, but stains the skin purple and the hair yellow; it has a caustic action, and may give rise to acute inflammation of the skin and

subcutaneous tissue, from erythema to phlegmonous inflammation. Severe conjunctivitis may be caused by the patient conveying ointment to the eyes by the fingers; in some cases balanitis has been observed. Chrysarobin must, therefore, be used with caution, and never be applied to large surfaces; it is even more difficult to manage than pyrogallic acid.

Other ointments which have been recommended are: naphthol, 5 to 10 per cent.; salicylic acid, 3 to 5 per cent.; salicylic acid, 3 to 5 per cent., with oil of cade, 25 to 30 per cent., with lard; sulphur and oil of cade (30 per cent. of each), with vaseline; soft soap mixed with oil of cade.

On the face and scalp, where dark or staining ointments, such as pyrogallic acid or chrysarobin, are contra-indicated, mercurial ointments may be used, such as calomel or turpeth (10 per cent.).

Small patches of psoriasis may be treated with plasters of oil of cade or pyrogallic acid, or better with *traumaticine* (1 part of gutta-percha dissolved in 9 parts of chloroform), containing chrysarobin or pyrogallic acid (10 per cent.). This medicated solution is painted on the patches, and, when dry, leaves an adherent pellicle. Another method consists in first painting each patch with a solution of the active drug—1 part of pyrogallic acid in 9 parts of chloroform, or 1 part of chrysarobin in 9 parts of sulphuric ether—and covering with traumaticine when dry. This mode of treatment is seldom practised; it is unsuitable for large surfaces; the pellicles become cracked, and require repainting; the patches of traumaticine give a dirty appearance to the skin, and are difficult to detach when the treatment is discontinued.

In children, pyrogallic acid and chrysarobin should never be used, on account of their toxicity; the best applications are dilute oil of cade or naphthol ointment.

Natural sulphur waters, in the form of prolonged baths, are useful in psoriasis.

PITYRIASIS RUBRA PILARIS, OR PITYRIASIS PILARIS.

This affection, first described by Devergie, is characterised by more or less marked desquamation of the palms and soles and afterwards of the rest of the body, by pityriasic seborrhœa of the scalp and face, and by the presence of special follicular lesions.

SYMPTOMATOLOGY.—The affection generally develops slowly. It begins with pityriasic lesions of the palms and soles, followed by pityriasis of the scalp; finally, squamous patches, similar to those on the palms and soles, appear on the body and face. According to Devergie, the lesions appear in this order, the follicular lesions

appearing later. But this is not always the case; the face may be the first part affected, or the follicular lesions may appear directly on the limbs or trunk.

In most cases the affected parts show only slight congestion of the subjacent dermis, but in some cases they are of a pale red colour, which disappears on pressure. In these cases the squames are larger, but always dry. Vesicles, exudation and crusts are absent.

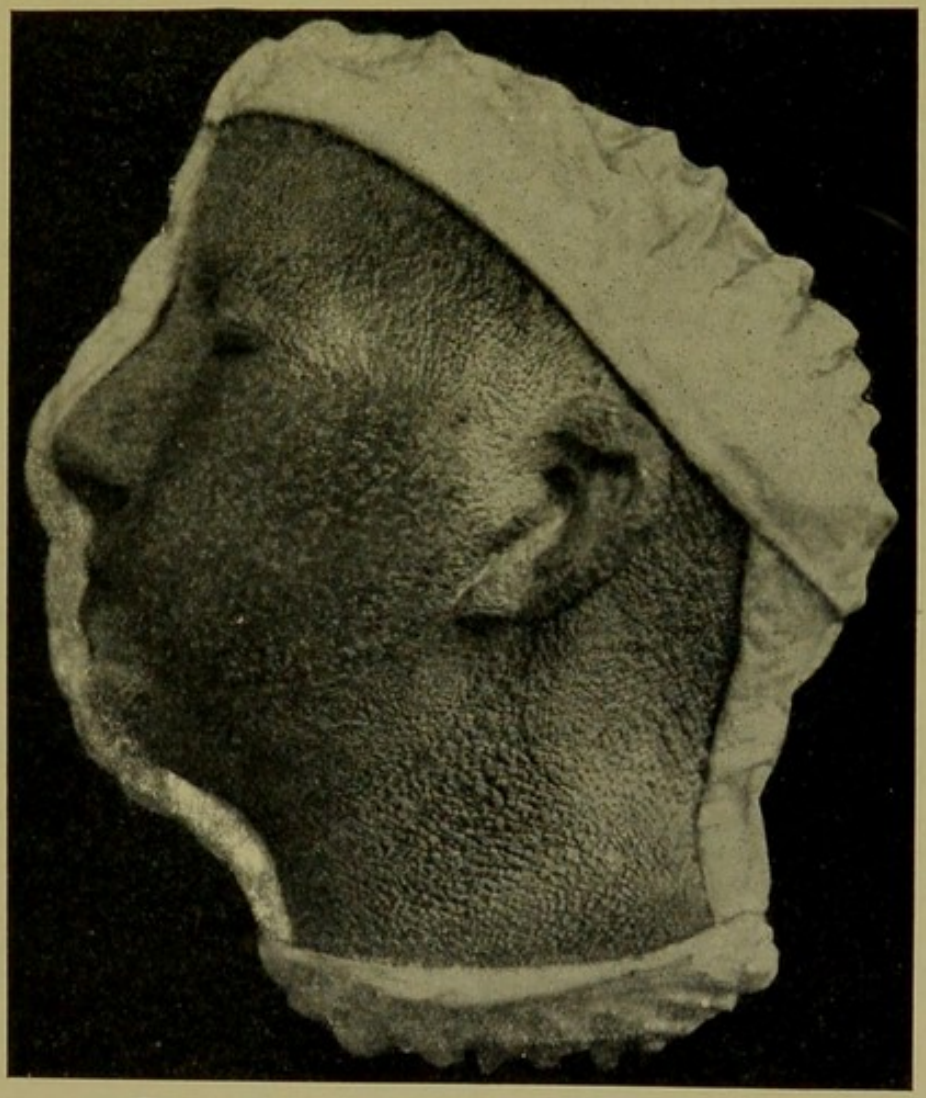


FIG. 38.—Pityriasis pilaris. (St Louis Hospital Museum.)

On the palms, the squames present certain peculiarities; they accumulate in the cutaneous grooves, marking them out in silvery white lines; they are also stratified. The epidermis is often thickened and callous on the thenar and hypothenar eminences.

On the face, the eruption is faintly red and covered with abundant fine squames, arranged according to the cutaneous furrows; these accumulate in the naso-labial furrow, but may cover the nose, forehead and rest of the face. When the whole face is affected, the

skin is floury, tense and rough, and the lower eyelids are everted (ectropion). The scalp is affected with abundant pityriasic desquamation, which attains its maximum in this situation. Sometimes the squames surround the hairs and glue them together, forming an inextricable tangle.

But it is around the hairs, or rather in the hair follicles, that the characteristic lesion is found—*circumpilary epidermic cones*. These cones are placed side by side on a more or less red surface of variable extent, sometimes on surfaces of normal colour. They are situated

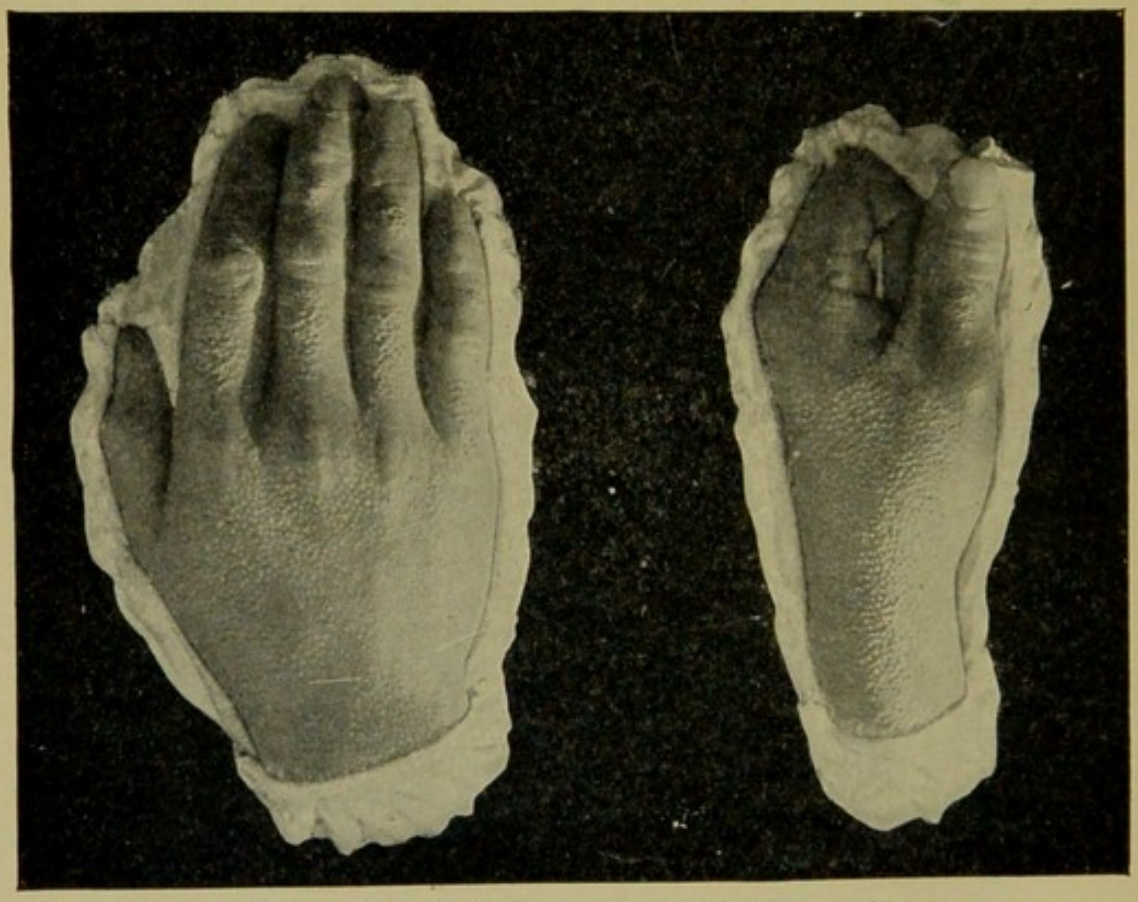


Fig. 39.—Pityriasis pilaris. (St Louis Hospital Museum.)

exclusively around the hairs, and give the sensation of a rough file to the finger. They consist of: (1) a dermic part, conical, with its apex downwards, imbedded in the base of the follicle; (2) an extradermic part, which forms a truncated cone resting on the base of the lower cone. The hairs are sometimes visible, sometimes invisible when they are covered by the epidermic cone; but, if the cones are pulled off, the hairs rise up. These circumpilary cones do not occupy the same regions as the pityriasic patches; they occur chiefly on the dorsal surface of the first and second phalanges of the fingers, on the back of the hand, on the

posterior surface of the forearm and arm, on the shoulder, neck, back, abdomen, knee, and sometimes on the penis. The presence of the cones on the back of the fingers is so characteristic that it settles the diagnosis in doubtful cases.

In regions where the circumpilary cones are absent, the cutaneous grooves are more marked and rendered more apparent by the presence of fine pityriasic squames.

The hair and nails are always more or less affected; the hairs fall easily; the nails are thickened and raised by subjacent epidermic desquamation, and present longitudinal striæ, but never fall off. Vidal has observed hypertrichosis.

The general health remains good, and the only symptoms are itching and pricking sensations. This dermatosis is usually slow in evolution, and lasts for a year or more; but relapses and exacerbations occur without apparent cause. The prognosis is favourable, and the affection always ends in cure, but it is very rebellious to treatment.

Etiology.—This is still uncertain. The disease is more common in males, and occurs at all ages. I have seen two cases, which were treated for pityriasis pilaris and cured, succumb some years later to pulmonary tuberculosis. This is more than a simple coincidence, for Milian has shown that patients affected with pityriasis pilaris, even apparently in good health, react to tuberculin. It would therefore appear that this dermatosis is of tuberculous origin.

Pathological Anatomy.—There is nothing characteristic in the lesions. The stratum corneum is hypertrophied, especially underneath the epidermic cones; the papillæ are infiltrated with embryonic cells. In the cones, the hairs are ensheathed by horny masses, due to hyperkeratinisation of the epithelial wall of the hair follicle.

Diagnosis.—In *exfoliative dermatitis* and *pityriasis rubra* there are no circumpilary cones nor pityriasic seborrhœa of the scalp. In *psoriasis* the squames are thicker and pearly white. The cones of pityriasis pilaris are easily distinguished from the flat, polygonal papules of *lichen planus*. At first sight, pityriasis pilaris may be confused with *keratosis pilaris*, but the two diseases have not the same distribution, and in keratosis pilaris there is no desquamation of the face and scalp. *Ichthyosis* is a congenital dermatosis, characterised by larger and more abundant squames; redness and cones are absent, and the articular folds are not affected.

Treatment.—Internal treatment by arsenic and carbolic acid has been tried without effect. Locally, glycerole of starch with 5 per cent. tartaric acid, or glycerole of oil of cade, may be applied. Frequent baths are also useful.

EXFOLIATIVE DERMATITIS. PITYRIASIS RUBRA.

Besides *primary pityriasis rubra* and *exfoliative dermatitis*, there are generalised desquamative erythrodermias, secondary to chronic eczema or psoriasis, which present the same symptoms as primary exfoliative dermatitis and pityriasis rubra, and must be regarded as cases of *secondary exfoliative dermatitis* or *pityriasis rubra*.

But pityriasis rubra and exfoliative dermatitis must be separated from pemphigus foliaceus and recurrent desquamative scarlatiniform erythema. The latter dermatosis, in spite of its relapses, is an affection of limited duration and benign character, with all the signs of an acute disease. However, it must be admitted that there are transitional forms between this dermatosis and exfoliative dermatitis, and that red desquamative eruptions occur, at first diagnosed as scarlatiniform erythema, which by their persistence and complications gradually assume the characters of exfoliative dermatitis.

Generalised erythrodermias of medicamentous or toxic origin, such as scarlatiniform mercurial eruptions, must of course be distinguished from the dermatoses in question.

Exfoliative dermatitis and chronic pityriasis rubra must be regarded as two forms of the same disease; the former is the subacute and comparatively benign form, the latter the chronic and severe form.

Primary and Secondary Exfoliative Dermatitis.

Exfoliative dermatitis has been described by Erasmus Wilson, Vidal, and Brocq.

ETIOLOGY.—The causes of the primary form are unknown. It appears as a protopathic affection in healthy subjects without previous skin disease, and does not appear to be contagious.

Secondary exfoliative dermatitis occurs after certain forms of chronic eczema. It must be distinguished from secondary chronic pityriasis rubra, which will be described later on, for it is similar in cutaneous symptoms and prognosis to primary exfoliative dermatitis.

In any case, exfoliative dermatitis is a rare disease, but less rare than the chronic form, represented by pityriasis rubra. It is more common in males, and is an affection of adult age; however, the Germans have described a malignant and usually fatal form of exfoliative dermatitis in sucklings, the nature of which is not determined.

SYMPTOMATOLOGY.—In this disease we have to consider three stages: the period of onset, the period of maturity, and the period of decline.

Period of Onset.—The onset of the *primary form* is generally insidious. One or more red patches appear on some part of the body, generally in the popliteal space, groin, fold of the elbow, around the waist, or on the genital organs. The patches are often pruriginous, and extend rapidly (eight or ten days) to the upper limbs and trunk, and then to the lower limbs; the face, hands, and feet are affected later. Occasionally the onset is sudden, and the whole body is affected at once.

When exfoliative dermatitis is *secondary to eczema*, the eczematous patches gradually extend and coalesce, becoming redder and exclusively squamous; finally the eruption becomes general.

Whether exfoliative dermatitis is primary or secondary, when the eruption becomes general, the whole skin is red and shining.

Epidermic exfoliation occurs from the sixth to the twelfth day in the primary form; it sometimes commences before the redness has become completely generalised.

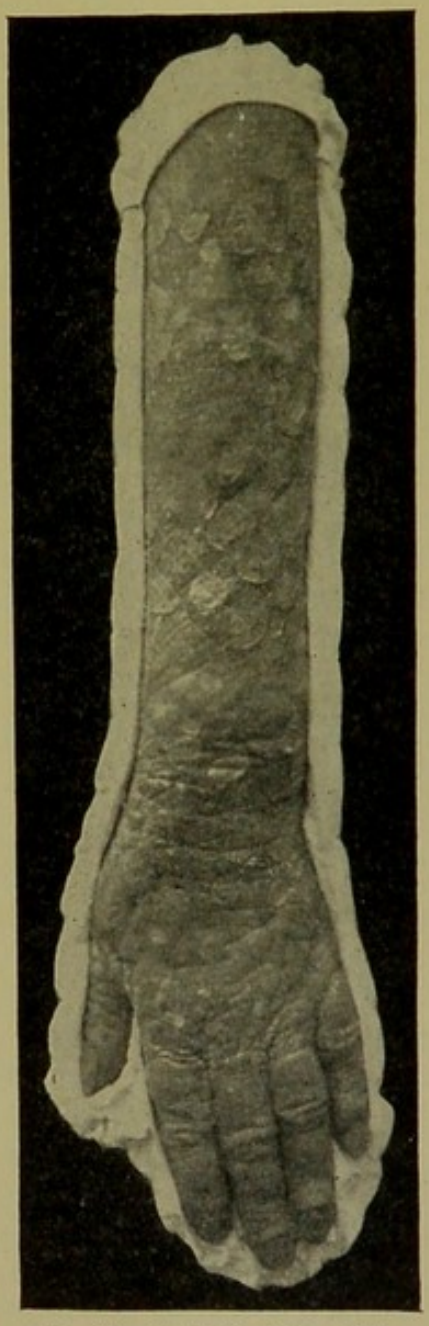


FIG. 40.—Exfoliative dermatitis. (St. Louis Hospital Museum.)

Period of Maturity.—The eruption is deep red like erysipelas, sometimes purple, and affects the whole surface of the body. The skin becomes thickened and tense. On account of this tension, the skin of the face loses its mobility, the lips are slightly swollen, the ears hard and swollen, the eyelids œdematous, and the lower lids everted (ectropion). The skin soon loses its smoothness, and becomes cracked, owing to desquamation, which takes place over the whole cutaneous surface. The scales are white or gray, large on the limbs and trunk, small and pityriasic on the scalp. They are always larger than those of pityriasis rubra. They are only partially adherent, and are sometimes imbricated like the tiles on a roof. The subjacent skin is uniformly red. Desquamation is continual and very abundant, the scales being formed incessantly and filling the patient's bed. It is always dry during the whole course of the disease, except sometimes in the

articular folds, where there is slight exudation which may be fœtid; in these parts the eruption has an eczematous appearance, but this is only a secondary effect, the eruption being essentially dry. The lymphatic glands are often enlarged, especially those of the axillæ and groins.

Lesions of the hair and nails.—These lesions are always present, and occur towards the end of the first month. The hairs are shed on a part or the whole of the body, the atrophy of the hair bulbs being comparable to that of alopecia areata. The nails are less affected; sometimes transversely striated, always dry, yellow, thick and brittle. In some cases they are shed.

Pruritus and its consequences.—Exfoliative dermatitis is a pruriginous affection. Itching is always present at the onset, but becomes less severe after a time, and eventually disappears, reappearing during relapses. It leads to scratching, excoriations, and sometimes slight exudation. Patients also suffer from heat when they are clothed, and from cold when they undress. The inflamed skin may undergo secondary infection, and become the seat of pustules, furuncles, carbuncle, phlegmonous inflammation, and abscess. Sometimes bullæ occur, like those of pemphigus.

General symptoms.—Fever is always present, with evening rises of temperature. At the onset the temperature may exceed 40° C., and always rises during an outbreak or complications. After a time the patient becomes wasted and debilitated, and suffers from loss of appetite and insomnia. Diarrhœa alternates with constipation.

Complications.—As a rule, the urine contains neither albumen nor sugar, but the amount of urea may be much lower than normal. The complications reported include articular (hydrarthrosis), cardiac, ocular (iritis, amaurosis), nervous (paralysis and paraplegia) changes, deafness, and mental disorders. The eruption may spread to the conjunctiva, and to the nasal, buccal, lingual, and pharyngeal mucous membrane, giving rise to more or less acute inflammation, with pseudo-membranous exudation and ulceration, and enlargement of the corresponding lymphatic glands.

Period of Decline.—In simple cases the disease lasts two or three months, the eruption then gradually disappears, the hairs and nails regrow, and the general health is re-established. Pigmented spots remain long after the eruption has disappeared. The duration may be prolonged by complications, relapses, and successive outbreaks up to six months or a year. However, even these cases usually recover; but death may occur from pulmonary, digestive, or renal complications, or from cachexia. Lastly, exfoliative dermatitis, like scarlatiniform erythema, may recur several times.

Pathological Anatomy.—In the *dermis* there is congestion and dilatation of the vessels, with embryonic infiltration of the papillæ and superficial part of the dermis.

In the *epidermis* there is disturbance in keratinisation. During the whole period of the formation of squames the eleidine disappears, and there is no stratum granulosum nor stratum lucidum. The cells of the stratum corneum contain nuclei, easily stained by carmine.

When the squames are no longer renewed, the Malpighian body and stratum granulosum are thicker than normal, and the cells of the latter contain many grains of eleidine; the stratum lucidum also reappears; the cells of the stratum corneum resume their normal characters and no longer contain nuclei.

The nerve changes described by some observers in exfoliative dermatitis are probably due to errors in technique.

DIAGNOSIS.—Exfoliative dermatitis is distinguished from *scarlatiniform erythema* by its evolution; from *pemphigus foliaceus* by the dryness of its squames, absence of bullæ, and tendency to recovery. *Eczema rubrum* is never so generalised as exfoliative dermatitis; its patches are circular and covered with small vesicles, some of which rupture and give rise to abundant exudation, which dries and forms small crusts; the duration of eczema rubrum is two or three weeks. In *scarlatiniform psoriasis* the eruption is papulo-squamous, and there are often characteristic patches of psoriasis elsewhere.

TREATMENT.—The chief indications are to moderate the cutaneous outbreak and favour diuresis. During acute outbreaks an exclusive milk diet should be ordered, afterwards a more nourishing diet from which fermentable foods are excluded. Vichy water or lime water should be added to the milk, especially if there is diarrhœa or digestive disturbance. There is no known drug, not even arsenic, which is capable of arresting the disease. Tonics may be prescribed, and quinine during febrile attacks.

Locally, linimentum calcis should be applied, or simply powdered starch, covered with wool. When the inflammation is very acute, prolonged baths, starch poultices and moist applications are indicated. Itching may be relieved by camphor or menthol ointment (1 per 100). Later on, zinc ointment may be used.

Primary Chronic Pityriasis Rubra.

This affection, described by Hebra, is less common in France than in Austria, where, however, it is also rare and only affects men. Its causes are imperfectly understood, but it has recently been connected with the toxinic cutaneous tuberculides, and I think with reason, for patients affected with this dermatosis often succumb to pulmonary tuberculosis.

SYMPTOMATOLOGY.—The *onset* is slow and insidious, without

prodromal symptoms. Red squamous patches appear simultaneously on several parts of the body, but principally on the articular folds (Kaposi). The patches are bright red and covered with fine pityriasic squames; they gradually extend and become confluent. Eventually the eruption covers the whole body, after several months, a year, two years or longer.

At its *acme*, the body presents a general redness covered with squames, without any other eruptive lesion. The squames, contrary to those of exfoliative dermatitis, are small, sometimes very small, and always dry. In exceptional cases they are a little larger. On the hands and feet the epidermic debris is thick, as if stratified. The dermis is thickened, and appears to be œdematous in places; in other places it is indurated. The whole skin is stiff and tense, hindering movement. Sometimes the redness of the skin becomes darker, purple, or even livid on the lower limbs. Itching is moderate in most cases. According to Hebra and Kaposi, patients suffer from continual shivering, and an almost constant sensation of cold.

In the *third stage*, the dermis, at first infiltrated with embryonic cells, undergoes a process of sclerosis, with all its consequences. The skin becomes atrophied, thin and shrunken, and stretched over the subjacent parts, impeding movement, so that patients close their eyes and open their mouths with difficulty. The palms and soles are hypersensitive, rendering prehension and walking impossible. The hairs become fragile and fall out; the nails may be thinned or thickened, but are always brittle.

As the disease advances the general health deteriorates, the patient becoming gradually weaker. The skin ulcerates in places; digestive disorders and colliquative diarrhœa supervene, and are followed by cachexia and death. Sometimes the end is hastened by pneumonia, or more often by pulmonary tuberculosis.

Benign Chronic Pityriasis Rubra. — Besides the malignant form of chronic pityriasis rubra, there is a benign or comparatively benign form. This has a much slower evolution than the malignant form, but the general health remains fairly good. This benign form has been observed by Vidal, Brocq, and myself.

It develops gradually in finely squamous patches, which are at first isolated and gradually coalesce, but for a long time leave intervals of healthy skin. The eruption becomes generalised, with the same objective and subjective phenomena as in the malignant form; the hairs and nails become altered and shed; ulcers and other trophic disorders may appear, especially on the lower limbs. But an interesting point which differentiates this benign form from malignant pityriasis rubra, is the recurrence of temporary remissions; the eruption disappearing for a time in places, the nails even growing again. This is not a cure but only a remission, for the

eruption always reappears, after a variable time, with the same characters. It may last many years with remissions and fresh outbreaks, but is never cured. However, the general health remains for a long time comparatively good, and renders the prognosis of this form much less grave than that of the malignant form.

PATHOLOGICAL ANATOMY.—According to Hebra, there is first of all embryonic infiltration of the dermis with fibrous tissue formation, then sclerosis with destruction of the glands and hair follicles, and thinning of the Malpighian layer. Sections in the late stages of the disease have the appearance of a cicatrix covered with a thin epidermic layer.

DIAGNOSIS.—Chronic pityriasis rubra is easily distinguished from *eczema* and *psoriasis*. When the latter become generalised and transformed into secondary pityriasis rubra, the diagnosis is established by the remains of the former eruption on some part of the body. In *pemphigus foliaceus*, the squames are large and moist, and there are remains of bullæ. Pityriasis rubra is sometimes difficult to diagnose from certain *premycosic erythrodermias*.

TREATMENT.—No medication has prevented death in any known case. The treatment is only palliative. Continuous baths sometimes give relief.

Secondary Chronic Pityriasis Rubra.

Some time ago I described a form of secondary pityriasis rubra occurring as a malignant transformation of some other chronic dry dermatosis, usually *chronic psoriasis*. This secondary form has the same aspect and the same fatal evolution as the primary form, and has the same small, dry squames. The three cases I observed died of pulmonary tuberculosis.

This secondary pityriasis rubra must be distinguished from *secondary pemphigus foliaceus*, which is a moist affection, also secondary to other chronic dermatoses, especially chronic eczema and chronic bullous pemphigus. It is also distinct, especially in evolution and prognosis, from *secondary exfoliative dermatitis*, described above. The latter is secondary to chronic eczema; secondary malignant chronic pityriasis rubra may also follow chronic eczema, but more often chronic psoriasis.

To sum up, there is a benign form of primary chronic pityriasis rubra, which we call primary exfoliative dermatitis; there are *two secondary forms of chronic pityriasis rubra:* a benign form, or secondary exfoliative dermatitis; and a malignant form, or secondary chronic pityriasis rubra.

DISEASES OF THE SEBACEOUS GLANDS AND HAIR FOLLICLES.

SEBORRHŒA.

THERE are two forms of seborrhœa, *seborrhœa oleosa* and *seborrhœa sicca*, according as the sebaceous matter remains in the liquid state, or dries on the surface of the skin. These two forms may occur in the same subject.

Seborrhœa Oleosa.

This is due to hypersecretion of the sebaceous glands. According to Unna, it depends as much on altered secretion of the sweat glands as on sebaceous hypersecretion, a view which receives support from seborrhœic eczema.

SYMPTOMATOLOGY.—It is characterised by a continual discharge of sebaceous matter on the surface of the skin, which becomes covered with a layer of grease, sometimes gray from admixture with dust. The subjacent skin is normal or slightly reddened, the orifices of the glands are dilated and discharge sebaceous matter, which has a stale odour, sometimes fœtid in generalised and intense forms.

There are no itching or burning sensations. The face is the seat of predilection, especially the sides of the nose, cheeks and forehead; it also occurs on the sternal region, back, limbs and genital organs. In rare cases it affects the whole cutaneous surface. Under the microscope the sebaceous matter shows altered epithelial cells, fatty granules, and crystals of cholesterine. It is a benign affection, but very rebellious to treatment.

ETIOLOGY.—This is the same as that of *acne*, which we shall study later. Two special causes of oily seborrhœa have been pointed out by Hillairet: (1) Syphilis; in some cases of secondary syphilis there is generalised seborrhœa, which appears to be of syphilitic origin, as it yields to specific treatment: (2) the influence of the nervous system. Hillairet has reported a case of seborrhœa which was due to transitory congestion of the spinal cord with paralysis of the four limbs, and was cured with the nervous affection.

DIAGNOSIS.—This is very easy. Sometimes oily seborrhœa occurs with dry seborrhœa, which we shall study next.

TREATMENT.—The affected parts should be treated with lotions containing bicarbonate of soda or borax and ether:

Sulphuric ether Borax	. . . .	aa 15 parts
Water	. . . . .	250 „

After washing with hot water, astringent lotions may be used; such as tannin, alum or resorcin (1 or 2 per 100). Local applications should always be in the form of lotions or powders; ointments and oily preparations should never be used.

Seborrhœa Sicca.

This results from the concretion of the sebaceous matter, mixed with dust and dirt. The crusts are generally soft, even when dry, and leave a characteristic grease mark on blotting paper, which is not produced by the crusts of eczema. The crusts consist of a mixture of sebaceous matter and epidermic squames.

SYMPTOMATOLOGY.—Dry seborrhœa affects the scalp, where the crusts adhere to the hairs, which raise them up as they grow. When the disease has lasted some time there is partial loss of hair. On the smooth parts, the crusts adhere more closely to the skin, sending prolongations into the glandular orifices. Underneath the crusts the skin is generally normal, sometimes slightly red and swollen; sometimes the congestion of the skin extends beyond the crusts, and the tissues are slightly thickened around them. The other seats of predilection of dry seborrhœa are: the nose, cheeks, forehead, eyebrows, external ear, chin, articular folds and genital organs, where seborrhœa may be followed by painful erosions with purulent discharge. The eruption forms patches of variable extent, sometimes covering large surfaces. In some persons, especially young girls, the whole face may be covered with a mask of crusts, which are reproduced after removal.

Fœtal Seborrhœa.—This is a variety of general seborrhœa. It consists more of epithelial debris than sebaceous matter. The crusts which occur on the scalp of newly born infants (*vernix caseosa*) are seborrhœic in nature.

Senile Acne Sebacea, or Local Seborrhœa.—This is an affection which occurs in adults, especially at an advanced age. It consists of a small patch with sharply defined borders covered with a crust, situated on the face, usually on the temples, nose or cheek. It is generally single, but may be multiple. After a time the crust falls off or is removed by scratching; the skin underneath then appears

red and greasy. The under surface of the crust presents processes which penetrate the dilated orifices of the sebaceous glands; it is always renewed when it falls off. In rare cases the crust is eventually no longer produced, and a cicatricial depression is left in its place.

In most cases the patch remains stationary indefinitely, but sometimes it extends; the subjacent skin is then red and friable, and bleeds easily; this is the first degree of transformation into epithelioma; later on, when the crust is removed the skin is ulcerated and vegetating, presenting all the characters of epithelioma. This epitheliomatous transformation may occur spontaneously or under the influence of some cutaneous irritation, such as scratching, or the application of caustics, which irritate the lesion instead of destroying it.

Senile Seborrhœic Warts.—These are flat, slightly raised papillomas, occurring on the face or on the back of the hands in old people. They appear in the form of gray spots covered with a very adherent seborrhœic coating. They are sluggish lesions, usually of no consequence, but sometimes degenerate into epithelioma.

DIAGNOSIS. — Dry seborrhœa is easily distinguished from *seborrhœic eczema* by the fatty nature of the crusts and by the absence of any subjacent eruption. In *simple eczema* the crusts are dry, laminated, and preceded by exudation. The crusts of *impetigo* are yellow and associated with pustules. In *pityriasis simplex capitis* the squames are dry, but often accompanied by dry seborrhœa. On the face, dry seborrhœa must be distinguished from *acneic lupus;* the latter is formed by a hard, red patch, covered with a dry, adherent, squamous layer, cicatricial in places, and surrounded by a red ring.

TREATMENT.—The crusts must first be removed by means of moist dressings or starch poultices. The greasy matter covering the skin can then be dissolved by decoction of quillaia, or solutions of borax or bicarbonate of soda with ether. After this, sulphur lotion, alcoholic solutions of naphthol ($\frac{1}{2}$ to 1 per 100), or resorcin (1 to 2 per 100) may be applied.

In local acne sebacea, compresses soaked in a saturated solution of chlorate of potassium give good results; also a chlorate of potassium ointment with vaseline (20 per 100). The compresses should be frequently renewed and the ointment applied continuously. (For the treatment of epitheliomatous transformation, see article on *Epithelioma.*)

Senile seborrhœic warts are easily destroyed by glacial acetic acid; this may also be used for small patches of local seborrhœa.

A course of hydrotherapy at one of the mineral spas is also useful in seborrhœa.

COMEDONES.

Comedones result from disordered secretion of the sebaceous glands and retention of sebaceous matter in the glands and their ducts.

Symptomatology.—Comedones have the appearance of black points (*acne punctata*) situated at the orifices of the glands; their black appearance is due to the mixture of sebum with dirt. If the base of the gland is pressed between the finger nails, a white filament formed by sebum emerges from the glandular orifice. Microscopic examination of this filament sometimes reveals an animalcule (*Demodex folliculorum*); this is an arachnid which plays no part in the production of the comedo, as was formerly supposed, and is found on the normal skin.

Comedones occur chiefly on the forehead and on the sides of the nose, sometimes in such numbers that the skin of the face appears studded with grains of powder. They also occur on the back and shoulders, sternal region and external ear, sometimes on the arms, and even on the penis.

They cause no functional trouble nor itching, but they constitute a troublesome disfigurement, especially when confluent. The comedo may become inflamed and suppurate, forming *pustular acne*, which we shall study later. Lastly, comedos sometimes coexist with oily or dry seborrhœa, which are also due to disordered secretion of the sebaceous glands.

Some authors have described a special form of comedo peculiar to boys under fifteen years of age, characterised by confluent black points forming circular patches on the forehead, temples and cheeks. After expression, these comedos leave cicatrices.

Etiology.—This is not very well understood, but the affection is chiefly observed in lymphatic subjects. The cause, like that of pustular acne, must be sought for in a functional disorder of the digestive system, and especially in the imperfect digestion of fat.

This affection has also been observed in workmen employed in the manufacture of chlorine (*chloric acne*). In these cases the comedos are large and numerous, and may cover the whole body; in some places they become transformed into pustular acne. According to Jacquet, the lesion is not caused by the chlorine vapour, but by substances used in the manufacture of chlorine.

Treatment.—Attention must first be directed to the digestive system. Gastro-intestinal fermentation must be checked by antiseptic cachets, and fatty foods excluded from the diet. Pancreatine may be prescribed to aid the digestion of fatty matter.

Locally, the follicles should be emptied by pressing the base

of the gland with the finger nails, or, better, by means of a *comedo-expressor*. If the comedos are confluent, this procedure is impracticable. The fatty matter should then be removed by means of hot water and soap, and lotions of bicarbonate of sodium, borax and ether.

Unna uses the following preparation:

Acetic acid	2 parts
Glycerine	3 „
Kaolin	4 „

This is applied to the comedos morning and evening; after which they are expressed. Ointments should be avoided.

Local douches with hot sulphur or alkaline thermal waters are also useful, as well as the application of alcoholic preparations, which cause contraction of the dilated follicles.

MILIUM, OR GRUTUM.

This condition results from fibroid or calcareous induration of the sebaceous glands. It appears in the form of whitish or yellow projecting granules, the size of a pin's head or millet seed. These concretions may be isolated or grouped, and are usually superficial and subepidermic, rarely intradermic. They occur chiefly on the face, especially on the eyelids, forehead and cheeks, near the orbit, fairly often on the neck and on the genital organs. Hardy described a larger variety of milium affecting the skin of the scrotum (*acne pisiforme*).

At the first appearance of milium, a little sebaceous matter may be expressed by pressing the granule between the fingers, but after a time the duct of the gland becomes obliterated. Milium is a very common affection, and causes no functional trouble; generally there are several granules, sometimes a large number. The lesion does not retrogress. It sometimes coexists with comedones or pustular acne. It is especially common in dyspeptic and nervous women.

Pathological Anatomy.—Each granule consists of a fibrous pouch formed by thickening of the wall of the follicle. This is closed on all sides, and contains an accumulation of gland cells, dessicated and arranged like the leaves of an onion. In the centre is a mass of fatty matter and crystals of cholesterine. Sometimes the contents are infiltrated with lime salts, making the granules as hard as stone. These chalky concretions were formerly called cutaneous calculi.

Treatment.—Each granule may be enucleated with a small curette, after incising the skin. The pouch is then cauterised with tincture of iodine or nitrate of silver. This treatment is often impracticable, owing to the multiplicity of the lesions.

INFLAMMATORY OR PUSTULAR ACNE.

This affection is characterised by red, painful papules with an indurated base, which often suppurate and form pustules, but sometimes undergo spontaneous resolution, or remain in the indurated state. These papulo-pustules occur on the face, back, shoulders and chest, and vary in size from a millet seed to a pea.

When the papules suppurate, a white spot appears in the centre of each papule, due to the presence of pus beneath the epidermis. Three or four days later, the pustule ruptures, the pus escapes, the papule gradually subsides, and the redness slowly disappears. When the pustules are large and the suppuration deep, cicatrices are produced. The pus often dries and forms small crusts on the top of the papules. Lastly, the pustule, after being partly emptied, may persist as a small, hard nodosity (*acne indurata*).

Inflammatory acne appears in successive crops, often encroaching on one another, so that all stages of evolution are met with in the same subject: papules, pustules, crusts, nodosities, and sometimes cicatrices. Comedones and seborrhœa are often associated with these lesions.

Several varieties of pustular acne have been described: *simple acne*, which consists of small isolated pustules, with little pain or inflammation; *juvenile acne*, which disappears spontaneously towards the thirtieth year; *phlegmonous acne*, in which the suppuration is not limited to the sebaceous gland, but extends to the surrounding tissues, and forms a small abscess, which leaves a cicatrix. These varieties may all be met with on the face in the same patient, generally a lymphatic or scrofulous subject, constituting *polymorphous acne*.

Prognosis.—Acne is a benign affection as regards the general health, but it is very disfiguring, especially when confluent. It is often very rebellious to treatment, and sometimes leaves cicatrices.

Pathological Anatomy.—The characteristic lesion is constituted at first by a perifolliculitis, then by a suppurative folliculitis. Comedones, when inflamed, may also give rise to acne. Suppuration in the papules of acne results from external infection of the sebaceous glands by staphylococci, especially the *staphylococcus albus*.

Diagnosis.—The only lesion with which acne can be confused is the *papular syphilide*. This mistake is often made, even by experienced physicians, who are afraid of diagnosing syphilis in private practice, especially in women. But the papules of syphilis are flatter than those of acne, coppery in colour, and not pruriginous;

also, they never suppurate. Acne is easily distinguished from the pustules of *sycosis,* which is generally due to infection of the pilo-

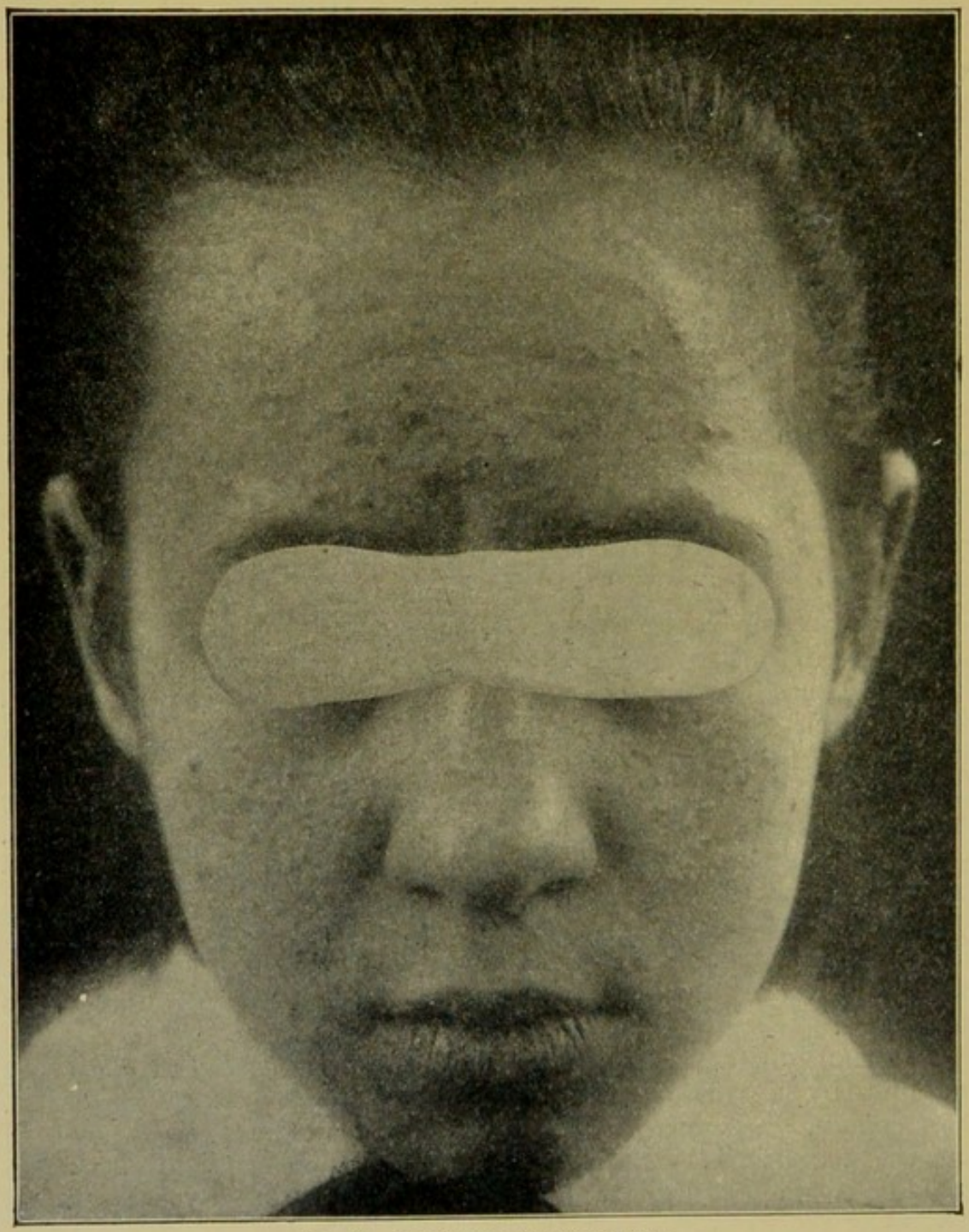


FIG. 41.—Papulo-pustular juvenile acne.

sebaceous follicles of the beard by the *trichophyton tonsurans;* the pustules of sycosis have a longer duration, and leave nodular indurations, larger than those of acne; moreover, they are always confined to the hairy regions.

ETIOLOGY.—Acne may be produced by the application of irritating substances to the skin, such as tar, oil of cade, pyrogallic acid, or chrysarobin. It may also occur after the internal administration of bromides and iodides. It is possible that these internal drugs act, not only by their cutaneous elimination, but also by the digestive disturbance which they produce. This hypothesis is supported by the results obtained by Féré, who succeeded in suppressing or attenuating bromide acne in epileptics who were taking large doses of bromide, by the simultaneous administration of cachets of naphthol.

The principal cause of spontaneous acne lies in functional disturbance of the digestive organs. Patients with acne often suffer from dyspepsia or dilatation of the stomach. I have for a long time taught that acne patients generally suffer from *hypochlorhydria*. "Dyspepsia favours the development of seborrhœa, then occurs an eruption of acne, which is only the result of inoculation of the seborrhœic skin by external microbes" (Barthelemy). In acneic patients, the outbreaks are often determined by the ingestion of spiced foods, game, pork, fermented cheese, alcoholic drinks, coffee or tea. But all dyspeptics do not suffer from acne; a certain predisposition is necessary, but this is still obscure. It is true that acne is often seen in lymphatic subjects, and sometimes in arthritics; but among the latter, cicatricial acne and acne rosacea are more common. Some authors think that there is a relation between acne and affections of the genito-urinary organs, but this idea is exaggerated. However, some women suffer from acne at each menstrual period.

TREATMENT.—In medicamentous acne, treatment consists in suppressing the cause and applying emollients. Antiseptic cachets may be prescribed.

In spontaneous acne, the following articles of diet should be forbidden: alcohol, tea and coffee, highly seasoned foods, game, fish, shellfish, high cheese, soups, sauces, and all fatty substances. Dyspepsia or dilatation of the stomach, if present, must be treated. Patients with hypochlorhydria should take a small wineglass of dilute hydrochloric acid (·3 per 100) at each meal; in cases of hyperchlorhydria, which are rare, alkalis are indicated. Cachets of benzonaphthol may be given to obtain intestinal antisepsis, and laxatives for constipation.

Diathetic drugs are of little importance: in arthritic subjects alkalis may be prescribed, and arsenic in lymphatic persons. Sulphur has been recommended, which probably acts as a laxative. Ichthyol, which has been recommended internally, has no effect.

Local Treatment.—Every evening one of the following lotions

may be applied by means of aseptic wool tampons, and left to dry during the night:—

(1) Solution of camphor in alcohol or eau-de-Cologne.

(2) Solution of perchloride of mercury (1 in 1000 to 1 in 500), with equal parts of water and alcohol.

(3)

Precipitated sulphur	15 parts
Camphorated alcohol	10 „
Water	250 „

(4)

Sulphide of potassium	1 part
Tincture of benzoin	2 parts
Distilled water	100 „

The best way of using sulphur is to apply the following preparation every evening, and leave it on during the night:—

(5)

Precipitated sulphur	6 parts
Powdered talc	2 „
Glycerine	60 „
Tincture of quillaia	10 „
Rose water	120 „

Every morning the skin should be washed with warm water, and a sedative ointment applied, such as zinc oxide or subnitrate of bismuth. If the skin is greasy, it may be dusted with the following mixture:—

Oxide of zinc	10 parts
Powdered starch	20 „
Boric acid	4 „

or with powdered talc mixed with precipitated sulphur (2 per 100).

In rebellious cases, soft soap, applied for four hours every evening, gives good results. Irritation may be relieved by tepid spraying, followed by a simple ointment.

It is often necessary to combine surgical treatment with the above measures. This consists in puncturing the pustules once or twice a week with a fine lancet, evacuating the contents, and then washing with a solution of resorcin (1 per 100). Acne is only cured when the small glandular core is evacuated as well as the pus. The thermo-cautery and galvano-cautery are not to be recommended, as they leave indelible cicatrices.

Hydrotherapy, in the form of sulphur baths and waters, is useful in generalised acne.

ACNE ROSACEA.

This affection is constituted by the combination of three different lesions: (1) erythematous congestion of the skin and permanent dilatation of the cutaneous capillaries; (2) the production of papules

and pustules similar to those of inflammatory acne; (3) hypertrophy of the sebaceous glands and proliferation of the periglandular tissue. Congestion of the skin and periglandular proliferation give acne rosacea a special character which distinguishes it from other forms of acne. These three elements, according as they are isolated or combined, give rise to three forms of the disease: (1) a *congestive* form, with or without *telangiectases;* (2) a *congestive, telangiectasic and papulo-pustular* form; (3) a *hypertrophic* form.

Symptomatology.—Acne rosacea occurs on the face; on the cheeks, nose and forehead. Bazin and Hillairet have observed it on the chest. It begins in the form of red congestive patches, which are at first intermittent, and appear after meals or exposure to cold; they are accompanied by heat and tension of the skin. Later on, the patches become permanent and the redness is complicated by dilatation of the cutaneous capillaries (*telangiectasis*). These telangiectases are at first visible only with a lens, but become visible to the naked eye as they grow larger. They are sometimes of a purple colour, especially in drinkers. The skin is turgid, slightly thickened and seborrhœic, presenting dilatations of the sebaceous orifices.

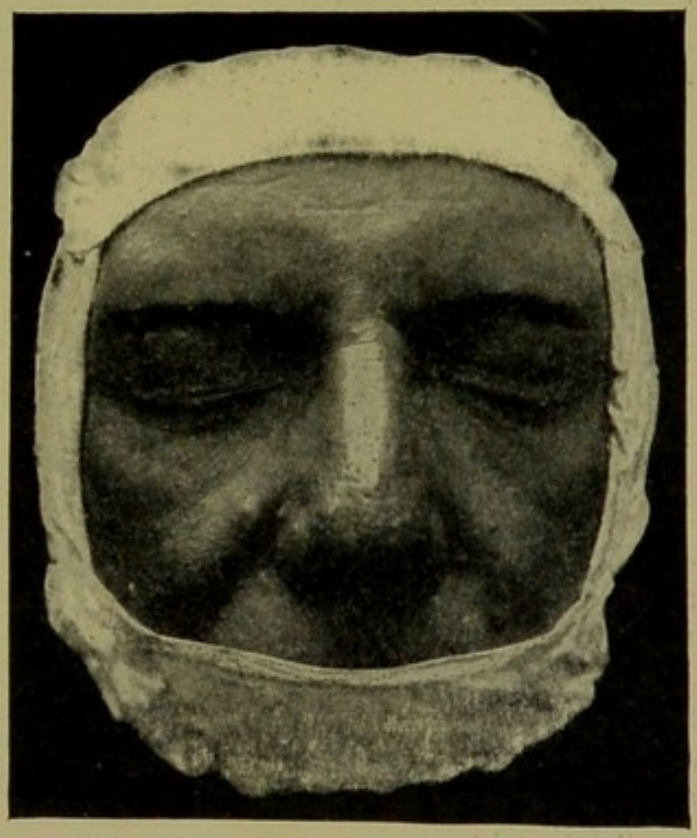


Fig. 42.—Acne rosacea. (St Louis Hospital Museum.)

At a later stage, acne papules develop on the red patches; these may remain papular, but often become pustular, presenting the characters of ordinary acne. Sometimes these papulo-pustules appear first, or at the same time as the congestion. In many cases acne rosacea stops at this stage; in other cases, it is complicated by connective tissue proliferation, constituting *hypertrophic acne.*

In this form, also called *elephantiasic acne* or *rhinophyma,* the affected parts are red or purple, thickened and irregularly mammillated, and traversed by dilated capillaries. The hypertrophy affects the sebaceous glands and the periglandular connective tissue. The orifices of the glands are dilated, and at the periphery of the

thickened parts are seen a few acne pustules. In some cases of hypertrophic acne the skin preserves its normal colour, there is no congestion nor telangiectases, but only a tendency to hypertrophy of the tissues. The nose may attain enormous dimensions, so great as to interfere with feeding and respiration.

PROGNOSIS.—Acne rosacea is benign, but very chronic. It causes more disfigurement than any other form of acne.

DIAGNOSIS.—It is sometimes difficult to distinguish acne rosacea from *lupus erythematosus* or from *acneic lupus*. Lupus erythematosus has a darker colour, and is formed by a slightly raised patch with sharply defined borders, while acne rosacea is diffuse; moreover, the surface of lupus presents crusts, squames and small white cicatrices; lastly, lupus has no precise distribution, while acne rosacea affects the cheeks and nose. Acne rosacea may also be mistaken for chilblain of the nose (*erythema pernio*), but the latter is an acute affection occurring in winter, accompanied by tumefaction of the parts, which does not occur in acne rosacea. *Acneiform syphilitic lesions* of the face are distinguished by their coppery colour, sometimes by their tendency to ulceration, and by their secondary cicatrices. *Seborrhœic eczema* of the face is sometimes very difficult to distinguish from acne rosacea; but, in seborrhœic eczema, the redness is not so bright and less diffuse, there are no telangiectases, and the red patches are covered with fatty crusts. However, seborrhœa is the principal cause of acne rosacea, so that seborrhœic eczema and acne rosacea belong to the same family. *Keratosis pilaris* is distinguished from acne rosacea by its finely granular appearance, and by its special distribution on the sub-malar and pre-auricular regions.

ETIOLOGY.—This is essentially that of ordinary acne, but there are a few special causes. Acne rosacea is more frequent in women, especially at the menopause. It is often hereditary. Alcoholism is an accessory cause. Hebra distinguished different colours according to the nature of the alcoholic drinks consumed; bright red in wine-drinkers, purple in beer-drinkers, and dark blue in spirit-drinkers. However, acne rosacea may occur in very sober individuals. Disorders of digestion and dilatation of the stomach are frequent causes of acne rosacea. But, apart from all these exciting causes, there is a primordial predisposing cause ; this is *arthritism*.

Other secondary influences have been mentioned, but these only act as exciting causes in predisposed subjects; residence at the sea-side, cold climates, and exposure to change of climate, for example. Persons affected with acne rosacea usually suffer from cold feet.

TREATMENT.—The general treatment and diet are the same as for inflammatory acne. As arthritism is often present, alkalis should be prescribed. Some authors recommend hammamelis, but I have

never found this efficacious. Cutaneous irritation and atmospheric changes should be avoided.

Local treatment is similar to that for ordinary acne. The face should be washed or sprayed every morning with hot water. Cutaneous irritation may be relieved by zinc ointment. Every evening the following lotion should be applied:—

Precipitated sulphur	15 parts
Camphorated alcohol	15 „
Water	250 „

This treatment should be carried out on five or six consecutive days, with intervals of two or three days, for two or three months.

Lotions of perchloride of mercury (1 in 1000 to 1 in 500), or chloride of ammonium (1 per 100), sulphur ointment, or soap plasters, may also be used.

In severe cases, linear scarification may be performed. The scarifications are made perpendicularly or obliquely to the dilated vessels, very close together, and parallel; after making one series, another series of scarifications is made crossing the first; they should be deep enough to cut the dilated vessels. These scarifications relieve the congestion of the tissues, and produce inflammation of the vascular walls, which leads to their obliteration. Hæmorrhage is arrested by hot water and pressure. The scarifications should be repeated every week for some time; in the intervals, powders or ointments may be applied, according to the condition of the skin.

In hypertrophic acne, scarification is often insufficient. The actual cautery may then be used, or, better, surgical removal of the diseased parts—decortication of the nose.

Mineral waters may be prescribed as in ordinary acne.

CICATRICIAL ACNE PILARIS.

This is also called *atrophic acne, acne necrotica*, or *acne variolæformis*. It is common in arthritic subjects, and may be local or general.

Local form.—This occurs chiefly on the forehead near the border of the hair, on the temples, sides of the nose, cheeks and chin. On the scalp, this affection is known as *folliculitis decalvans*.

General form.—The lesions affect not only the face and beard, but also the back and chest.

In both these forms the disease is characterised by red papules, occasionally purple, varying in size from a millet seed to a hemp seed. The papules suppurate like those of ordinary acne, but the papulo-pustules of acne pilaris are often depressed in the centre,

because they are traversed by a hair. They become covered with a yellow crust which persists for several days; after this has fallen off, a circular, depressed, indelible cicatrix is left, at first red then white, somewhat resembling that of a syphilitic pustule or a pustule of variola (varioliform acne). The eruption appears in successive crops, which leave numerous cicatrices on the affected parts.

PATHOLOGICAL ANATOMY.—The lesion of cicatricial acne is characterised by pilo-sebaceous folliculitis and perifolliculitis, extending rather deeply into the periglandular connective tissue. It is therefore a special form of inflammatory acne.

ETIOLOGY.—This form of acne is generally observed in arthritic subjects, and appears between the thirtieth and fortieth years. Some authors have attributed it to syphilis, but they have confused it with the acneiform syphilide.

DIAGNOSIS.—The *acneiform syphilide* is much more disseminated than acne pilaris, and there are usually other signs of syphilis.

TREATMENT.—This is the same as in inflammatory acne. The following ointment is useful:—

Precipitated sulphur	5 parts
Soft soap	5 „
Lard	30 „

FOLLICULITIS DECALVANS. PSEUDO-ALOPECIA AREATA.

The affection called by Lailler *acne decalvans* has been studied by Quinquaud under the name of *folliculitis decalvans*. It occurs on the scalp in the form of small pustules or abscesses, the size of a pin's head, from the centre of which emerges a hair, which can easily be pulled out. The hair is destroyed and falls spontaneously; it does not grow again, for the inflammatory process produces atrophy of the hair follicle. On the affected parts the scalp becomes smooth and dull white, resembling alopecia areata. The patches of alopecia are irregular, of various dimensions and scattered here and there over the scalp. In their early stages they have a red spot in the centre, the remains of the circumpilary pustule. Quinquaud claims to have found a micrococcus in the pustules, with which he produced folliculitis with loss of hair by inoculation. The only difference between the acne decalvans of Lailler and the folliculitis of Quinquaud is that, after a phase where the loss of hair is preceded by the appearance of folliculitis, the alopecia may continue to develop in Lailler's disease, without any pustule, as in alopecia areata.

Pseudo-Alopecia Areata.—Apart from folliculitis decalvans, Brocq has described, under the name of pseudo-alopecia areata

(*pseudo-pelade*), an affection of the scalp characterised by slight redness and tumefaction of the scalp around a certain number of hairs. These hairs come out easily when pulled upon. The inflammation soon subsides, but the affected parts of the scalp remain white, smooth and atrophied, and destitute of hairs and even down.

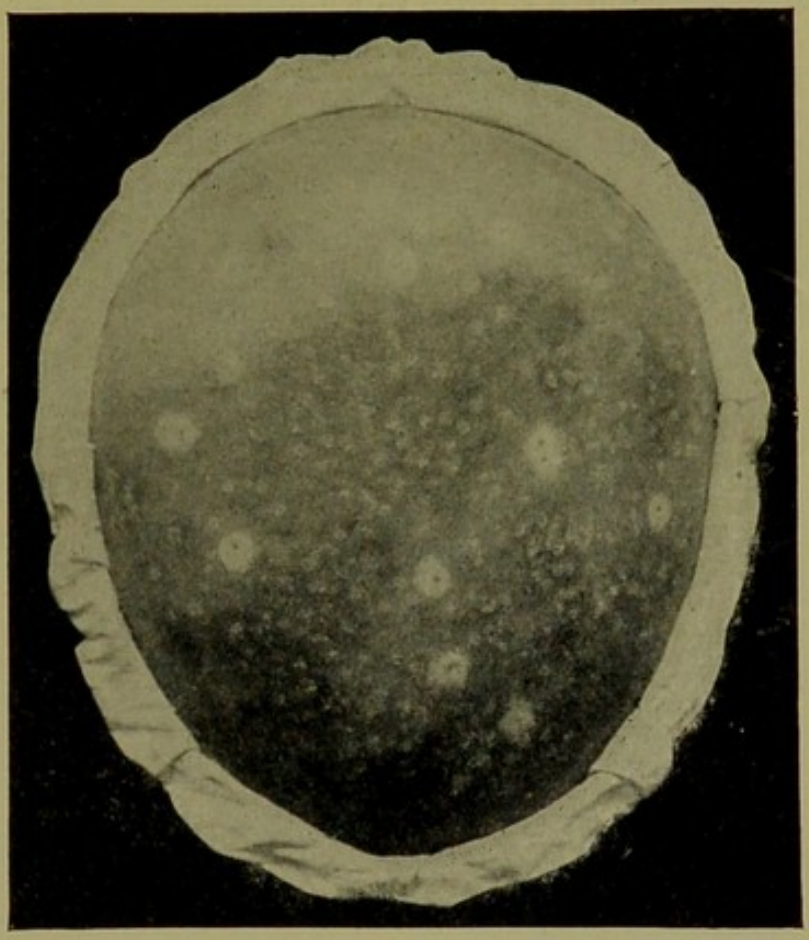


Fig. 43.—Folliculitis decalvans. (St Louis Hospital Museum.)

The inflammation invades the neighbouring parts by irregular prolongations, contrary to what occurs in true alopecia areata.

Diagnosis.—These affections are easily distinguished from true *alopecia areata*, in which the scalp never shows any signs of inflammation, nor of acne pustules.

Treatment.—A lotion of perchloride of mercury (1 in 500), or resorcin (1 in 100) should be applied to the patches every morning, followed by sulphur or turpeth ointment (10 per 100).

CHELOID ACNE.

This is a form of acne pilaris in which the follicular and perifollicular inflammation ends in deep fibrous induration of the dermis resembling cheloid.

Cheloid acne occurs almost exclusively on the nape of the neck below the hair. It begins with a group of elements of acne pilaris, around which the connective tissue hyperplasia undergoes fibrous transformation. This results in the formation of hard nodosities, more or less voluminous and of a deep red colour, sometimes covered with small telangiectases similar to those seen in cheloids. Most of

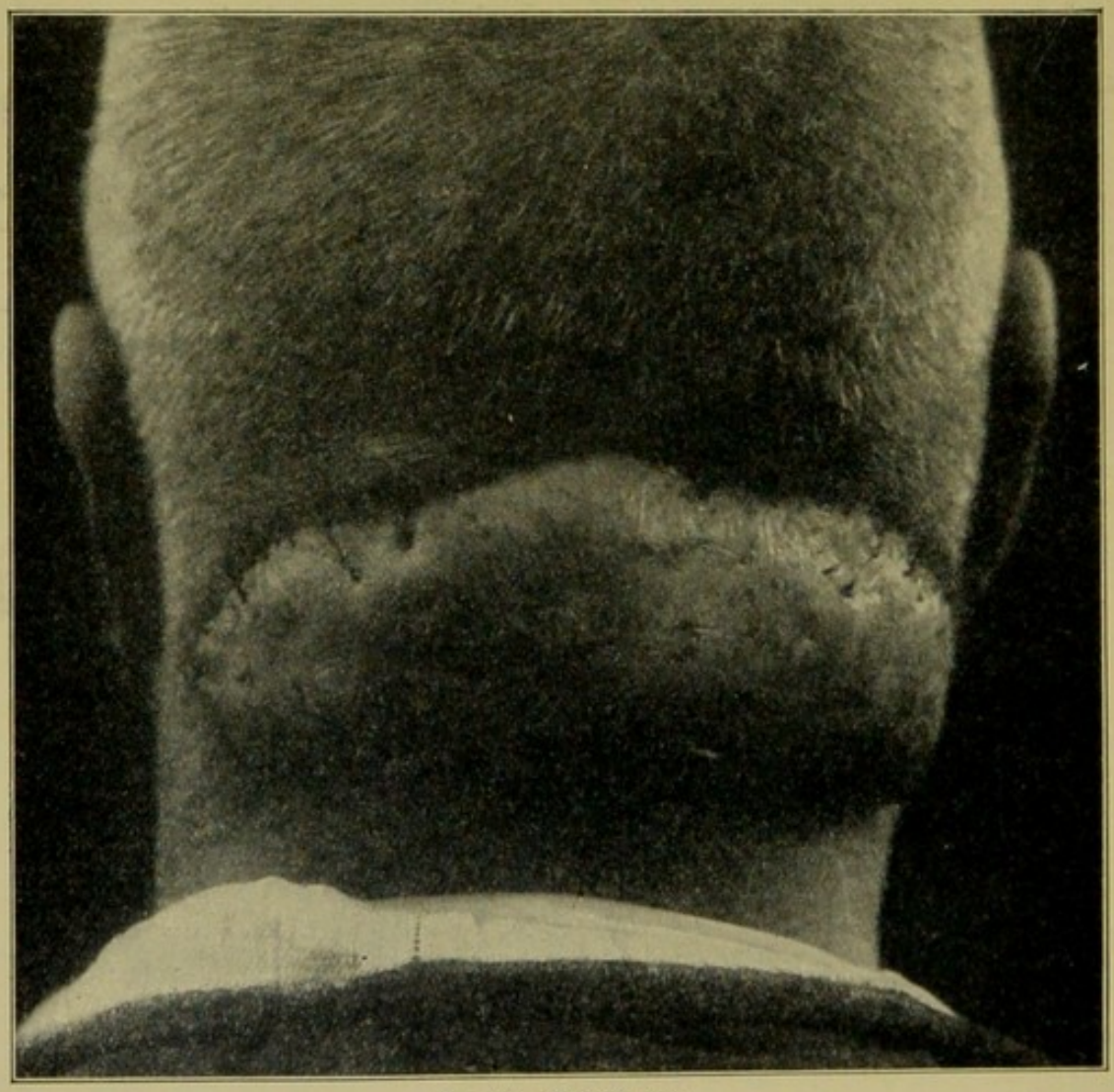


Fig. 44.—Cheloid acne.

the hair follicles are destroyed; in those which survive the hairs are deviated, and often grouped here and there in the form of tufts, which rise perpendicularly or obliquely from the cheloidal patch. Pustules of acne pilaris and inflammatory acne are often present on various parts of the face, showing the acneic origin of the affection. Cheloid acne is very sluggish and very rebellious to treatment.

Etiology.—This is the same as that of ordinary acne. The

thickness of the integuments in the region of the nape of the neck, together with friction against the collar, favour its development.

DIAGNOSIS.—This is easy. Cheloid acne can only be confused with a furuncular eruption; but *furuncles* generally end in suppuration and the expulsion of a core, and the nodosities they leave behind them are not united in the form of patches traversed by hairs, as in cheloid acne.

TREATMENT.—An attempt should be made to abort the pustules by repeated applications of tincture of iodine. When suppuration occurs, the pustules should be evacuated by puncture and pressure, followed by the application of gauze compresses soaked in a saturated solution of boric acid, or perchloride of mercury (1 in 1000); these should be applied day and night. The nodosities which remain after this operation should be covered with mercurial ointment, which hastens their resolution. If this treatment fails, scarification usually gives good results.

But the treatment which seems to me now most efficacious, and superior to scarification, is *radiotherapy*. Another mode of treatment, recently introduced into my clinic, is linear scarification, followed immediately by the application of *high frequency* currents. This method appears to give as good results as radiotherapy, and is free from the dangers of the latter.

SIMPLE SYCOSIS, OR FOLLICULITIS OF THE BEARD AND HAIRY REGIONS.

This is a chronic affection which develops on the hairy regions of the skin, and is characterised by the production of suppurative folliculitis. It is a simple *non-trichophytic sycosis*, distinct from *trichophytic sycosis*, and was described by Bazin under the name of *arthritic sycosis*.

SYMPTOMATOLOGY.—It occurs most frequently on the chin (*sycosis menti*), cheeks and upper lip, more rarely on the eyebrows and in the nostrils; it has been observed on the pubis.

On the face, it appears on one or both sides, in the form of circumpilary pustules, more or less isolated, accompanied by burning or pricking sensations. The skin around the pustules is red and thickened. The pustules multiply and invade the neighbouring regions, coalescing in places to form indurated, mammillated masses. These masses vary in size from that of a pea to that of a nut, or larger. Some of the pustules rupture and discharge a little pus, which dries in yellow or brown crusts. After a time the hairs fall out, but often grow again; but if the inflammation has destroyed the hair papilla, the loss of hair is permanent. Underneath the

crusts are ulcerations, which become covered with red, exuberant granulations which bleed easily. The inflammation may extend to the subcutaneous tissue and produce deep induration, and sometimes subdermic suppuration.

Sycosis of the eyebrows may be associated with chronic blepharitis with destruction of the eyelashes. In the interior of the nostrils, sycosis is often preceded by eczema pilaris. This eczematous inflammation of the nostrils is usually accompanied by chronic coryza. The liquid exuded from the nostrils irritates the upper lip and causes suppuration in the hair follicles of the moustache. This is

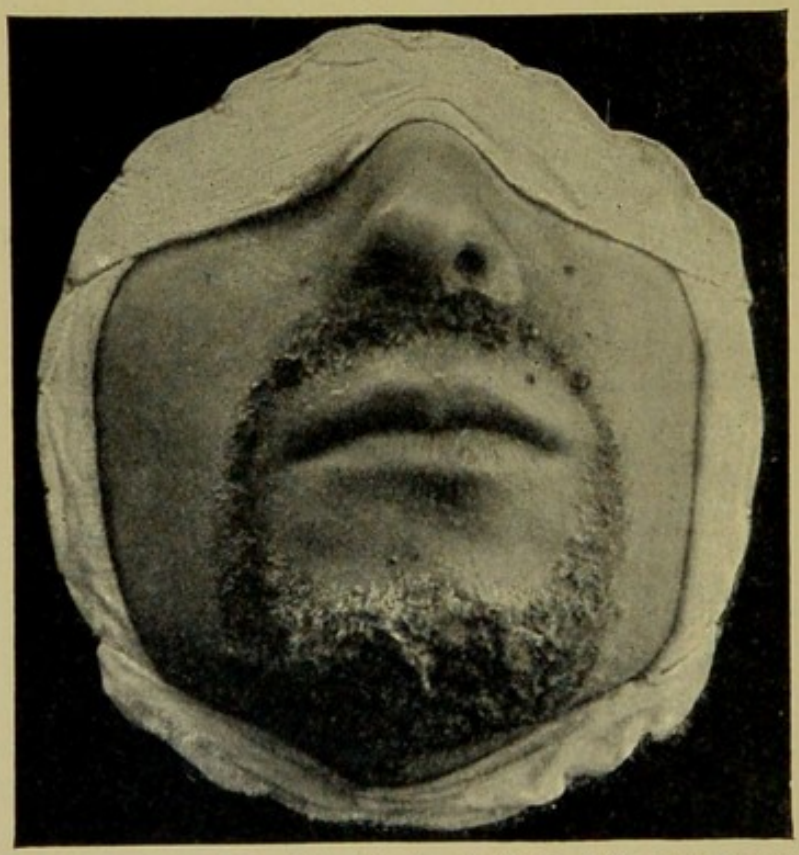


FIG. 45.—Sycosiform folliculitis of the beard. (St Louis Hospital Museum.)

the mode of origin of *sycosiform eczema* of the upper lip, which is the most common form of simple sycosis of the face, and corresponds to Bazin's *arthritic sycosis.* In this form the suppurative inflammation of the hair follicles is usually limited to the moustache. It is a very obstinate affection.

On the pubic region, sycosis is generally secondary to eczema, and is not so severe as on the chin and cheeks.

Sycosis runs a chronic course and may last for years, leaving more or less marked cicatrices with loss of hairs.

ETIOLOGY.—Simple sycosis is generally secondary to eczema, rarely primary. In eczema of the beard, if there is a formation of

peripilary pustules only, without inflammation of the follicles, it is a case of simple *eczema pilaris;* but if the inflammation reaches the follicles, it constitutes *simple sycosis.*

Unna and Bockhart found ordinary pus cocci in the hair follicles in simple sycosis, and called it *coccogenic sycosis,* to distinguish it from trichophytic sycosis. Simple or coccogenic sycosis is due to accidental inoculation of the hair follicles with staphylococci, following a cut by the razor, for instance. Tommasoli claims to have discovered a bacillus, pure cultures of which when inoculated on himself and on rabbits gave rise to a form of sycosis (*bacillogenic sycosis*), which presented similar symptoms to those of coccogenic sycosis, but much more intense.

To sum up, simple sycosis is a microbial dermatosis; most commonly a complication of eczema pilaris, by secondary inoculation of the follicles by staphylococci.

Diagnosis.—*Eczema pilaris* differs from sycosis in the lesions being more superficial, and in the absence of deep pustules forming nodosities; there are sometimes small circumpilary pustules, but these are superficial. However, there are transitional forms in which it is difficult to decide whether they are cases of eczema pilaris or sycosis, especially as eczema pilaris after a time often ends in sycosis.

Lupus of the beard may be mistaken for sycosis, but at the periphery of the patch of lupus characteristic tubercles can usually be found. *Ulcerative syphilides* have hard borders and a coppery colour. Lastly, *trichophytic sycosis* differs from simple sycosis in being often unilateral, in being preceded by pityriasis alba, and in the presence of the trichophyton fungus.

Treatment.—The hair should be cut as short as possible; this is preferable to shaving, which may lead to fresh inoculations. The parts should then be treated with a solution of resorcin (1 per 100) three times a day. Dermic and subdermic abscesses should be opened, and the hairs should be epilated to allow escape of pus. When the follicular infection is not very deep, the application of resorcin lotion is often sufficient without epilation. In this form of cutaneous suppuration resorcin seems to be almost a specific.

Epilation should be performed at several sittings, on account of its painfulness. In sycosis of both cheeks, two or three weeks are necessary to remove all the hairs. After each sitting the resorcin lotion should be applied by means of a small steam spray; or, if the inflammation is too acute, boracic starch poultices may be applied. After the inflammation has subsided, ointments should be used containing calomel, turpeth, or yellow oxide of mercury; or, if these are not well tolerated, styrax and oil of almonds (1 in 2), or

sulphur, tar or tannin ointments. A very good ointment is the following:—

Tannin	1 part
Precipitated sulphur	2 parts
Vaseline	20 „

These ointments should alternate with sedative applications. Epilation should be repeated as long as the new hairs do not appear healthy and well implanted. The treatment of sycosis is very prolonged, especially when it affects the moustache, because in this situation it is constantly aggravated by the nasal discharge which gave rise to it. The diseased hairs must be epilated indefinitely.

Exuberant granulations may be touched with nitrate of silver or tincture of iodine, and nodosities scarified. Asepsis is indispensable.

MOLLUSCUM CONTAGIOSUM.

This affection, named by Bateman in 1817, is characterised by small globular tumours with a central depression.

SYMPTOMATOLOGY.—The lesions are hard and resistant, semitransparent like pearls, the same colour as the skin or slightly reddened, and vary in size from that of a millet seed to that of a pea; sometimes they are as large as a nut. They usually have a broad sessile base, and form small hemispherical tumours, sometimes acuminate or club-shaped; in the latter case they are pedunculated. But the essential character is that each tumour is umbilicated; in the centre there is a depression, from which the contents of the tumour can be expressed, in the form of a semi-solid mass with a milky appearance.

These little tumours are generally isolated, and few in number; but some cases of generalised molluscum contagiosum have been observed. The face is the region usually affected, especially the eyelids, forehead and cheeks; they also occur on the neck and genital organs, more rarely on the back, chest and limbs. They sometimes become confluent and altered in shape by mutual pressure. In the absence of treatment they may persist indefinitely; but they are sometimes cured spontaneously by evacuation of their contents, or by inflammation of the tumour, which suppurates and disappears, leaving a superficial cicatrix.

DIAGNOSIS.—It is impossible to mistake molluscum contagiosum for any other tumour of the skin; its small size, semi-transparency and umbilication render it easily recognisable. *Fibroma molluscum*, even in its early stages, has only a remote resemblance to molluscum contagiosum; it is usually pedunculated, of larger size, and never umbilicated. *Sebaceous cysts* are larger tumours with no umbilication,

and sometimes present a black spot indicating the orifice of the sebaceous gland.

PATHOLOGICAL ANATOMY.—Molluscum contagiosum is due to a *peculiar modification of the sebaceous gland* (Renaut). Some authors have regarded it as a tumour formed by interpapillary prolongations of the epidermis; but if a section of the tumour is made in its early stage, passing through the umbilication, it will be seen that the dermis, as well as the epidermis, is prolonged up to the umbilication, which would not be the case if the tumour originated in the epithelial layers only. In the same section it can be seen that the umbilication is only the excretory duct of the altered sebaceous gland. The latter appears to be spread out in the form of a reversed fan, and its lobules are separated by prolongations of the dermis, which have been mistaken for elongated papillæ by the partisans of the epithelial origin of molluscum contagiosum. If the cells of one of these lobules are examined from below upwards, the central cells of the lobule are seen to become globular, and are filled with hyaline granules which soon coalesce into one or more masses of translucent matter. These hyaline masses (molluscum corpuscles) finally push the protoplasm and nucleus of the cell against the cell wall. Between these modified cells, at the upper extremity of the lobule, are found other cells which arise from the stratum granulosum and are charged with eleidine.

In some of the hyaline masses are found round bodies, enclosed by a fine refractile membrane and containing a nuclear mass, which were regarded by Bollinger and Neisser as gregarines, similar to those found in the rabbit's liver. These round bodies are nothing more than products of cell degeneration, as has been shown by Renaut and confirmed by my own observations.

ETIOLOGY.—Molluscum contagiosum is a contagious disease, as Bateman believed. Cases of contagion have been reported by Cazenave, Hardy, Besnier and others. It is also inoculable (Retzius, Vidal, Haab), and it is by auto-inoculation that the lesions multiply on the same subject; but the nature of the parasite is unknown. The disease is chiefly observed in children and young subjects.

TREATMENT.—The tumours should be incised and the contents evacuated by means of a curette, after which the sac should be cauterised with nitrate of silver. Another method is to destroy the tumours with the thermo-cautery or galvano-cautery. If the tumour is pedunculated it can be removed with scissors. In schools and children's hospitals it is well to isolate the patients in order to prevent contagion.

ACNE CORNEA.

"Acne cornea, described by Cazenave, occurs in the form of yellow, gray or black acuminate elevations, which feel like a file or brush; by pressing the base of these little tumours they project still further and are sometimes expelled, when the gaping orifice of the sebaceous follicle shows their mode of production and where they were implanted. This follicular modification is met with indifferently on all parts of the body, on the forehead, nose, trunk or limbs; these little tumours, sometimes arranged in groups, sometimes disseminated, cause neither itching nor burning, and constitute an inconvenience rather than a real disease. Their progress is very chronic, and the horny secretion may persist for months or years, unless modified by suitable treatment" (Hardy).

The lesion appears to be situated in the neck of the pilo-sebaceous follicle, the horny epidermis of which is considerably thickened. The hairs, included in the plug, become atrophied.

DIAGNOSIS.—Acne cornea must be distinguished from *keratosis pilaris*, which occurs in infancy, and chiefly affects the posterior surface of the arms and antero-external surface of the thighs. *Pityriasis rubra pilaris* is characterised by elevations pierced by a hair, covered with a dry adherent squame, and situated on an erythematous base, and by pityriasic desquamation, more or less general, but most marked on the face, scalp, palms and soles.

TREATMENT. — Hardy obtained good results by means of baths, followed by the application of iodide of mercury ointment.

ACNE CORNEA VEGETANS.

("DARIER'S DISEASE.")

This is a special form of acne cornea, which was described by Bazin under the name of general hypertrophy of the sebaceous system. More recently, Darier, believing that this affection was caused by parasites (psorosperms), described it under the name of *vegetating follicular psorospermosis*, a name which must now be abandoned, as it was based on error.

Acne cornea vegetans begins in the form of small hard crusts the size of a pin's head, situated at the orifices of the follicles. The crusts are at first the same colour as the neighbouring skin, but afterwards become gray, brown, or even black; they are very adherent, and imbedded in funnel-shaped depressions which correspond to the dilated orifices of the pilo-sebaceous follicles. The crusts are sometimes excoriated by scratching, which gives them a

hæmorrhagic tint. These elements, at first isolated, soon become confluent and form more or less extensive patches, which feel like a file. The regions of predilection are the groins, flanks, epigastric and hypochondriac regions, chest, face, scalp, back, and articular folds of the limbs.

At a more advanced stage, the horny crusts are replaced by red papillomatous vegetations, pressed against each other, the size of a large pea or larger. These vegetations have a central aperture, from which sebaceous matter, pure or mixed with pus, and of a fœtid odour, can be expressed. This transformation of the lesions is seen chiefly in the groins.

This affection causes no functional symptoms, but it is of unfavourable prognosis, owing to the chronic nature of the lesions and the failure of treatment.

DIAGNOSIS.—In its early stages this dermatosis may be mistaken for ordinary *acne cornea*, but in acne cornea vegetans the horny elements are broader and flatter. When papillomatous vegetations are present, the diagnosis is more easy. *Molluscum contagiosum* is seldom so generalised, and its elements are not formed by horny projections, but by small umbilicated tumours resembling pearls.

PATHOLOGICAL ANATOMY.—Acne cornea vegetans is a kind of keratinisation of the pilo-sebaceous follicles. If one of the horny plugs is soaked in liquor potassæ and then stained with Gram's solution, round nucleated masses with a thin membrane are found in the deeper parts of the plug. These bodies are sometimes free, sometimes enclosed in epithelial cells. They were originally mistaken for psorosperms, which were wrongly considered to be the cause of the disease. In the vegetating stage, the orifice of the follicle is dilated to form a cystic cavity, from the bottom of which arise the vegetations, formed by a connective tissue core extending from the dermis, covered by epithelial cells. In these cells and in those of the follicle are found a number of round bodies. The parasitic nature of these bodies was contested by Borck, Torok and myself, who regard them as the products of cell degeneration. This view is now generally accepted, and the hypothesis of psorospermosis has been definitely abandoned.[1]

The disease does not appear to be contagious, and attempts at inoculation have failed.

TREATMENT.—The horny lesions should be softened by means of soap spirit and frequent baths; after which salicylic acid or sulphur ointments may be applied. When there is exudation, absorbent powders such as calcined magnesia or dermatol are useful. Mercurial preparations do not give good results.

[1] Darier states that the bodies which he originally regarded as coccidia or psorosperms are really epidermic cells defectively keratinised (*Précis de Dermatologie*, 1909).—ED.

KERATOSIS PILARIS.

This affection (formerly called *lichen pilaris*) is characterised by the formation of dry, horny elevations, situated at the orifices of the hair follicles. There are two varieties: (1) white keratosis, in which the lesions are grayish white; and (2) red keratosis, in which the colour is red or purple. The former occurs chiefly on the trunk and limbs, the latter on the face.

Keratosis of the Trunk and Limbs.—This is formed by conical elevations, the size of a pin's head, situated at the orifice of the hair follicles; the cones are pointed or blunt, and give the sensation of a file. Sometimes the lesion consists of an accumulation of epidermic squames in the follicles. The colour is dull white, sometimes blackish brown, rarely red. Along with the cones there are white spots, resembling cicatrices, which are elements in process of resolution. The hair is sometimes completely destroyed, sometimes broken off at the orifice, and represented by a black spot; or it may be thinned and coiled up under the squames inside the follicle, becoming uncoiled when the epidermic cone is removed.

The lesions occur chiefly on the external and posterior surface of the arms and deltoid regions, the posterior surface of the forearms, the external and posterior surface of the thighs, on the calves and buttocks, around the knees and elbows, and on the sides of the abdomen. The unaffected regions are the thorax, axillæ, palms and soles, groins, and folds of flexion of the limbs.

Subjective symptoms are slight, and consist chiefly in pricking sensations rather than true itching.

Keratosis of the Face.—In this form the cones are very small and acuminate, chiefly appreciable to the touch, with a downy hair in the centre, and surrounded by a red areola. The inflammatory areolas of neighbouring follicles coalesce with each other and form red rough patches of varied extent, chiefly affecting certain regions. At the edges of each patch the cones are smaller, and the colour fades. The redness of the patches may be uniform, or formed by vascular dilatations; it disappears on pressure, but is increased by friction and by emotion. In the midst of the patches are seen white depressed spots without hairs; these are the remains of atrophied follicles.

This form of keratosis is chiefly observed on the forehead, where it forms two symmetrical patches affecting the whole, the external third, or the internal third of the eyebrows. It is always accompanied by a certain degree of alopecia; the hairs are scanty, deviated, and often replaced by white atrophic spots. The space between the eyebrows, the upper part of the chin and the ears are less often

affected. The other seats of predilection are the cheeks and the inferior maxillary and pre-auricular regions; the whole region of the beard may be affected.

There are sometimes changes in the nails, which become dry and brittle, and lesions on the scalp. According to Brocq, four kinds of scalp lesions may occur: (1) moniliform aplasia of the hairs, which are surrounded at their base by small red elevations; (2) small, irregular patches of alopecia, in which the dermis is dull white, as if cicatricial, sometimes with small red circumpilary papules near them; (3) more marked alopecia; (4) slight alopecia, with dry desquamation of the scalp, or with true seborrhœa.

Prognosis.—Keratosis pilaris is always chronic, and rebellious to treatment, but tends to disappear with advancing age, leaving, however, cicatricial spots and partial alopecia. On the face and arms it is disfiguring.

Pathological Anatomy.—The lesion commences in and around the sheath of the hair, where it attains its maximum development. Each hair follicle is surrounded by fibro-plastic cells and embryonic nuclei. Between the follicles the dermis is also infiltrated with embryonic cells. At an advanced stage the hair follicles are atrophied, and, according to Lemoine, the sebaceous glands have often disappeared in the midst of the connective tissue hyperplasia. The horny mass situated in the follicular infundibulum is composed of epidermic stratification around the hair.

Etiology.—Keratosis pilaris is chiefly observed in lymphatic subjects, especially women. It appears to be hereditary, and appears between the second and twentieth years, increasing at puberty. Some authors regard it as a variety of ichthyosis (anserine ichthyosis). It is true that it is often complicated by xerodermia and sometimes by ichthyosis, but the lesions are always distinct from one another.

Diagnosis.—Keratosis pilaris must not be mistaken for *chronic lichen simplex*, nor for *pityriasis rubra pilaris*, which has a certain resemblance to it, but differs from it by its large red squamous patches, and by the abundant desquamation on the palms and scalp. Red keratosis of the face is easily distinguished from *acne rosacea* and *lupus erythematosus*. Keratosis of the scalp and beard must be distinguished from *alopecia areata*.

Malcolm Morris has described, under the name of *eczema follicu-lorum*, a dermatosis characterised by groups of small elements analogous to those of keratosis pilaris, but which develops at adult age and in regions different from those of keratosis.

Treatment.—In lymphatic subjects, cod-liver oil, arsenic and iodine preparations should be prescribed.

Locally, the parts should be washed with salicylic soap, after

which salicylic ointment or plaster is applied. In keratosis of the face, which is more rebellious, the following ointment is useful:—

Salicylic acid	1 part
Tartaric acid	1 „
Soft soap	40 parts

In keratosis of the scalp, sulphur ointment should be applied once or twice a week. Lastly, the more prominent lesions may be destroyed with the galvano-cautery.

ADENOMA SEBACEUM.

This lesion, studied by Balzer and Menetrier, occurs on the face, especially in the naso-labial furrows, at the root of the nose,

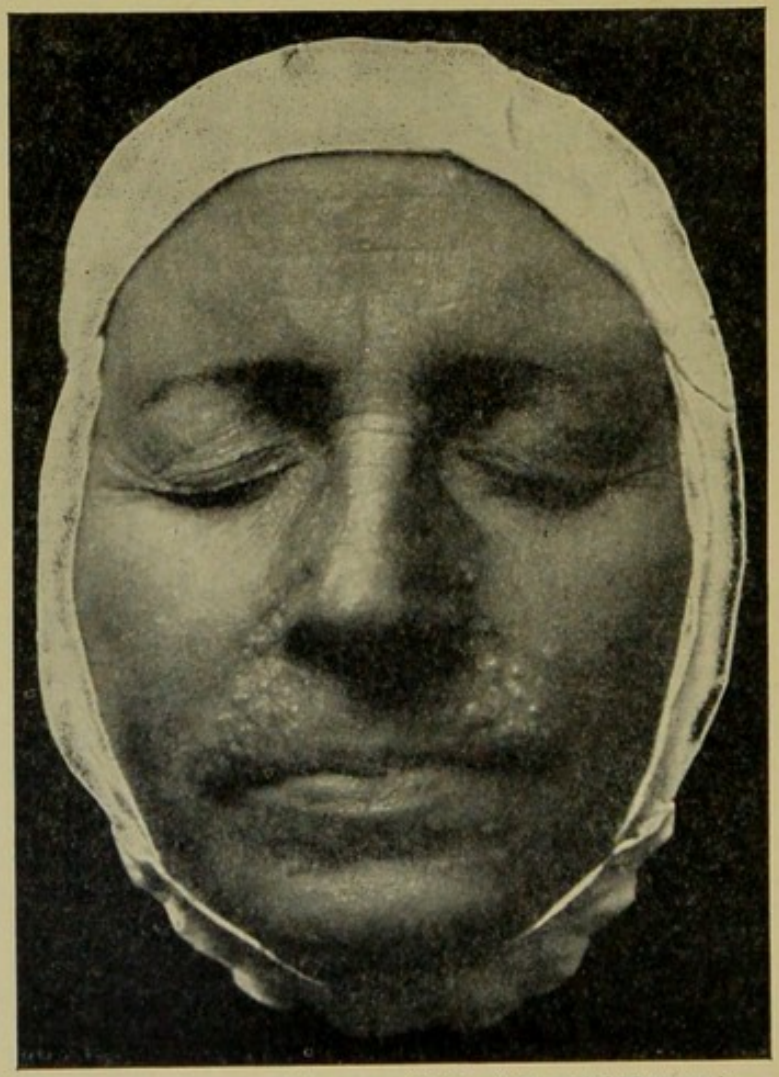


Fig. 46.—Adenoma sebaceum. (St Louis Hospital Museum.)

on the forehead and around the mouth, rarely on the chin, and still more rarely on the rest of the body.

Sebaceous adenomas are small tumours, varying in size from that of a pin's head to that of a pea, and arranged symmetrically. They are confluent (in the naso-labial furrows) or disseminated (on the cheeks, forehead, chin and external ear), painless, sessile, spherical or slightly acuminate, of hard consistence and grayish colour, resembling grains of boiled sago. When punctured, a little colourless fluid escapes.

They are sometimes studded with white points (cystic formations). They appear never to disappear spontaneously.

Histologically, these little tumours are benign sebaceous epithelial adenomas, formed by hypertrophy of the sebaceous glands, with cystic degeneration in places.

ETIOLOGY.—This is obscure. The affection appears to be congenital, and commences in infancy. It is sometimes met with in several members of the same family, and occurs more often in females than in males.

DIAGNOSIS.—It must be distinguished from *warty nævi* of the face, which have the same size and the same distribution (naso-labial furrows, cheeks, chin and forehead); but sebaceous adenomas are grayish white, while warty nævi have a red colour which disappears under pressure.

TREATMENT.—The tumours should be destroyed with a fine thermo-cautery or galvano-cautery.

DISEASES OF THE SUDORIPAROUS GLANDS.

DISORDERS OF SECRETION.

THE secretion of sweat may be altered in quantity (*hyperidrosis* or *anidrosis*), in colour (*chromidrosis*), in odour (*bromidrosis*), or in chemical composition (*uridrosis*).

Hyperidrosis.—Apart from the general sweating observed in fevers, pulmonary tuberculosis, and miliaria, we are only concerned with essential hyperidrosis, general or local, especially observed in obese, nervous or arthritic subjects, which appears under the influence of emotion, or elevation of temperature. The secretion of sweat in the different dermatoses is dealt with in their individual descriptions.

General hyperidrosis is sometimes preceded by pricking sensations. The sweating is most profuse on the forehead, scalp, axillæ, chest, groins, genital folds, palms and soles. It is sometimes complicated by *sudamina* and *miliaria*.

Local hyperidrosis, or *ephidrosis*, occurs on the forehead, scalp, axillæ, palms and soles. *Palmar ephidrosis*, which is more common in the summer, may be produced by slight emotion In *plantar ephidrosis*, the epidermis may be macerated and become detached, exposing the reddened, sensitive dermis, and rendering walking almost impossible; this form is often associated with bromidrosis.

In cases of nervous disorder ephidrosis may be limited to one half of the body.

Bromidrosis.—This is probably due to chemical changes in the sweat, or to decomposition of the sweat remaining on the skin, setting free fatty acids which cause the peculiar odour.

Chromidrosis.—This is a modification in the secretion of the sweat gland which gives a blue, yellow, green, red or black colour to the sweat. Chromidrosis occurs on the lower eyelids, which assume a slaty blue colour; on the chest, abdomen, scrotum, arms and feet. I have observed a case of intermittent red chromidrosis affecting the thenar eminence and antero-external surface of the wrist, and another case of red chromidrosis affecting the hands and forearms. The colouring matter can be removed by brisk rubbing.

Chromidrosis must be distinguished from a red coloration of the hairs, which is observed in the axillæ in some persons. This is a parasitic affection called *lepothrix*, and it is characterised by small red concretions fixed on the hair and enclosing bacilli, which appear to be the cause of the affection.

Hæmatidrosis is rather a capillary hæmorrhage than a sudoral secretion, and occurs in hysterical subjects. The liquid does not contain blood corpuscles, only their colouring matter.

Uridrosis.—This is the urinous sweat sometimes observed in cases of uræmia. The urea forms a powdery deposit on the forehead and other parts of the face.

Anidrosis.—This occurs in chronic eczema and psoriasis, in xerodermia pigmentosa, exfoliative dermatitis, chronic pityriasic rubra, ichthyosis, keratosis pilaris, and in the senile skin; also in diabetes, cancer, and tuberculosis with chronic diarrhœa. In all these diseases the skin is dry and sometimes pityriasic.

TREATMENT.—In *general hyperidrosis*, white agaric, phosphate of lime or sulphate of atropin ($\frac{1}{120}$ grain daily) may be prescribed, together with local applications of eau-de-Cologne or spirit of lavender.

In *plantar bromidrosis* and *ephidrosis*, the feet should be washed night and morning in a decoction of walnut leaves containing alum or borax; then alcoholic lotions, followed by a dusting powder of salicylic acid (3 parts), starch (10 parts) and talc (90 parts). This powder is similar to that employed in the German army.

General treatment is also required for obesity, arthritism, nervous conditions, etc.

SUDAMINA. MILIARIA.

This is a vesicular eruption which sometimes occurs after excessive sweating. It must be distinguished from *miliary fever* or sweating fever.

ETIOLOGY.—Sudoral eruptions are common in acute and febrile diseases (enteric fever, typhus, scarlatina, acute rheumatism, puerperal fever, tuberculosis, etc.). They may also occur in healthy subjects after profuse perspiration.

SYMPTOMATOLOGY.—There are two forms: *crystalline miliaria* and *red miliaria*.

In **crystalline miliaria** the skin is covered with miliary transparent vesicles (*sudamina*) the size of a pin's head, containing clear liquid. The vesicles sometimes coalesce to form small bullæ; they are chiefly situated on the chest, abdomen, flanks, neck, and arms, and are rare on the face. Anatomically, they are small cysts formed in

the horny layer of the epidermis, and due to dilatation of the sweat ducts, the orifices of which are plugged with a layer of epidermis.

In **red miliaria**, the vesicles, produced in the same way, are surrounded by a slight congestive areola, which gives the skin an inflammatory aspect. If the lesion persists for some time, the vesicles lose their transparency and become turbid (*white miliaria*). At a more advanced stage they are filled with pus cells (*yellow miliaria*), and the inflammation of the dermis is more marked.

Sudoral eruptions are sometimes accompanied by pricking and itching, especially in red miliaria. The vesicles are often excoriated by scratching. Their duration varies from two to five days, but red miliaria sometimes lasts longer, occurring in successive crops. The vesicles rupture, and are followed by slight desquamation.

DIAGNOSIS.—Miliaria must be distinguished from *eczema;* this is never so generalised, and is accompanied by squames and crusts, which never occur in sudoral eruptions. The liquid in eczematous vesicles is alkaline, that in sudoral vesicles is acid. Diagnosis from certain forms of *acute lichen* is more difficult, but the elements of miliaria are smaller, more crowded, and more ephemeral; when punctured, liquid escapes, while the lesions of lichen are always hard and solid.

TREATMENT.—In *sudamina* the parts should be dusted with starch or lycopodium powder, or, better, with talc, subnitrate of bismuth, or oxide of zinc. In *red miliaria,* bran or starch baths should be prescribed in addition.

DYSIDROSIS, OR CHEIRO-POMPHOLYX.

This dermatosis is characterised by an eruption of vesicles containing clear liquid, affecting the extremities.

SYMPTOMATOLOGY.—The eruption is nearly always preceded by more or less burning or itching, after which appear small transparent vesicles, resembling boiled sago grains, deeply situated in the epidermis. At first isolated, they become confluent, but never rupture. After a time they become yellow. When punctured, liquid escapes—acid, neutral or slightly alkaline, according to the date of the lesion. When left alone, the liquid is gradually absorbed, and the epidermis exfoliates, leaving a smooth, red, painful surface.

Dysidrosis occurs most often on the hands, in the interdigital spaces, and on the sides of the fingers. Sometimes it raises the whole epidermis of the palm, and may spread to the dorsal surface and to the arm. In rare cases it may affect the whole body, when it resembles an eruption of miliaria, and may simulate eczema. The feet are sometimes affected like the hands, and occasionally the face. In some cases the vesicles suppurate, owing to secondary infection.

The eruption lasts for ten days or a fortnight; in severe cases, for several weeks. Recurrence is common.

PATHOGENY.—Tilbury Fox attributed dysidrosis to rapid secretion and retention of sweat in the ducts of the sweat glands. This pathogeny is probably correct, for microscopic examination of the lesions shows that the vesicle is formed by dilatation of the sweat duct

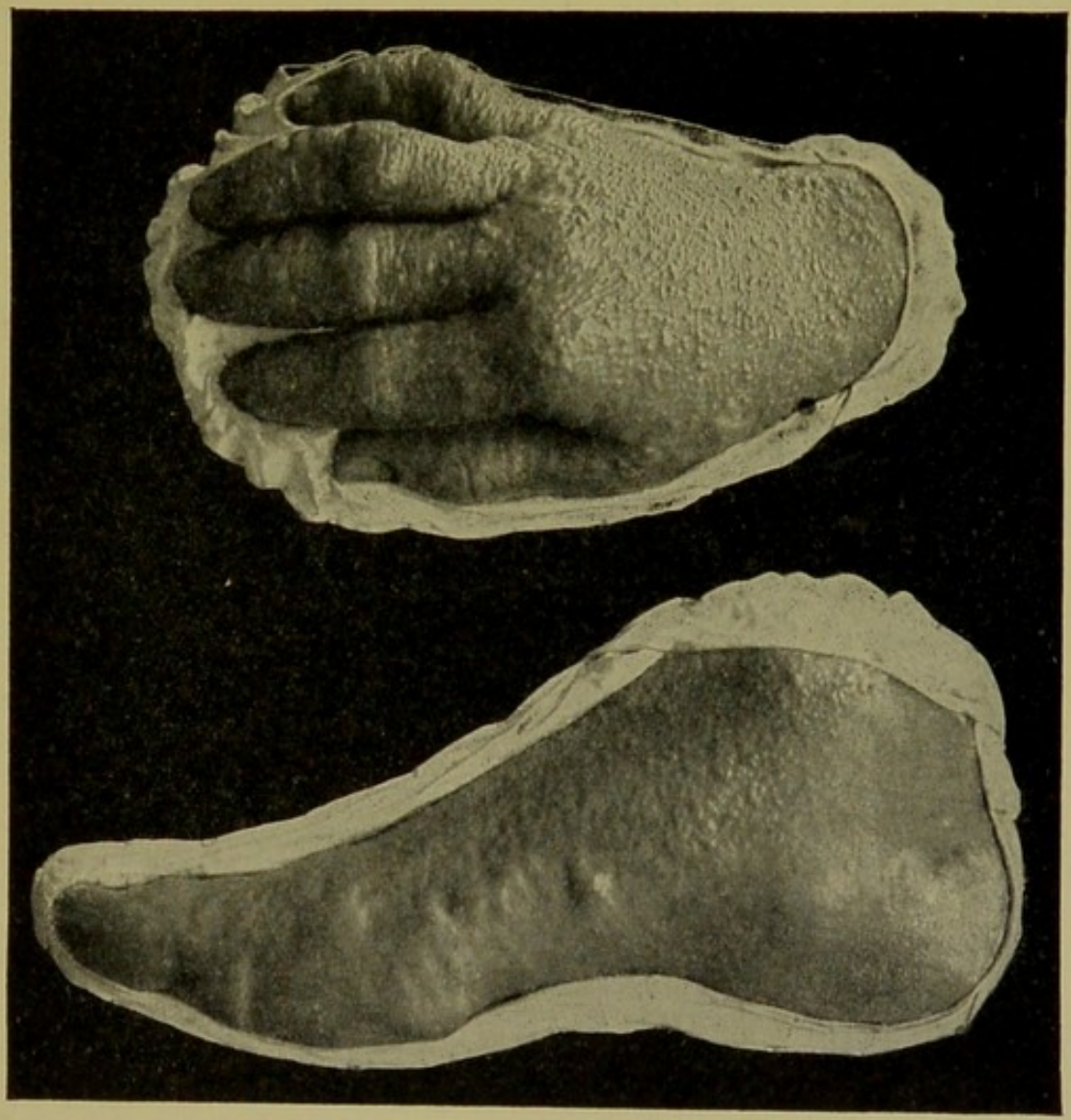


FIG. 47.—Dysidrosis. (St Louis Hospital Museum.)

where it passes through the epidermis, this dilatation causing elevation of the horny layer.

ETIOLOGY.—Dysidrosis occurs usually in the spring and summer, in subjects who perspire profusely; sometimes in dyspeptics, or under the influence of high temperature. It appears also to be often of nervous origin.

DIAGNOSIS.—Dysidrosis differs from *eczema* in the size of its

vesicles, and in the absence of serous exudation and crusts; however, it may be followed by true eczema. *Sudamina* are smaller, more superficial and ephemeral. *Hidrocystoma* (A. Robinson), which occurs chiefly on the face, differs only in the deeper situation of the dilatation, which is situated in the dermis instead of in the epidermis.

TREATMENT.—Itching may be relieved by bran or starch baths. According to the condition of the skin, it should be dusted with salicylic and talc powder (2 per 100), or covered with zinc ointment. When there is acute inflammation, the affected parts should be dressed with wool soaked in linimentum calcis. When the vesicles suppurate, moist aseptic dressings should be applied.

DISEASES OF THE HAIR AND NAILS.

ALOPECIA AREATA.

THIS is a form of alopecia affecting the scalp and beard, characterised by smooth, circular patches, completely destitute of hair.

SYMPTOMATOLOGY.—Alopecia areata begins with slight itching at the affected parts, where the hairs become dry and dull, and are easily pulled out. Very rapidly, generally in a few days, the hairs fall out, leaving one or more circular or oval patches completely bald; sometimes a few thin atrophied hairs remain, which are easily pulled out. On these patches the skin is smooth, often shining like ivory, and slightly depressed in the centre. In some cases, especially at first, it is slightly tumefied. Sometimes the patches are milky white (achromic alopecia). The patches vary in size from half an inch to four inches in diameter. There may be only one patch, or several patches, which may coalesce to cover an extensive surface, with polycyclic borders. In severe cases the whole scalp may be denuded (*alopecia decalvans*).

The disease may affect the beard, eyebrows and eyelashes, chest, axillæ and pubis, and in some cases all the hair on the body falls off. Apart from this general alopecia, the beard alone may be affected.

On examination of the hairs from a patch of alopecia, they are found to be dry and slightly atrophied, but not friable like hairs affected with ringworm. Under the microscope, the hair is thin, atrophied and decolorised, especially towards the root, which is tapering, or irregular in the form of a tap-root. In the centre of the hair there are sometimes brown granules arranged along the axis of the hair, sometimes air-bubbles occupying the medulla of the hair, which has completely disappeared. The free end of the hair is fibrillated. No parasites of any kind are found in the hair or on its surface. Sometimes the hairs are broken like ringworm hairs, when the alopecic patch somewhat resembles a patch of ringworm.

The nails may also be affected in alopecia areata, becoming cracked, striated, and liable to exfoliate (Crocker, Arnozan, Gaucher).

Evolution.—This is sometimes very rapid, the patches quickly spreading and uniting by confluence; in other cases, a single patch or several patches may remain stationary; sometimes successive patches appear.

The duration of the disease may be several months. After a time, downy hair appears on the patches, and the skin resumes its

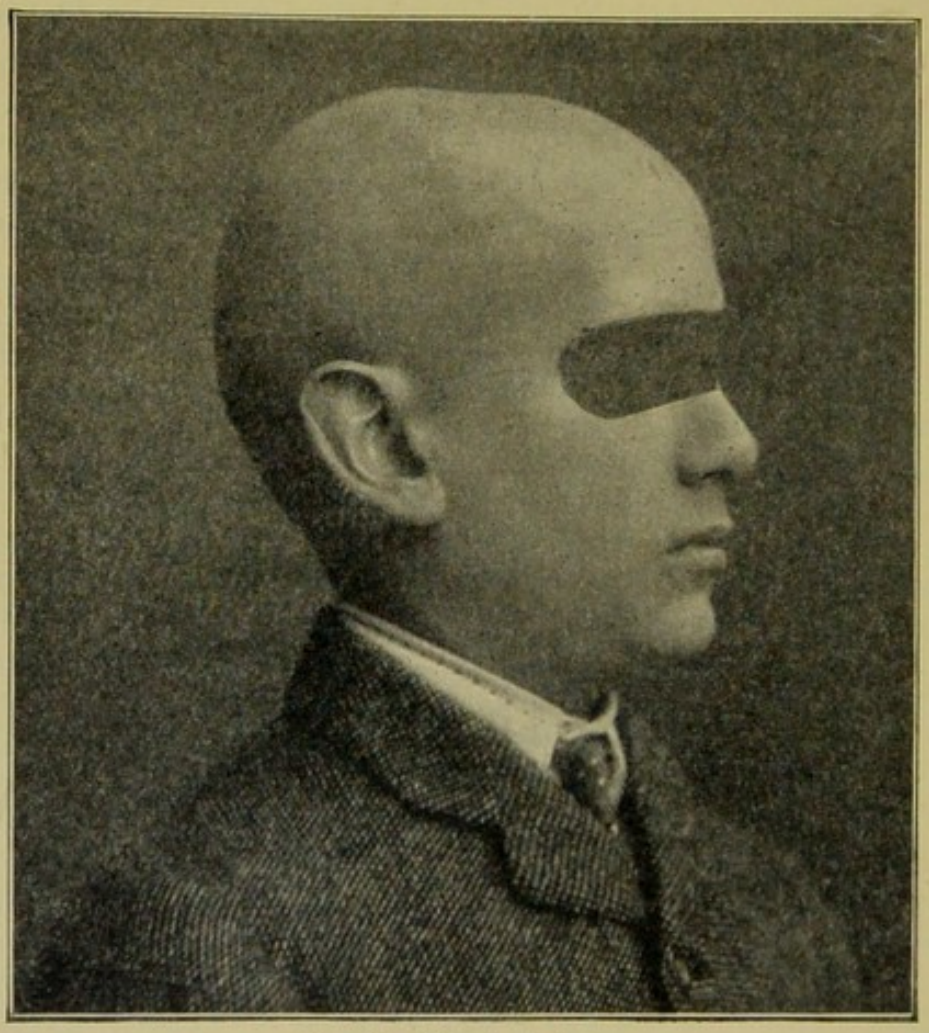


Fig. 48.—Alopecia decalvans.

normal colour; then new hair grows, at first almost colourless, but gradually assuming the normal colour, finally becoming strong and healthy. Sometimes the hair remains permanently white, and is more scanty than before.

In some cases, both treated and untreated, the patches of alopecia remain permanent. Even when cured, recurrence often takes place after a year or two years. I have seen cases recur after ten years or more.

PATHOLOGICAL ANATOMY.—The epidermis, dermis, and cutaneous glands appear to be intact. There is only achromia of the Malpighian layer, and slight infiltration of the dermis. The hair follicles, on the contrary, are atrophied, and contain no trace of a hair. Sometimes, however, they may contain a rudimentary hair which does not reach the surface (Balzer).

In 1874 Melassez discovered a parasite formed by groups of round or oval spores situated on the epidermic scales, but not in the hairs. It has been proved that these spores play no part in alopecia areata. Vaillard and Vincent described a micrococcus situated between the bulb of the hair and the wall of the hair follicle. Sabouraud described another microbe present in alopecia areata, but neither of these have been proved to be the cause of this disease.

ETIOLOGY.—Epidemics of alopecia areata have been observed in schools and barracks, and there are data which prove that the disease may be spread by means of toilet articles, head-dresses, and pillows. On the other hand, there are facts which seem to contradict the contagiousness of the disease; for example, a husband or wife may be affected without transmitting the disease to the other. Again, daily observation shows the influence of a nervous temperament in the development of alopecia areata, which sometimes occurs after strong emotions or excessive brain work without any exposure to contagion. Hebra and the German school regard alopecia areata as a trophoneurosis.

Tilbury Fox, to reconcile these divergent opinions, maintained that there were two forms of the disease, the one contagious and parasitic, the other of nervous origin; but it must be borne in mind that it is at present impossible to distinguish parasitic from nervous alopecia. The presence of a single patch instead of several, and the symmetry of the lesion have been mentioned as characters pointing to a nervous origin; but this is not proved.

In reality, apart from trophoneurotic alopecias due to a definite nerve lesion or nervous disorder, there is only one form of alopecia areata, which is only contagious on account of permanent or temporary loss of equilibrium of the nervous system, and is only contagious in its microbial stage. The probable but unknown infective agent of alopecia areata acts chiefly by its toxins, and the influence of these may last long after the disease has ceased to be contagious. It is because of the inconstancy, rarity, and peculiarity of contagion that this has been denied by certain authors, for want of sufficient experience. Formerly I denied it myself at a period when I was too young to have observed it, but undoubted facts convinced me later of the possibility of contagion.

Alopecia areata occurs at all ages, but chiefly in children and

adults. It may affect cats and horses, and be transmitted from them to man.

DIAGNOSIS. — It is impossible to mistake alopecia areata for traumatic *cicatrices*, or for the cicatricial patches left after *favus*. It differs from *ringworm* by the resistance of the hairs, which can be pulled out without breaking; but in cases of pseudo-tinea tonsurans this sign is absent, and the diagnosis must be made by microscopical examination, which shows the absence of spores in alopecia areata. It must also be distinguished from alopecia secondary to acute diseases, such as enteric fever; in the latter the loss of hair is diffuse, and not in patches. The alopecia of *secondary syphilis* is more irregular as a rule, but sometimes occurs in patches resembling those of alopecia areata, when the diagnosis must depend on other signs of syphilis. Alopecia occurs in *lupus erythematosus* of the scalp, but the patch of lupus is recognised by its red border and cicatricial centre. Alopecia areata must be distinguished from *folliculitis decalvans;* this causes irregular patches of alopecia, in the centre of each of which can be seen a suppurating follicle, or a red cicatricial spot, the remains of the healed follicle.

TREATMENT.—On the supposition that alopecia areata is a parasitic disease, the hairs round the patches should be epilated. Previous to epilation, the hair should be cut short and the scalp treated with a solution of perchloride of mercury (1 in 1000 to 1 in 500). After this, antiparasitic and irritant preparations should be applied, to stimulate the growth of hair. Besnier recommends glacial acetic acid, applied every three or four days. If this is too irritating, the acid may be diluted with alcohol (1 in 5). Pure acetic acid should never be used when the whole scalp is affected. In the intervals between the applications of acetic acid, the affected parts should be painted every day with one of the following mixtures:—

(1)	Acetic acid	1 part
	Chloral	3 parts
	Ether	30 „
(2)	Ammonia	5 to 10 „
	Essence of turpentine	10 to 20 „
	Alcohol	100 „
(3)	Tincture of cantharides Tincture of rosemary	equal „

Hillairet and Vidal recommend pure tincture of cantharides; a second application being made after the inflammation set up by the first has disappeared. But this drug should not be left in the hands of patients. Camphor blisters may also be used. Moty has proposed injections of five or six drops of a solution of perchloride of

mercury (1 in 100), made at several places in each patch. In some cases, especially in alopecia decalvans, high frequency currents give good results, and stimulate regrowth of hair.

The general condition of the patient should be improved by hydrotherapy, sulphur baths and douches, and tonics.

LEPOTHRIX.

This affection is characterised by the presence of small concretions fixed to the hairs, chiefly those of the axillæ and genital regions. Sometimes the hairs are completely surrounded by the concretions, sometimes the latter form small round masses fixed at intervals, but more numerous at the free end of the hair. The concretions are brownish red, sometimes black, and are often associated with red sweat in the axilla; they are very adherent to the hairs, which are rather fragile. The hair follicles are intact. These concretions consist of a hard granular substance enclosing bacilli, which may penetrate the cortical layer of the hairs. These bacilli have not been cultivated.

TREATMENT.—The affected parts should be shaved, after which perchloride of mercury lotion (1 in 1000) should be applied every day for a week.

TRICHORRHEXIS NODOSA.

This is a peculiar change in the hairs, which become swollen at several places and then split into two parts, the extremities of which are brush-like. Before splitting, the hair presents at intervals from one to five small round swellings, which occupy its whole circumference. On the slightest traction the hair breaks off at one of these swellings, but its root remains firm. This affection affects the beard principally, but is also met with on the scalp, and on the pubis in women. Some authors regard it as a trophic disorder, others as parasitic.

TREATMENT.—Besnier recommends epilation of all the diseased hairs, followed by applications of tincture of cantharides. Jadassohn has had good results with pyrogallic acid ointment (2 per 100).

TRICHOPTILOSIS.

In this condition the hairs are dry, and split at their ends or in part of their continuity. It is probably a trophic disorder. It affects the hairs of the scalp, especially in women, and sometimes

those of the beard. It is sometimes observed in ringworm, pityriasis capitis, and eczema of the scalp, after prolonged fevers, or during cachectic diseases.

TREATMENT.—The hair should be cut short. The general condition of the patient and any local concomitant affection also require attention.

MONILIFORM APLASIA.

In this affection the hairs present constrictions in their whole extent, which produce a moniliform appearance. The hairs are short, dry, thin, downy and brittle. The constricted parts are colourless, because the medullary substance is atrophied; the normal parts, which are apparently enlarged, contain a large quantity of pigment. Moniliform aplasia may affect the hairs of the scalp, axillæ, pubis and chest. In a case of Hallopeau's, the hairs presented a circumpilary papule at their base, resembling keratosis pilaris; cicatrices with alopecia were present here and there, due to atrophy of the hair follicles. According to Hallopeau and Brocq, keratosis pilaris is closely allied to moniliform aplasia, and according to Hallopeau the latter is hereditary and sometimes congenital.

Moniliform aplasia must not be mistaken for trichorrhexis nodosa, nor for annulated canities.

TREATMENT.—If the affection is associated with keratosis pilaris, the latter must be treated. The treatment of moniliform aplasia consists chiefly in keeping the hair cut short and stimulating the scalp with glycerole of oil of cade.

CANITIES.

This is decolorisation of the hairs, which become white. It may be congenital or acquired, local or general.

General *congenital canities* is seen in albinos; local congenital canities, which is more common, forms disseminated tufts in the hair or beard.

Acquired canities occurs usually after a certain age. Heredity plays a part in premature canities, but it may develop rapidly after violent emotion, or after severe diseases, especially neuralgia of the head. These conditions show the influence of nervous disorders in the production of canities. In alopecia areata the new hairs are often white at first.

In some cases (*annulated canities*) the decolorisation is partial, the hairs presenting alternate white and coloured parts, an appearance which may be mistaken for moniliform aplasia.

TREATMENT.—This is only palliative. The whiteness may be concealed by means of hair dyes (nitrate of silver, carbon, etc.), but preparations containing lead should be avoided, on account of the danger of lead poisoning. In circumscribed canities secondary to alopecia areata, repeated epilation may be followed by a growth of normally coloured hair.

HYPERTRICHOSIS.

This is an abnormal development of the hair, which may be general or local. In the *general* form, which is rare, the whole surface of the body is hairy, except the palms and soles, the ends of

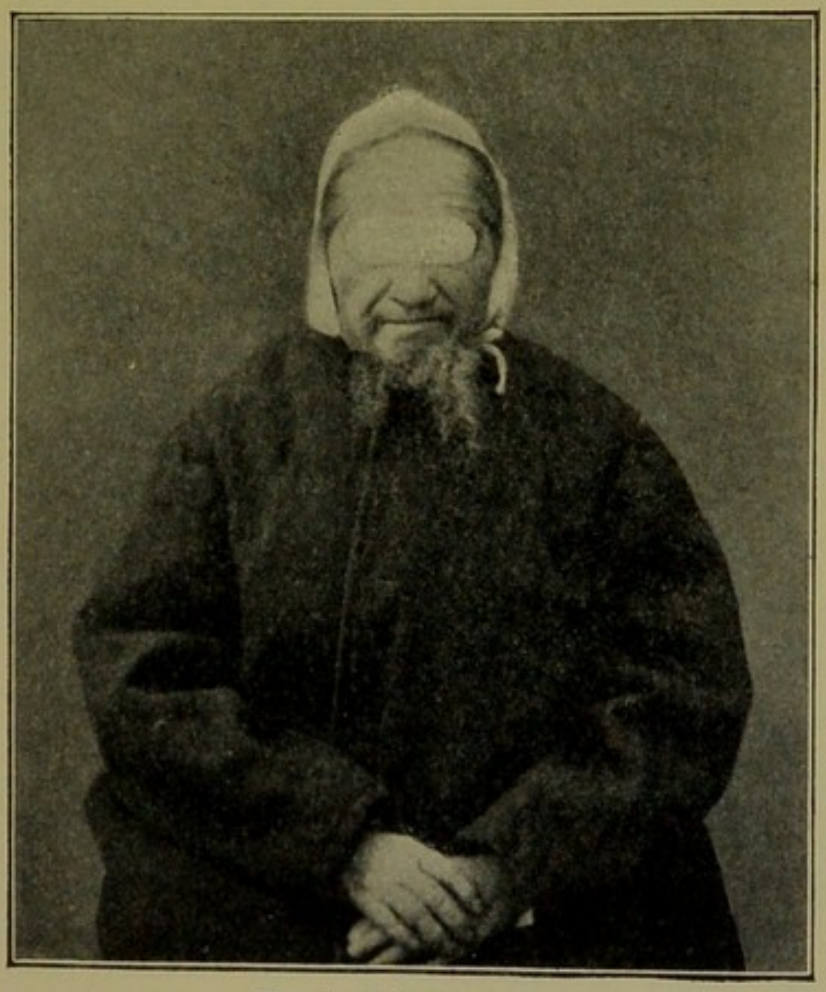


FIG. 49.—Hypertrichosis.

the fingers and toes, the internal surface of the labia majora, the prepuce and glans penis.

Local hypertrichosis may be congenital or acquired. Congenital local hypertrichosis affects the lower part of the spine, and differs from *hairy nævus* by the absence of pigmentation and changes in the skin.

Acquired local hypertrichosis is sometimes spontaneous, like the beard which develops in some women; sometimes it is caused by repeated irritation, such as violent scratching caused by very pruriginous affections, or by the application of numerous blisters. It may be observed in men, on any part of the body; on the nose, in the form of tufts of hair, between the eyebrows, on the ears, or in the nostrils. In women, it may occur in the same regions as in men; it often occurs on the cheeks, chin, upper lip, chest, legs and thighs, where it is continuous with the hairs of the vulva.

Hypertrichosis of the chin and upper lip develops in some women, especially brunettes, at the menopause. But even before this physiological period, some women grow a beard like men; this anomaly, which is sometimes exploited as a freak, is a sign of degeneration.

TREATMENT.—Epilation is only a palliative measure, which requires constant repetition. The only means of suppressing hypertrichosis is destruction of the hairs by *electrolysis*. This is performed as follows: a fine needle, connected with the negative pole of the battery, is introduced to the bottom of the hair follicle; when the hair bulb is destroyed the current is shut off. The needle should be of platinum-iridium, and should consist of a metallic cylinder half an inch long, a thin stem about an inch in length, and a terminal part about half an inch long, as fine as possible, and bent at an obtuse angle about a quarter of an inch from the point. The point of the needle is inserted gently along the hair as far as the bulb; if there is any resistance, it shows that the point of the needle is not in the follicle. When the needle is properly in place, it is connected with the negative pole, and a current of 2 to 5 milliamperes is turned on. The positive pole is grasped by the patient, or placed on his arm or shoulder. When the hair is destroyed, the positive pole is removed, and the needle withdrawn.

According to Brocq, as soon as the current passes, an erythematous ring is produced round the needle, and almost immediately afterwards a white froth; finally, when the current has been passed long enough, a small brown ring appears, on which a transparent vesicle develops after the needle is withdrawn. I do not advise waiting for the appearance of this phenomenon before withdrawing the needle, for fear of producing a scar. The approximate length of time during which the current should be passed varies from three to ten or twenty-five seconds. Slight traction should be exercised on the hair with epilating forceps during the operation. When the hair yields easily it is removed, and the needle withdrawn. Another hair is then treated, but never a neighbouring one, to avoid the production of visible scars. When there are many hairs to be

destroyed, instead of removing them one by one, they may be all removed together at the end of the sitting. When the operation is skilfully performed there should be no appreciable cicatrix; but, as a matter of fact, subsequent cicatrices are not uncommon. Moreover, it is often necessary to repeat the operation, either because some hairs grow again which have not been completely destroyed, or because a new crop of hairs develops after the first have disappeared.

In fact, the treatment of hypertrichosis by electrolysis is not so satisfactory as is generally supposed. If the X-rays were free from danger they might be employed instead of electrolysis, but the dosage of the rays is uncertain, and when they are sufficiently intense to destroy the hair follicles there is a risk of producing cicatrices with all their consequences, including possible epithelioma. Perhaps better results may be obtained with radium.

ALOPECIA.

Alopecia is general or partial loss of hair, which may be produced by various causes. It is divided into three main groups: (1) that due to a physiological condition or a general disease; (2) that which depends on disease of the scalp; (3) that due to mechanical causes.

First Group.—This includes congenital alopecia, senile alopecia, and premature alopecia. *Congenital alopecia* is rare, and perhaps hereditary; it may be local or general. It may disappear with age, and the hairs eventually grow. It is sometimes associated with moniliform aplasia and keratosis pilaris.

Fig. 50.—Congenital alopecia. (Audry.)

Senile alopecia depends on senile cutaneous atrophy; it nearly always commences at the vertex, and extends to the neighbouring parts.

Premature alopecia begins between the seventeenth and twentieth years. Some authors include it with *pityriasic alopecia*, but in many cases no concomitant pityriasis is observed. The hairs which fall are replaced by other finer ones, which fall in their turn. This form of alopecia is symmetrical, and commences on the vertex or front of the scalp, or on the temples, extending over most

of the scalp, which becomes smooth and shiny. It is due to progressive atrophy of the dermis, involving the hair follicles. It is often hereditary, and is common in arthritic subjects and in persons who perform much intellectual work.

Alopecia due to disorder of the general health may occur in the course of, but more often during convalescence from, eruptive fevers, such as enteric fever; after scarlatiniform erythema, after childbirth, and in chlorosis, diabetes, tuberculosis, cancer, syphilis, etc. In all these morbid conditions the loss of hair varies in degree, but is nearly always diffuse.

Second Group.—This includes two subdivisions: (1) alopecia secondary to local dermatoses of the scalp, in which the loss of hair is only a secondary and inconstant symptom; pityriasis capitis, seborrhœa, eczema, seborrhœic eczema, psoriasis, impetigo, etc.; (2) that due to dermatoses, in which the alopecia is the essential symptom; ringworm, favus, alopecia areata, the various forms of folliculitis, folliculitis decalvans, sycosis, keratosis pilaris of the scalp, etc. These forms of alopecia are studied with the diseases which cause them.

Third Group.—This includes: (1) *occipital alopecia*, which occurs in restless infants, and is due to repeated rubbing of the back of the head against the pillow; (2) *trichomania* or *trichotillomania*, an affection in which patients constantly scratch the head, owing to pruritus of the scalp, and finally pull out the hairs.

TREATMENT.—The hair should be kept cut short, and stimulating lotions applied to the scalp (see Alopecia areata).

DISEASES OF THE NAILS.

We shall only deal here with hypertrophy, atrophy, and anomalies of coloration of the nails. The parasitic onychoses are considered with favus, ringworm, etc. *Hypertrophy* of the nails may be congenital or acquired, uniform or irregular. The nail becomes brown and thick, especially in the middle; it may become raised in a conical form, or curved on itself like a claw (*onychogryphosis*). The treatment consists in cutting and filing the horny formations, and applying salicylic acid ointment (10 per 100).

Atrophy of the nails may also be congenital or acquired. The nail is smaller and thinner than usual, and sometimes disappears. This change is often secondary to a concomitant cutaneous affection, but sometimes it seems to be primary.

Hyperchromia of the nails occurs in association with hyperchromia of the skin.

Achromia of the nails may be total or partial; in the latter case it is characterised by dull white spots.

A great number of dermatoses are accompanied by changes in the nails, which have been mentioned in the descriptions of these dermatoses. Enteric fever and other eruptive fevers, certain nervous diseases (neuritis, tabes), and chronic diseases cause nutritive changes in the nails.

Special mention must be made of onychoses which appear to be primary, and not connected with any parasitic or cutaneous disease. I have observed two cases which appeared to be due to congenital syphilis.

DYSCHROMATOUS DERMATOSES.

ACHROMIA.

THIS name is applied to more or less complete disappearance of the normal pigment of the skin.

Achromia may be congenital or acquired; when congenital it constitutes *albinism*, which may be general or partial. Acquired achromia includes two types: *true achromia* or *leucodermia*, and *vitiligo*. The former is characterised by simple decolorisation of the skin without peripheral hyperchromia; vitiligo, on the other hand, is achromic in the centre and hyperchromic at the borders.

True achromia is always secondary. It follows papular and ulcerative syphilides, and may occur in the course of leprosy, in atrophy of the skin, and sclerodermia; it is one of the characteristic signs of alopecia areata.

VITILIGO.

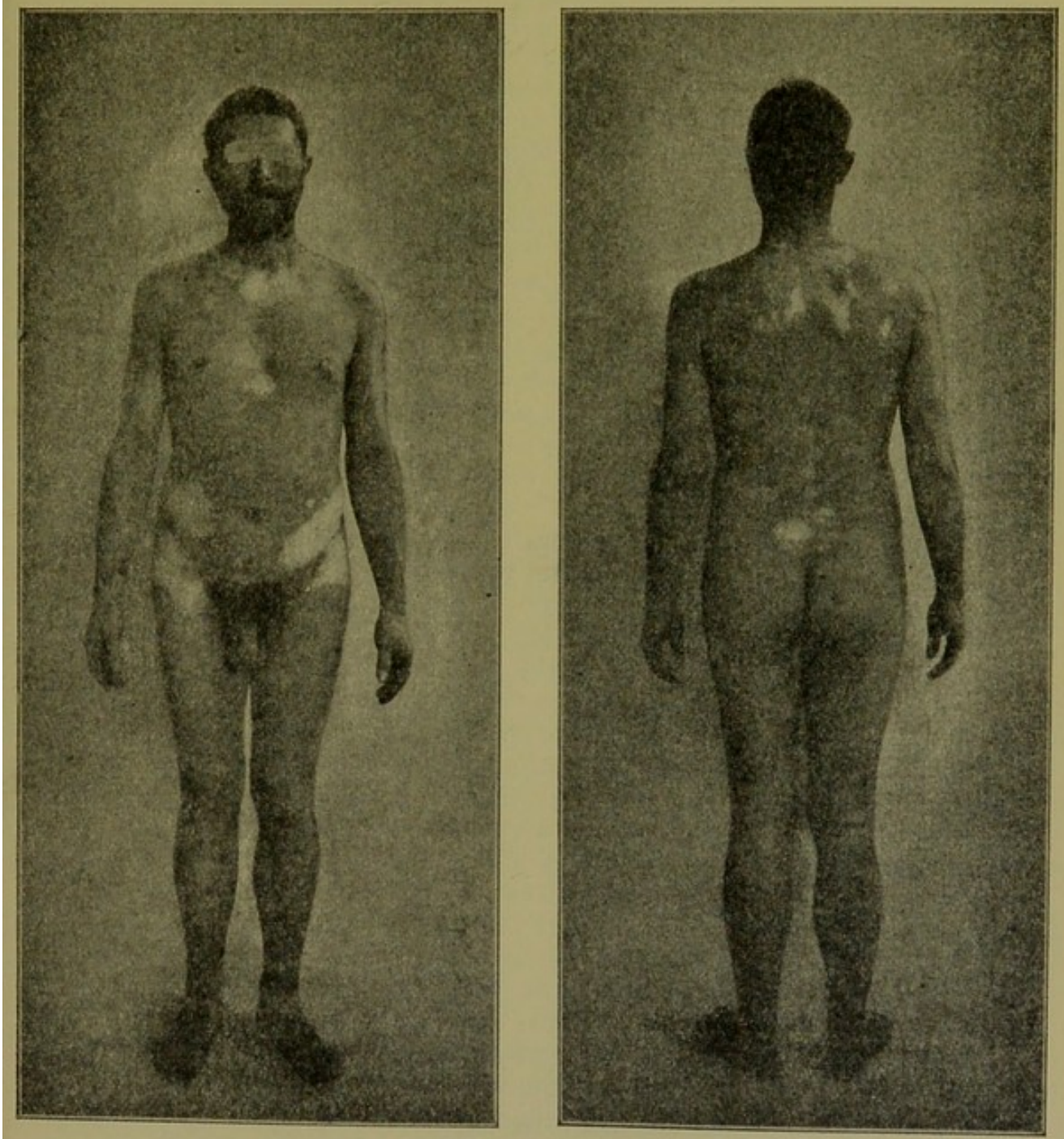
Vitiligo (from *vitilus* "a calf," signifying a skin speckled like that of some calves), is a pigmentary dystrophy, an irregular distribution of the cutaneous pigment. It is characterised by the development, in one or more parts of the body, of white patches surrounded by a zone of hyperpigmentation which merges gradually into the healthy skin. There is therefore both achromia and hyperchromia, so that the term leucodermia, used by some authors, is incorrect. The onset of vitiligo is insidious and may escape notice, especially if it commences on covered parts of the body.

Vitiligo may occur on various parts of the body; generally symmetrically, on the limbs, especially the hands, neck, shoulders, pelvic region, and genital organs. The last situation is important, for, as a rule, there are one or more patches of vitiligo on the pubis, labia, scrotum, or penis, when it is present on some other part of the body.

The patches are of various sizes; they may remain small during their whole evolution, but often spread gradually during the course

of years. The outlines may be irregular or circular; the skin is smooth and supple; the hairs on the patches become white.

In some subjects vitiligo may change its position (vitiligo



Figs. 51 and 52.—Vitiligo.

ambulans); in others, the lesions become attenuated during the summer or during the winter.

Sensation on the achromic areas is usually normal, and cases have been reported in which tactile sensibility was diminished, but

this was not constant either for the achromic or the hyperchromic parts. Also, the skin sweats less under the influence of pilocarpine.

Vitiligo and alopecia areata are sometimes observed in the same subject; the alopecia then persists for years, and there is only a partial regrowth of hair, of different colour to the original. According to Besnier, vitiligo sometimes develops in a patient already affected with alopecia areata; sometimes, in a subject affected with vitiligo of the scalp, alopecia areata follows the white patches of vitiligo. Besnier has seen two brothers, one affected with vitiligo, the other with alopecia areata. The relation between vitiligo and trophoneurotic alopecia is not definitely settled.

Diagnosis. — Vitiligo must be distinguished from patches of *sclerodermia*, which are sometimes surrounded with a pigmented ring; in sclerodermia, the skin is thickened and has lost its suppleness. In *anæsthetic leprosy*, there is no hyperchromia around the patches; moreover, dark red spots are present, and there is anæsthesia. In the *pigmentary syphilide* there is no real decolorisation of the parts, which appear whiter by contrast with the hyperchromic parts. *Pityriasis versicolor* is easily recognised by the presence of desquamation.

Prognosis.—Vitiligo is generally incurable; however, in young people it may disappear after some years.

Etiology.—This is still obscure, but it appears certain that vitiligo is of nervous origin. Leloir and others have found, in some cases, nerve lesions underneath the patches, characterised by breaking up of the myelin, multiplication of the nuclei, and sometimes disappearance of the axis cylinder in some of the nerves.

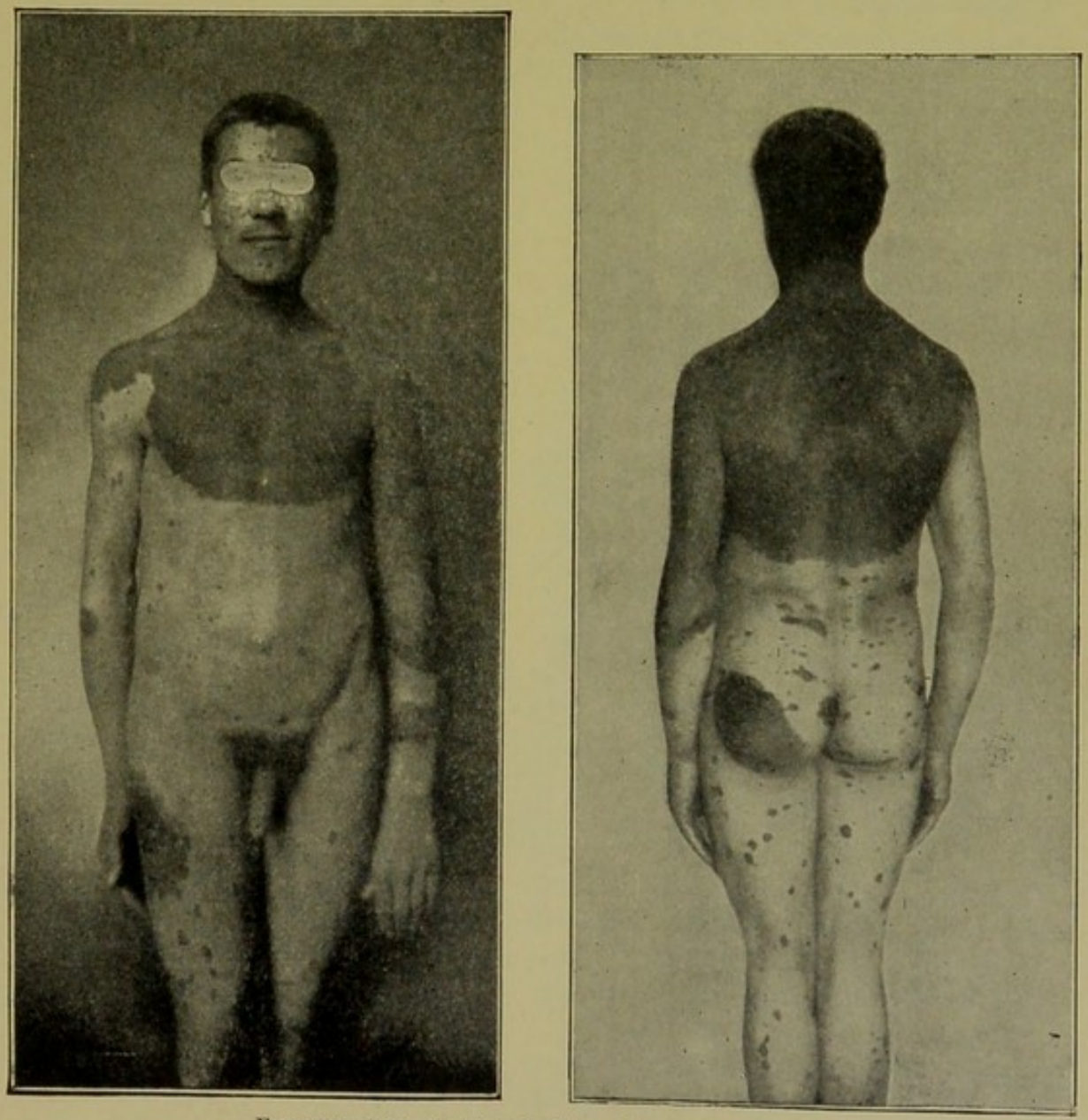
Sometimes vitiligo appears after an injury or strong emotion. It sometimes occurs in nervous affections, such as exophthalmic goitre, the preataxic stage of tabes, and among the insane. All these forms of *secondary vitiligo* have both a nervous pathogeny and a nervous etiology. But subjects who are free from any nervous affection may have vitiligo; these cases depend on an auto-intoxication produced by a disorder of nutrition, characterised by insufficient oxidation of nitrogenous matters. In fact, in patients affected with *primary vitiligo* there is a diminution in the excretion of urea and an increase in nitrogenous extractive matters; sometimes slight albuminuria. This primary vitiligo has still a nervous pathogeny, but a toxic etiology; the nervous change being secondary to the auto-intoxication. In some cases there is a local cause, such as the pressure of a truss; the vitiligo is then secondary to nerve changes caused by compression.

Treatment.—This includes cold douches to the spine, electric baths, and the continuous current. Besnier claims to have obtained good results by the prolonged administration of potassium bromide,

together with salt baths. The hyperchromia may be diminished by mercurial plasters. In dark subjects, the achromic parts may be darkened with a weak solution of nitrate of silver.

HYPERCHROMIA.

In its widest sense, hyperchromia is an exaggeration of the coloration of the skin, whether this is due to the normal colouring



FIGS. 53 and 54.—Partial nigrities; flat pigmentary nævi.

matter of the skin, to cutaneous pigment (*melanodermia*), or to some other colouring matter.

Hyperchromia may be congenital or acquired; the former includes pigmentary *nævi* and *nigrities*, which shows itself in the form of isolated black patches of various sizes, situated in one or more regions of the body. In reality, the black patches of partial nigrities, whatever may be their extent, are also pigmentary nævi. As regards acquired hyperchromia, there are several groups, including: first of all, *lentigo* and *chloasma;* then hyperchromia of internal origin; parasitic hyperchromia; that due to traumatism or some external cause; and lastly, that produced by the ingestion of certain drugs.

Hyperchromia of Internal Origin.—First of all must be mentioned *Addison's disease*, in which the cutaneous pigmentation is so marked that it originated the name of "bronze skin." The regions first affected are the lumbar region and flanks, then the neck, face and hands, finally the whole body. The mucous membrane of the palate and cheeks often has the same colour.

Fig. 55.—Acanthosis nigricans.

In *leprosy*, there are also pigmentary changes; in certain parts where the pigment accumulates the colour varies from tawny yellow to dark brown.

In *pellagra*, the pigmentation of the hands and face follows erythema, and is accompanied by desquamation.

Sclerodermia is also associated with large pigmentary patches.

We need only mention here the *pigmentary syphilide* and the pigmentation secondary to various syphilitic eruptions.

In tuberculous and malarial *cachexia* there is sometimes a more or less pronounced earthy pigmentation. In *cancer*, the skin has a characteristic yellowish or straw colour. In general *xanthelasma* the skin, even in the absence of biliary jaundice, has a peculiar colour, named by Besnier *xanthochromia*. In *saturnine intoxication*, the hæmatogenous

icterus arising from the rapid destruction of blood corpuscles gives the skin a peculiar gray or lead colour.

In certain *nervous diseases*, exophthalmic goitre for instance, pigmentary patches may occur on various parts of the body. We may also mention a curious case observed by Girode, in which there was melanodermia distributed in the direction of the left thoracic and brachial nerves, without antecedent zona.

Among the hyperchromias of internal origin may also be included a special dermatosis, named by Pollitzer and Janowsky *acanthosis nigricans;* a better name would be *papillomatous melanodermia.* This affection occurs exclusively in patients suffering from abdominal cancer. The pigmentation, accompanied by a warty or papillomatous condition of the skin, occurs chiefly on the neck and nape, and in the articular folds, axillæ, groins and intergluteal fold. On the trunk it may be complicated by warts, similar to flat senile warts.

Parasitic Hyperchromia.—*Phthiriasis* (pediculosis) is the type of parasitic diseases which eventually lead to pigmentation of the skin, owing to frequent and prolonged scratching (vagabond's disease). In severe cases, pigmentation of the nails and buccal mucous membrane has been observed, similar to that in Addison's disease. Melanodermia may also occur in chronic *scabies*, and blue spots are produced by *pediculi pubis.*

Hyperchromia of Traumatic or External Origin.—The pigmentation so frequently observed in chronic dermatoses, such as chronic eczema, lichen, and prurigo, is due to repeated scratching. The brown coloration of the skin occurring in connection with varicose veins of the lower limbs is probably due to obstruction of the venous circulation.

In some persons, the prolonged pressure of a truss or corset may cause local pigmentation of the skin; also, the application of blisters, tincture of iodine, chrysarobin, nitrate of silver, etc., especially in dark-skinned persons. Prolonged exposure to the sun, wind, or fire may also cause pigmentation. Accidental or artificial tatooing may also be mentioned in this connection.

Medicamentous Hyperchromia.—The most common form of this is the slaty colour of the skin observed after repeated cauterisation of the throat or other mucous membrane by *nitrate of silver;* sometimes after the prolonged administration of this drug internally, in epilepsy for instance.

Arsenic, when given for long periods, sometimes causes a brown discoloration of the skin. The red patches of psoriasis, under the influence of arsenical treatment, may become tawny; but cases of psoriasis may be of a bistre colour in patients who are not taking arsenic.

LENTIGO.

This is an eruption of small round spots, varying from a pin's head to a lentil in size, and of a yellowish brown or dark brown colour. They occur usually on the cheeks and forehead, sometimes on the neck, back of the hands, and other exposed parts of the body. This localisation shows that their production is influenced by the sun's rays (*ephelides* or "freckles"); however, they may be found on parts of the body which are not exposed. We must therefore assume a certain predisposition, which is met with chiefly in red-haired individuals, and in anæmic and lymphatic subjects.

Lentigo generally appears between the ages of ten and twenty-five, most commonly in the spring and summer. It disappears in the winter, to recur in the following spring. It often disappears or fades in old age.

Lentigo must be distinguished from *pigmentary nævi*, which are congenital spots (birth-marks) usually larger and less numerous, and may occur on any part of the body. In *xerodermia pigmentosum*, telangiectases are present, in addition to pigmentation; the skin becomes dry, and exfoliates in places, and cancerous lesions develop in certain parts of the skin.

MALIGNANT SENILE LENTIGO.

This dermatosis was first described by Hutchinson. It begins by a pigmentary spot situated on the face, on the upper part of the cheek, eyelid or conjunctiva. There may be one or more spots of variable size, which become darker or fainter, and disappear at certain points.

Malignant lentigo may persist for years; but, after a time, an epithelioma develops on or around the spot, and grows rapidly. When this tumour is removed, it almost always recurs; finally, the glands become involved.

Although malignant lentigo usually occurs in old people, Dubreuilh observed a case in a woman aged forty, which had commenced at the age of eighteen.

CHLOASMA.

This is constituted by pigmentary spots which occur chiefly on the forehead and temples, but may be met with on other parts of the face, on the cheeks and chin. They are not small spots like

those of lentigo, but larger and irregular, yellowish brown, with sharply defined or diffuse borders. On the forehead, chloasma extends to the margin of the hairs.

This affection is met with exclusively in women, and usually appears during pregnancy, when the face becomes pigmented, like the areola of the breast, the linea alba, and other parts of the body. However, it may occur in women who have never been pregnant, but who suffer from some uterine or ovarian affection. Chloasma often disappears after pregnancy, but may only become attenuated; it usually disappears at the menopause.

TREATMENT.—Persons predisposed to lentigo may escape it by avoiding the sun's rays, and by wearing veils and gloves. Curative treatment is difficult to carry out, both in lentigo and chloasma. Hardy recommended the following lotion, applied every night, and diluted with tepid water:—

Perchloride of mercury . . .	1 part
Sulphate of zinc	2 parts
Acetate of lead	2 „
Alcohol	Q.S.
Distilled water	250 „

This causes desquamation, and the lotion should be discontinued when this becomes well marked. During the day zinc ointment should be applied. Kaposi employed more energetic treatment, which consists in the application of compresses soaked in a solution of perchloride of mercury (1 per 100) and retained for four hours; this forms a blister, which is punctured, and then dressed with zinc ointment.

Unna recommends a rather painful form of treatment, which consists in: (1) the application of resorcin paste (50 per 100) for three or four hours; (2) after removing the paste and cleansing the skin, the application of a gelatin-glycerine varnish, which forms a coating to the epidermis. After a few hours this coating is removed and brings away the epidermis with it. The new epidermis which grows under the coating presents no abnormal pigmentation, but this reappears some time afterwards; hence this procedure is not to be recommended.

Daily applications of solution of borax, or painting with tincture of iodine may also be tried.

PIGMENTARY NÆVI.

The term *nævus* is applied to all congenital lesions of the skin, characterised by exaggerated development of the cutaneous vessels, or by an abnormal production of pigment. The lesion is usually

permanent, and limited to one region of the body. Some nævi develop some years after birth.

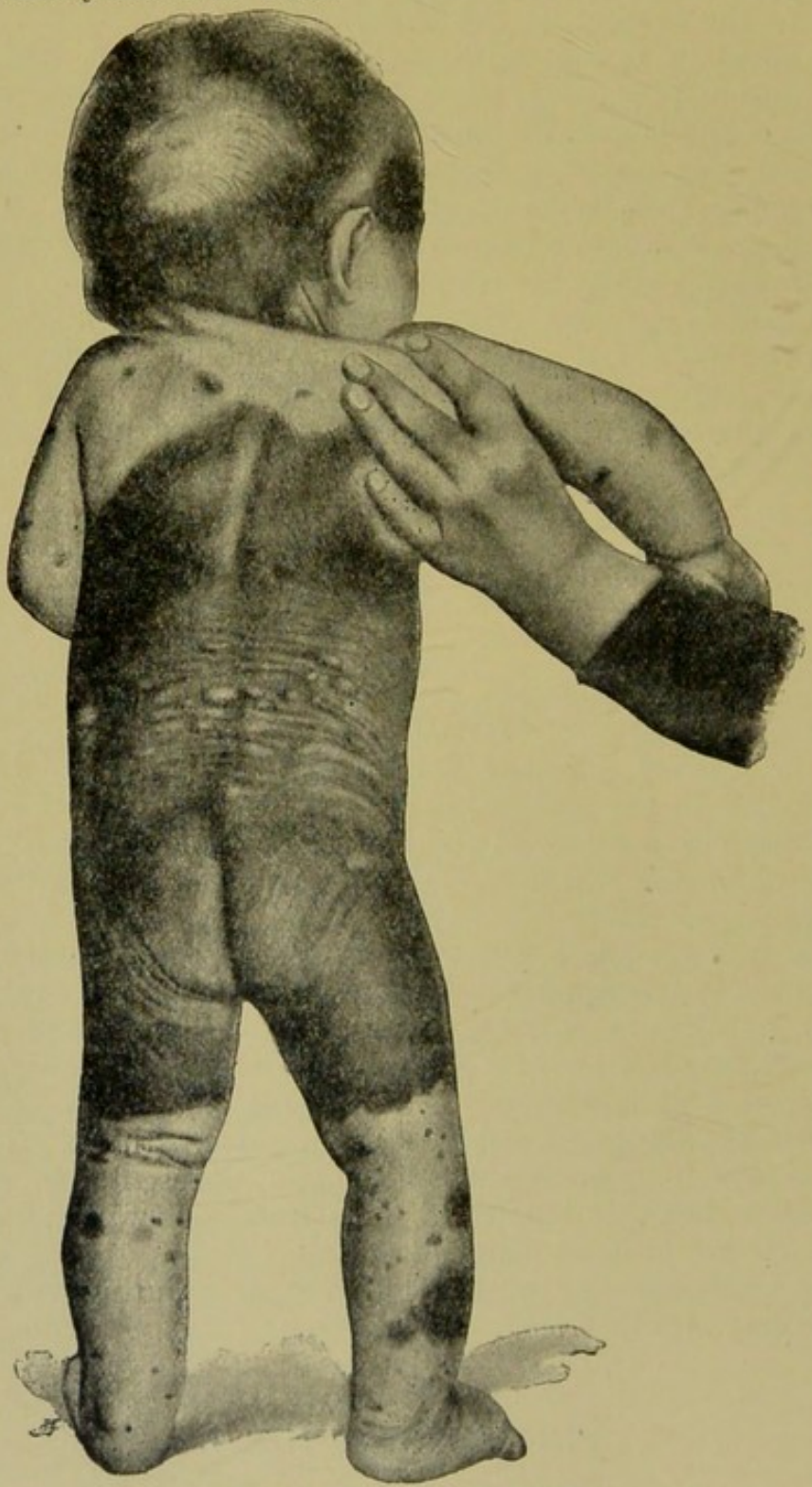


Fig. 56.—Hairy pigmentary nævi.

In accordance with this definition, nævi are divided into two groups: *pigmentary nævi* and *vascular nævi;* the latter being again

subdivided into hæmatic and lymphatic, according as they are formed by dilatation of the blood-vessels or lymphatics. We are only concerned in this paragraph with pigmentary nævi.

Pigmentary nævi are formed by patches of pigment, with or without thickening of the subjacent skin. They are subdivided into flat nævi, warty nævi, and hypertrophic nævi.

Flat Pigmentary Nævi.—These are superficial spots with a smooth surface, round, oval or irregular in form, varying in size from that of a lentil to large patches, light or dark brown in colour, generally multiple and disseminated, rarely single, and sometimes covered with hairs (hairy nævi). Lenticular nævi, when situated on the face, are sometimes rather attractive in appearance, and are commonly known as "beauty-spots."

Warty Nævi.—In this form the skin is thickened and studded with small elevations, formed by groups of hypertrophied papillæ; they are usually of a brown colour, which often becomes black from mixture with seborrhœic secretion and dirt. They often present thick or fine hairs on the surface. They may occur on any part of the body, but are more commonly observed on the scalp, neck, back and limbs. On the palms and soles they become complicated with callosities. They may coexist in the same subject with vascular nævi. They often occur along the course of nerves (zoniform nævi), or along the lines which demarcate their areas of distribution. Hebra has reported a case of nævus which covered the pelvis and upper part of the thighs, like bathing drawers.

Hypertrophic Nævi.—These are tumours rather than nævi. They increase after birth, and then form large, pedunculated or sessile growths. (See *Molluscum fibrosum.*) On section, they present a yellow gelatinous appearance. They are formed by young connective tissue, rich in cells; some are due to proliferation of fatty tissue (*nævi lipomatodes*).

Pigmentary nævi rarely disappear; on the contrary, they tend to increase and persist during life. Their prognosis is aggravated by the possibility of an epithelioma developing on the surface, a complication which is common in the working-classes, where nævi are often exposed to irritation.

PATHOGENY.—The distribution of some nævi along the course of nerves seems to support the theory of their nervous origin; but, according to Kaposi, this distribution is due to the fact that the embryonic elements which form the cutaneous areas develop separately, and these areas are superposed on the areas of distribution of the nerves.

TREATMENT.—Flat pigmentary nævi may be destroyed by caustic acids or Vienna paste, but care must be taken not to produce cicatrices, which are more disfiguring than the original nævus. Large

warty nævi may be treated with *electrolysis.* The treatment of pigmentary nævi by *radium* has not given such remarkable results as in vascular nævi.

Treatment by X-rays may also be employed. According to Gastou, this should be applied in strong doses, about 5 units H per sitting. A series of two or three sittings should be given at intervals of a week. This generally produces radiodermatitis. When this has subsided the series is repeated, with intervals of three weeks between each series. In the case of hairy and warty nævi very penetrating rays are required (8 to 9 of the radiochromometer).

Hypertrophic nævi require surgical treatment.

VASCULAR DERMATOSES.

ANGIOMA. CUTANEOUS HÆMATANGIOMA.

Hæmatangiomas or *vascular nævi* are constituted by an abnormal vascularisation of the skin, limited to one part of the body, congenital or appearing during the first months of life. They are subdivided into *flat nævi*, and raised or *tuberous nævi*. The old distinction of arterial and venous nævi is incorrect, as all nævi are capillary.

Flat Nævi.—These vary in size from small spots to more or less extensive patches (port-wine mark). The colour may be pale red, bright red or purple. The borders are usually round, sometimes irregular. They are sometimes covered with hairs, and may be situated over the track of a nerve. They may occur on any part of the body, but are more common on the face, around the natural orifices and on the nape of the neck, less common on the scalp. They may occur on the mucous membranes. They become paler by compression; redder and more turgid under the influence of effort, emotion and crying.

Tuberous Nævi.—These project more or less above the skin. Their surface is often lobulated, giving them the appearance of mulberries or raspberries. They have the same distribution and properties as flat nævi, but are of a darker colour and more turgescent. They may be transformed into erectile tumours.

The surface of vascular nævi sometimes presents a warty appearance like that of some pigmentary nævi. This condition is due to papillary hypertrophy and hyperkeratosis of the skin covering the nævus. Warty vascular nævi are sometimes observed on the face, and may be mistaken for sebaceous adenomas, but differ in their red colour. They may also occur elsewhere (Fig. 58). I have seen a zoniform, warty vascular nævus, which occupied the whole length of one of the lower limbs, in the form of a linear band situated over the course of the internal saphenous nerve.

The evolution of nævi is variable. The majority increase during the first years of life, and then persist indefinitely; small nævi sometimes shrink and disappear spontaneously, leaving a white or

pigmented cicatricial spot. But large nævi have a tendency to extend both superficially and deeply; on the face they may invade the buccal mucous membrane or the conjunctiva, and extend towards the deeper parts, which become displaced or atrophied by pressure.

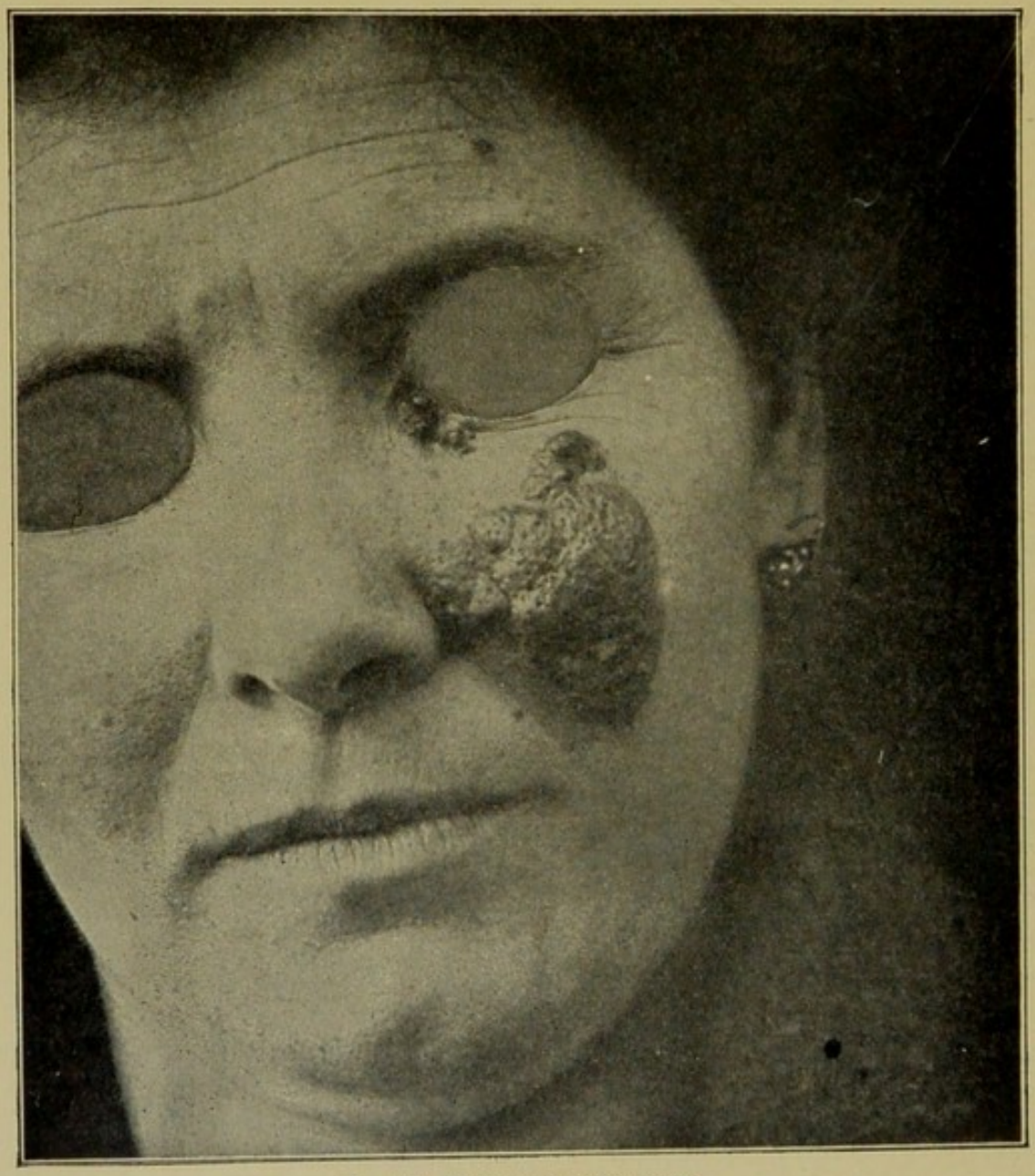


Fig. 57.—Tuberous vascular nævus.

Large nævi are therefore serious, on account of the disfigurement and neuralgic pains which they produce, and their tendency to increase in size and extent. Tuberous nævi, when exposed to traumatism, may give rise to severe hæmorrhage; they may also become inflamed or gangrenous; after which they are sometimes cured.

PATHOLOGICAL ANATOMY.—Nævi are situated in the papillæ and superficial layer of the dermis. They are formed by dilated, tortuous vessels, which communicate with each other; some old, some newly formed. Around the vessels is a formation of young connective tissue, which varies in quantity in different forms of nævi. These changes are more marked in tuberous and cavernous nævi.

ETIOLOGY.—The causes of vascular nævi are no better known than those of pigmentary nævi. The idea that they are caused by maternal

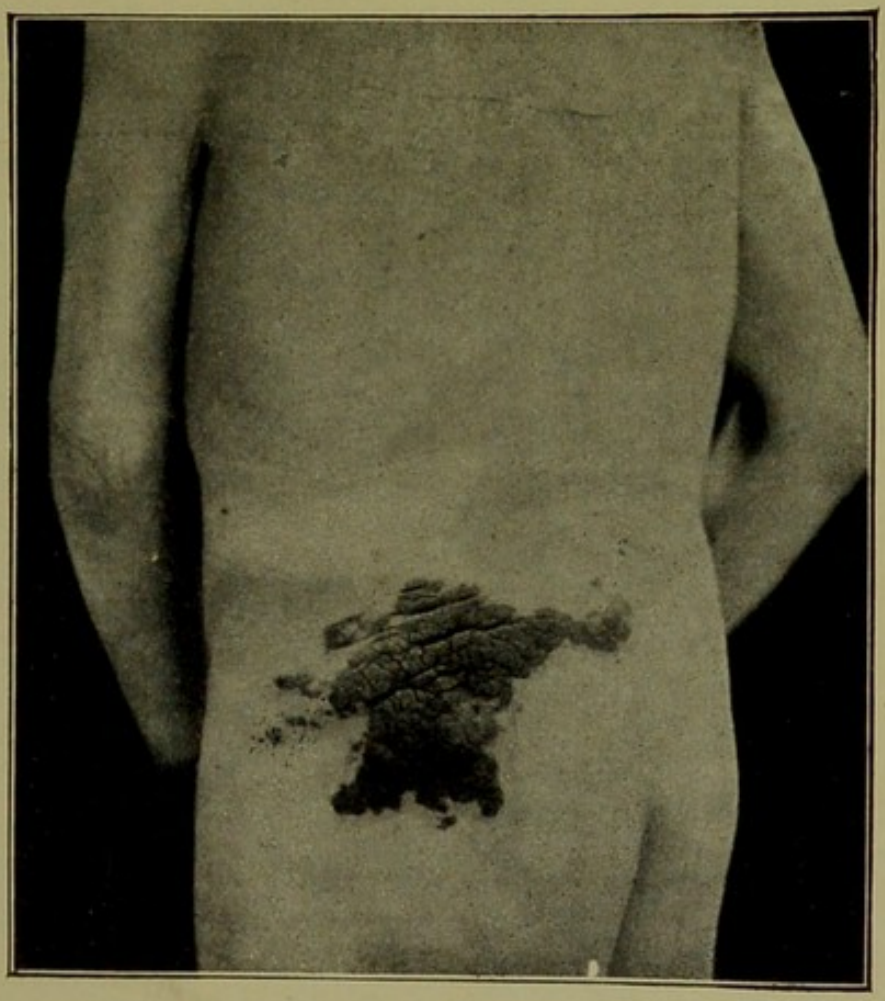


FIG. 58.—Warty nævus.

impressions acting on the fœtus during pregnancy does not rest on a scientific basis, but it must be admitted that there are certain facts which tend in favour of this idea.

TREATMENT.—If a nævus, instead of shrinking after birth, shows signs of increasing, it requires treatment. Small superficial nævi may be treated with caustics, such as Vienna paste, vaccination, scarification, the electro-cautery or thermo-cautery. Larger nævi may be treated by electrolysis, the needle being attached to the

positive pole of the battery; this produces coagulation of fibrin. The negative pole causes destruction of tissue, and should only be used for tuberous nævi. More rapid results are obtained in the

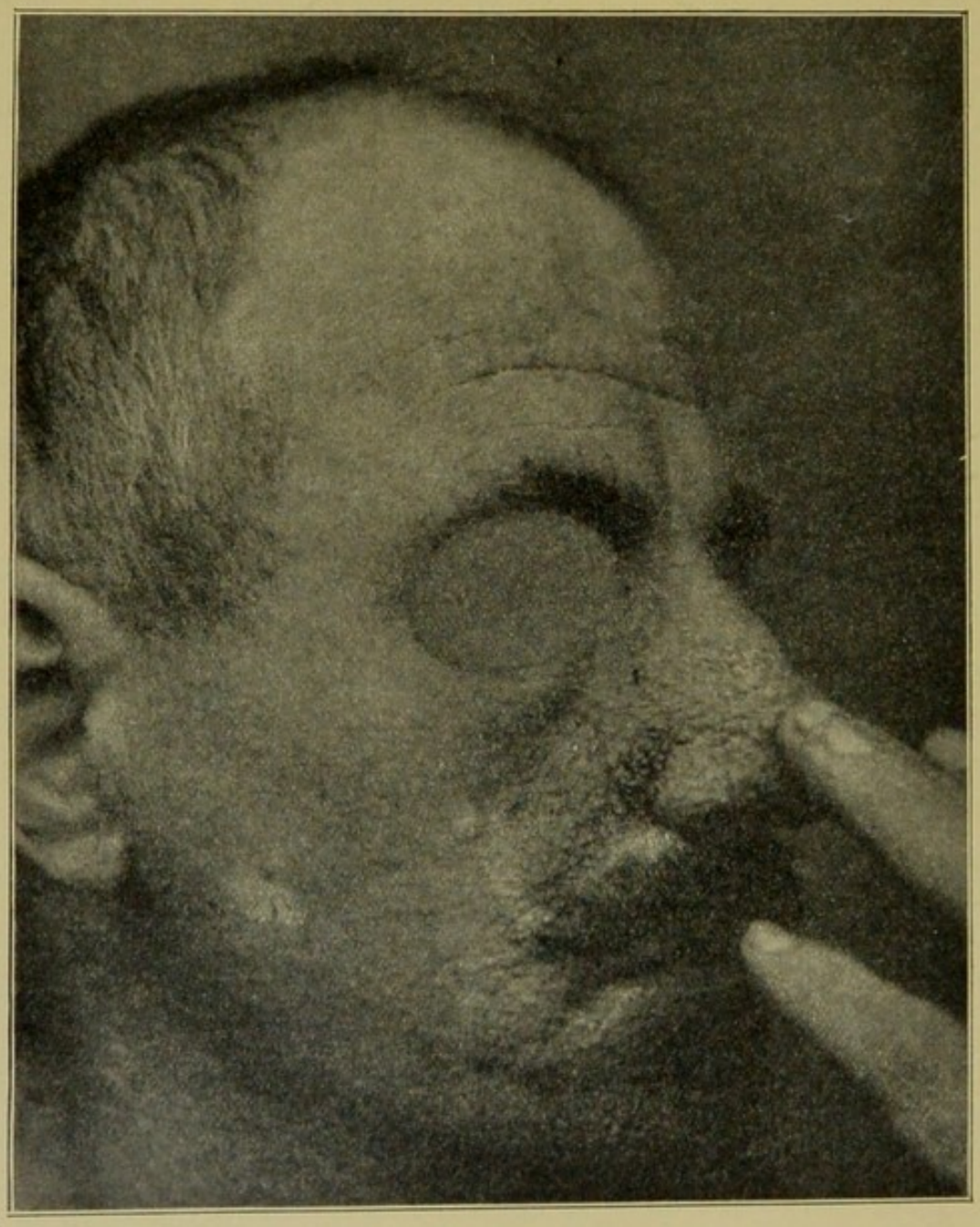


Fig. 59.—Warty vascular nævi.

latter by bipolar electrolysis, both positive and negative needles being inserted into the nævus.

But all these methods, including electrolysis, which is certainly the best of them, may now be supplanted either by X-ray or radium

treatment. However, some authors hold that, in the case of tuberous nævi and erectile tumours, radiotherapy is inferior both in rapidity and in certainty to electrolysis, especially bipolar electrolysis.

Treatment of Vascular Nævi by Radiotherapy (Gastou).[1]—In the absence of radium, which is the best treatment for nævi, radiotherapy may be used both in adults and in children, provided the child can be induced to remain quiet long enough.

In the treatment of nævi by X-rays, as in all other lesions, the following points must be considered : (1) the age of the patient, and the nature and situation of the lesion ; (2) the time of exposure ; (3) the distance of the part treated from the anticathode ; (4) the quality of the rays ; (5) the quantity of rays given at each sitting.

In a general way, the following technique is the best :—

(1) Frequent sittings, with weekly intervals.
(2) Duration of sittings varying with age of patient, and situation and variety of nævus ; the duration varying with the quantity of H to be absorbed at each sitting.
(3) Intervals between sittings calculated so that after the absorption of a dose of 5 or 6 H in the adult, and a smaller dose in proportion to age in children, treatment is suspended for two or three weeks.
(4) Absorption at each sitting of a dose of 1½ to 5 H, according to the age of the patient and the variety of the nævus.
(5) Employment of rays with slight penetrating power (4 or 5 of radiochromometer).

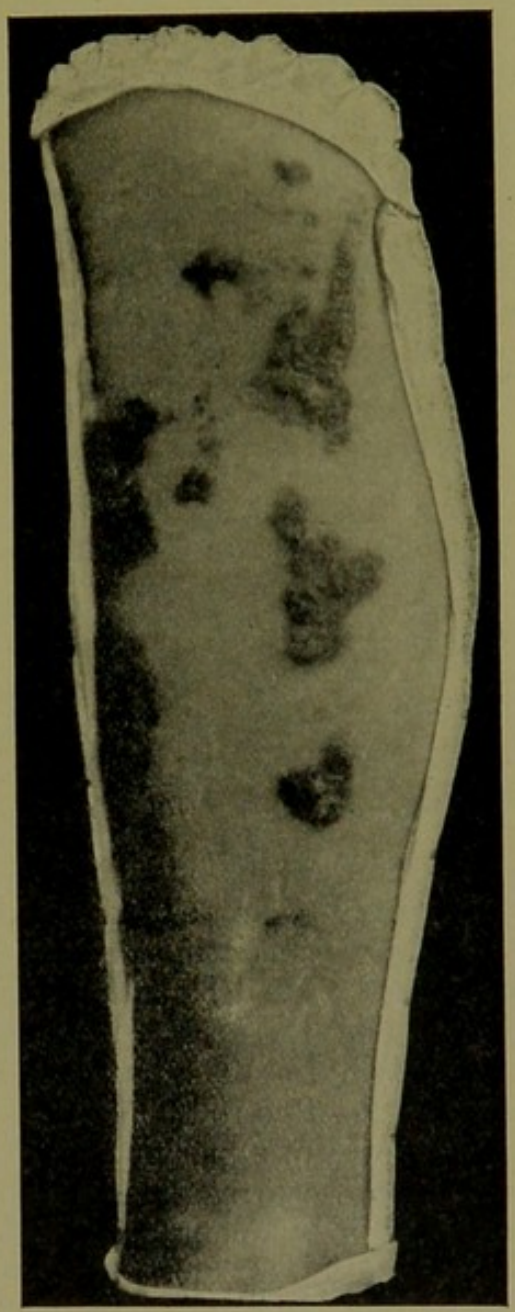


Fig. 60.—Zoniform warty vascular nævus of the leg. (St Louis Hospital Museum.)

These are the general indications, but there are variations in the technique for each variety of nævus.

The results obtained by radiotherapy are excellent, and similar to those obtained by means of radium. The resulting surface is

[1] For description of radiotherapy, see p. 277.

white and not cicatricial. By conforming with the above rules, no hæmorrhage is produced. The reaction is bullous and not ulcerative, and may be treated with moist compresses and sterilised vaseline. The reaction lasts for a month or six weeks, without producing chronic radiodermatitis. The technique differs in flat and in tuberous nævi.

In *flat nævi*, sittings should be given weekly, using a dose of 1½ H in children, 2 or 3 H in adults. Rays No. 4 of the radiochromometer should be used. After three weeks, there should be an interval of two weeks; that is, after the absorption of 3 or 4 H in a child, and 5 H in an adult. After this, an additional 5 H should be given. A reaction is generally produced when 8 to 10 H have been absorbed. After the reaction has subsided, the treatment is repeated. Ulcerative radiodermatitis is avoided by stopping the treatment every time 5 H has been absorbed at several sittings.

In *tuberous nævi* the technique is the same, except that the doses at each sitting are 2 to 3 H for a child, and 5 H for an adult. The treatment is suspended after the absorption of 8 H in children and 15 H in adults, when radiodermatitis appears; after this is cured the treatment is renewed.

Erectile tumours require more energetic treatment; but this should be graduated so as not to produce immediate radiodermatitis. First of all, three or four sittings of 2 H are given at weekly intervals, using the less penetrating rays. If there is no reaction, two sittings of 5 H are given with the same rays (radiochromometer 4). A bullous reaction then occurs, and after this is cured, sittings of 5 H are given with more penetrating rays (radiochromometer 7 to 8). After two sittings, the treatment is continued at intervals of two or three weeks. After a time the tumours gradually subside.

To sum up, in the treatment of vascular nævi by the X-rays it is necessary to provoke inflammation or reaction by a sufficient dose of the less penetrating rays, then to modify this inflammation in the direction of cell destruction by more penetrating rays, always taking care to delay superficial radiodermatitis as far as possible.

Treatment of Vascular Nævi by Radium (Wickham and Degrais).[1]—Before 1906, radium had only been applied to vascular nævi by way of experiment. Some operators obtained decolorisation, but only after a reaction, and on small areas, in flat nævi. In fact, with the insufficient apparatus then available, and the absence of any indication for dosage, it was impossible to carry out this treatment, especially in extensive flat nævi and tuberous nævi. It is only since we have learned to regulate the dosage of radium, by means of improved apparatus, that we have been able to apply to nævi the dosage which we formulated in 1906. The recent rapid progress dates from this period.

The first case treated was a flat nævus on the cheek of a child of six months. Knowing from our experience in the treatment of epitheliomata that the external radio-activity of the apparatus would produce a slight reaction in a given time, the length of

[1] For description of radiumtherapy, see pp. 285, 338.

exposure was determined beforehand. This nævus was cured in two months.

However, it appeared that a reaction should as far as possible be avoided, and as we had succeeded by exact dosage in curing epitheliomas without inflammation, we endeavoured to obtain the same result in the case of vascular tumours.

The treatment of angiomas by radium depends on the property of *decongestion* possessed by this substance. Owing to this property, it decolorises flat nævi, and by prolonging its action the more deeply penetrating rays continue the process observed on the surface, and finally lead to diminution, or even complete levelling, of large vascular tumours.

Radium, for the treatment of nævi, is used in the form of finely powdered radium mixed with sulphate of barium. This is distributed as evenly as possible on a thin layer of varnish spread on a metal plate. This forms an apparatus the utilisable activity of which is proportional to the intensity and quantity of the salt employed, as well as to the surface area of the apparatus. By knowing the dimensions of the apparatus and its therapeutic surface in square centimetres, the initial activity and weight of the salt employed, the utilisable spherical radiation, the tenure in alpha, beta, and gamma rays, and the extent of surface treated at each application, we can determine the length of exposure required for a given case.

Apparatus may also be used in which the radium is fixed on linen by means of varnish. This form of apparatus is flexible, and can be moulded to irregular surfaces.

The treatment consists in the application of the apparatus to the nævus, differing according to the form of the nævus and the dosage to be employed.

In flat nævi, large doses may be given by direct application of powerful apparatus for short periods, which causes a certain degree of curative inflammatory reaction; or small doses transmitted through graduated screens, for longer periods.

In cavernous nævi, doses must be used which do not produce inflammation of the surface, or only a slight reaction.

The doses may be given directly by short but frequently repeated applications, or by various forms of filtration. Among the different methods of technique which we have tried, that of *crossed fire*, with or without filtration, has given good results in raised nævi, and is especially useful in angiomatous tumours. This method consists in exposing the tumour to several apparatus at the same time in opposite directions, either directly or with the interposition of screens; the length of exposure being less than that which would produce a cutaneous reaction in the case of each apparatus. The

more penetrating rays cross each other in the deeper parts of the tumour, and the duration of their action corresponds to the product of the duration of application of each apparatus by the number of apparatus. By this method, all the rays act, the less penetrating

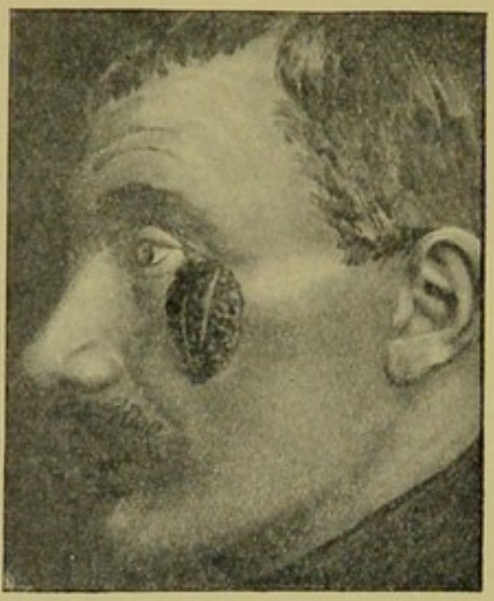


Fig. 61.—Tuberous nævus. (Wickham and Degrais.)

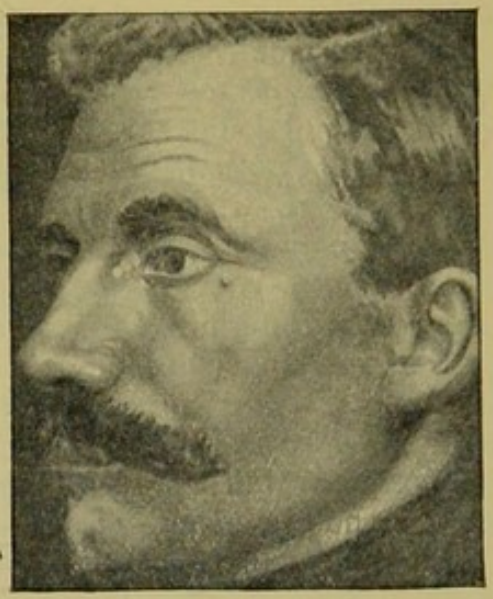


Fig. 62.—The same, after treatment by radium.

Fig. 63.—Tuberous nævus. (Wickham and Degrais.)

Fig. 64.—The same, after treatment by radium.

and the more penetrating, especially the latter, and the tumour, acted on in all its parts, undergoes rapid resolution, without any solution of continuity of its surface.

In most cases we used a method of filtration which lets through

the beta and gamma rays; but, whatever may be the method of technique adopted, the value of the results always depends on the dosage.

FIG. 65.—Tuberous nævus. (Wickham and Degrais.)

FIG. 66.—The same, after treatment by radium.

FIG. 67.—Tuberous nævus. (Wickham and Degrais.)

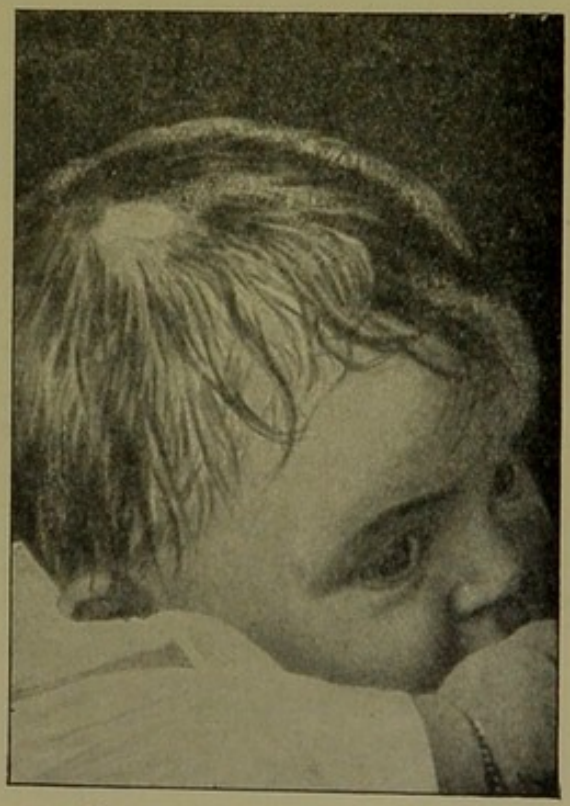


FIG. 68.—The same, after treatment by radium.

The results as regards vascular tumours are remarkable; although in the flat forms the ultimate result is sometimes doubtful, in the

tuberous forms of nævi, especially in infants, when the tumours are actually growing and other forms of treatment are inconvenient, painful, or even dangerous, the favourable action of radium is beyond question. In no case have we had actual failure; there has always been considerable diminution, and often complete disappearance of the nævus.

With regard to flat nævi, great reserve is necessary in the paler forms, for these may easily be concealed by powders or other means. In the more highly coloured forms, a too powerful action may cause depressions; but the dose which gives the best result in each case can nearly always be determined by practice. We should always endeavour to reduce the inflammatory reaction to a minimum, and by means of prolonged applications with appropriate filtration, decolorisation may be effected in many cases without any marked reaction.

By this means the skin is sometimes restored to its normal condition. When stronger doses are necessary, the down on the skin disappears and the skin is left with a less downy surface than normal. It requires a fairly acute reaction to render the skin whiter and smoother than the healthy skin, but the result is preferable to the presence of a highly coloured and projecting nævus.

After these acute reactions (when they have been considered necessary) it is not a cicatrix, in the usual sense of the word, which is formed, but a special tissue, to which we have given the name of tissue of restitution. In some cases pigmentation developed at the edges of the parts treated, and in one case small telangiectases; but, as a rule, the tissue of restitution is smooth, supple, and level with the surrounding skin.

Radium may be applied to any region, even the eyelids, where short and frequently repeated applications cause no inflammation of the conjunctiva. Nævi of the lips soon disappear after short applications.

To sum up, the elective action of radium which we have established, a kind of specific action, by which angiomas are decolorised and levelled, is especially useful in projecting, tuberous, erectile vascular nævi, even in certain monstrosities, and constitutes a therapeutic agent which appears to us superior to all others. The convenience and painlessness of the applications adds to their value in the treatment of children and young infants, who can be treated during sleep.

Wickham and Degrais give the following details for the treatment of vascular nævi by radium :—

(A.) *Flat superficial nævi.* On each of the spots to be treated, apparatus is applied of 500,000 activity, bearing from 1·5 to 2 milligrams of radium per unit of surface. The duration and number of applications are as follows :—

1. For pale nævi : two or three sittings of one hour to one and a half hours ;
2. For red nævi : three or four sittings of one and a half to two hours ;
3. For purple nævi : two sittings of two to two and a half hours.

(B.) *Nævi level with the surface, but extending deeply:* three or four sittings of three to three and a half hours.

(C.) *Projecting nævi, with a smooth or mammillated surface:* These are treated like the preceding, but they sometimes offer considerable resistance, and the first applications only result in reducing them to the level of the skin. They are then treated again like ordinary flat nævi.

With regard to *filtration*, this may be effected by small wads of cotton wool, from 1 to 1½ centimetres thick, enveloped in gold-beater's skin. More recently, Wickham and Degrais have used metal screens, introduced by Dominici, which have a much greater power of absorption than the wool screens, and only allow the ultrapenetrating rays to pass.

CUTANEOUS TELANGIECTASIS.

By this name must be understood acquired telangiectases, while nævi are congenital telangiectases. The former, like the latter, are formed by dilatation of the capillaries and fine blood-vessels of the skin, and by the formation of new vessels. Telangiectases thus differ from cutaneous varicosities, which are produced only by the dilatation of pre-existing veins.

Telangiectases appear in the form of spots or patches, varying in colour from pale red to dark purple, and covered by fine arborescent vessels. *Secondary telangiectases* may occur in certain dermatoses, such as keratosis pilaris, cheloid, lupus, sclerodermia, acne rosacea, xerodermia pigmentosum, etc. They may also occur in heart disease and in cirrhosis of the liver, in the form of small tuberous spots, of a bright red colour, resembling acquired nævi, which will be shortly described.

Apart from these symptomatic forms, there is an idiopathic form of *primary generalised telangiectasis*, the etiology of which is unknown. I have, however, observed a case of generalised telangiectasis in a woman after oophorectomy, and another case in a diabetic patient.

These idiopathic telangiectases occur as disseminated patches, which are more marked in some regions than in others, but may occur anywhere, even on the palms and soles. Sometimes they consist of small isolated spots, sometimes large, red, irregular patches studded with spots and lines of deeper red, with small areas of healthy skin here and there. Under a lens, these spots and patches are seen to be formed by a very close network of capillary vessels. The colour disappears momentarily under pressure of the finger; it is increased by exertion, fatigue, and changes of temperature, and is more marked in the evening, after the fatigue of the day.

The affection develops gradually, but may remain stationary for long periods. Some of the smaller spots may disappear. It is an affection of adult age; in a case of Hillairet's, it commenced at the age of fifty. It does not appear to be hereditary.

In another more common form the telangiectases develop in persons over forty years of age, in the form of small, bright red, punctiform spots, slightly raised, varying in number, and usually situated on the trunk. These telangiectases, which are connected with senility, I have described as *acquired nævi.*

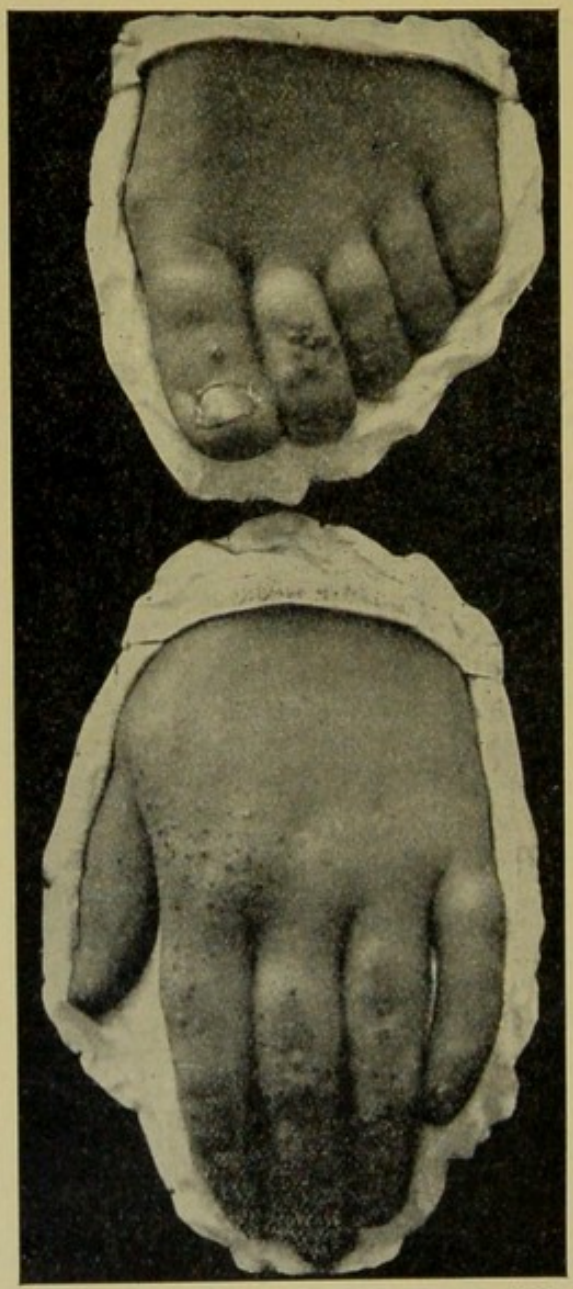


Fig. 69.—Verrucose telangiectases (angiokeratoma). (St Louis Hospital Museum.)

VERRUCOSE TELANGIECTASIS, OR ANGIOKERATOMA.

This lesion (first described by Bazin by the name of *nævus a pernione*, and later by Dubreuilh, Mibelli, and Pringle) occurs on the extremities, usually on the dorsal surface of the fingers and toes.

It consists in small elevations with a purpuric appearance, which disappears under pressure, generally covered with warty projections the size of a pin's head or hemp seed, and of a yellowish gray or purple colour. It begins in the first years of life, and then increases. It may occur in several members of the same family. The majority of cases suffer from cyanosis of the hands, and chilblains. Some authors think that angiokeratoma is tuberculous in nature.

The lesion is at first a telangiectasis, with secondary development of keratoma.

TREATMENT consists in destruction of the lesions by electrolysis or the galvano-cautery.

CUTANEOUS LYMPHANGIOMA.

This is a tumour formed by the exaggerated development of the lymphatic vessels at a certain part of the skin. Lymphangioma may be circumscribed or cystic.

Circumscribed lymphangioma occurs principally on the face, lips, tongue, neck, and trunk. The lesion consists of a mulberry-like mass of vesicles, which are opaque at their base and more or less transparent at their apex, resembling sago grains. The base of the tumour is indurated. Around the principal mass there are always detached islands and scattered isolated elements. On puncture of the vesicles, a clear, serous liquid escapes, charged with lymphatic cells. Lymphangioma is subject to inflammatory outbreaks, after which the lesion increases.

Histological examination shows that the lesion is formed by dilatation of the papillary and subpapillary lymphatics.

Lymphangioma may occur in all countries, but is more common in the tropics. It may be congenital, or may appear some time after birth.

Cystic lymphangioma forms a large tumour; the congenital multilocular serous cyst, which occurs usually in the cervical region, but may be met with elsewhere.

TREATMENT.—The treatment of circumscribed lymphangioma consists in destruction of the tumours by means of electrolysis or the galvano-cautery, or in excision.

LYMPHATIC VARIX.

We shall only deal here with dilatation of the cutaneous lymphatics; varix of the subcutaneous lymphatics belongs to the domain of surgery. Lymphatic varix of tuberculous origin will be described with cutaneous tuberculosis.

Cutaneous lymphatic varices usually occur in regions which are rich in lymphatic vessels, such as the groin, the antero-internal surface of the thigh, the anterior abdominal wall, the prepuce and penis, and the bend of the elbow. They may also occur in mucous membranes, chiefly on the buccal mucous membrane. The lymphatic dilatations appear in the form of translucent, vesicular elevations, resembling grains of boiled sago, arranged in lines or in irregular groups. The dilatations communicate with each other, so that lymph can be made to flow from one to another by pressure. They may coexist with varices of the subcutaneous lymphatic vessels.

In the same way that varicose veins may give rise to hæmorrhage, lymphatic varices may produce a discharge of lymph after strains, fatigue or injury. The lymph is an alkaline liquid, at first

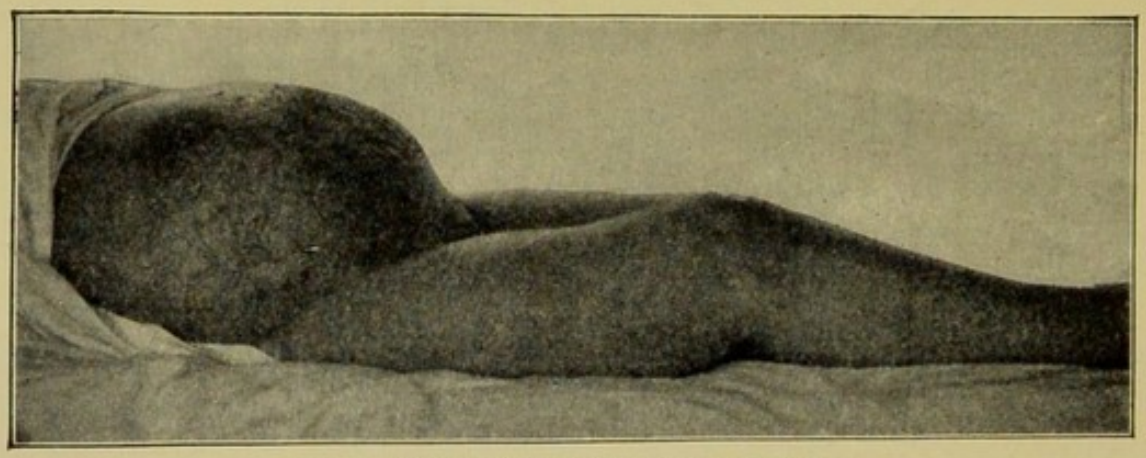


Fig. 70.—Lymphatic varices of the abdominal wall.

colourless, then white; it coagulates on exposure to air, and stiffens the linen.

Lymphatic varices are often observed on limbs affected with elephantiasis, and on regions which have been the seat of repeated attacks of erysipelas, as occurs in lymphatic subjects. They are then of mechanical origin, and are due to constriction or obliteration of the lymph channels, resulting from repeated inflammation. These varices develop in the papillary body of the dermis; the epidermis covering them is thinned, but otherwise unchanged. The varices are lined with flat epithelium, but they have no proper wall, the cavities being formed in the connective tissue of the papillary body.

Treatment.—This is the same as in elephantiasis. The varices should be obliterated by means of electrolysis.

HYPERTROPHIC AND ATROPHIC DERMATOSES.

EPIDERMIC HYPERTROPHIES.

ICHTHYOSIS.

Ichthyosis is a congenital, cutaneous deformity, characterised by disordered keratinisation of the epidermis, resulting in continual desquamation, and giving the skin the appearance of the skin of fishes. Although the affection is congenital, it is not apparent at birth, and only develops during the first months of life, sometimes not for a year or two. It then gradually increases till adolescence, and persists during life. Although some improvement may occur under treatment, ichthyosis is an incurable disease.

Like all defects in conformation, the affection is hereditary. Sometimes it skips a generation, but it always affects several members of the same family.

ETIOLOGY.—The cause of ichthyosis is unknown, but in some cases it appears to be connected with dystrophic congenital syphilis.

SYMPTOMATOLOGY.—The skin is dry and covered with desquamating epidermic scales, partially or completely adherent and sometimes imbricated. The whole epidermis presents a crackled appearance. Beneath the squames there is no trace of redness nor inflammation. The distribution is symmetrical and almost general, but more marked on the external surfaces of the limbs and around the knees and elbows, less marked in the groins, axillæ and articular folds, and on the face, where it is sometimes completely absent; the genital organs are little affected, and on the palms and soles it may be absent The intensity of the affection is proportional to the dryness of the skin, the regions where there is least cutaneous secretion being most affected. For the same reason, ichthyosis is less marked in summer, owing to increased cutaneous secretion.

The hairs are atrophied, and almost absent on the regions most affected. When the scalp is affected, the hair is dry and scanty.

The nails are also dry and brittle. There is no itching, but often a feeling of tension in the skin. Sometimes cutaneous sensibility is diminished. When itching is present, it is due to the presence of eczema, a not uncommon complication. Ichthyosis causes no disturbance of the general health, but the subjects are generally puny individuals, and their vulnerable skin is often the seat of artificial eruptions.

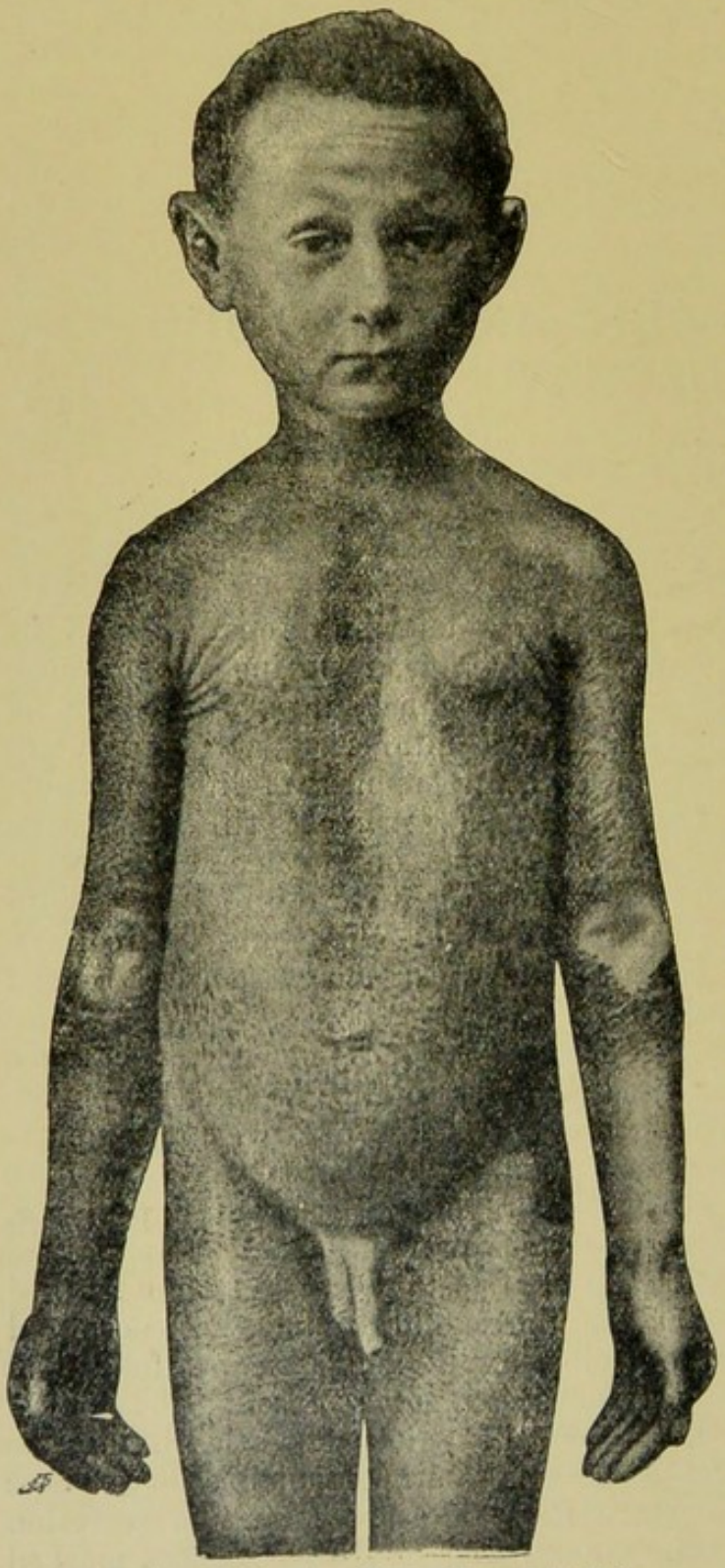


Fig. 71.—Ichthyosis. (Hecker-Trumpp-Apert.)

Varieties.—The most common form is *ichthyosis simplex*, which slightly resembles the skin of a fish. It is characterised by thin, shining, pearly, epidermic scales; sometimes broad, sometimes fine or pityriasic; white (*ichthyosis alba*), dirty gray, brown or black (*ichthyosis nigricans*).

Alibert gave the name *serpentine ichthyosis* to a form which resembles the skin of a snake. In this form the epidermis is gray, with darker patches in places, and covered with adherent scales; the skin is marked by furrows, forming rectangular or lozenge-shaped areas.

In another variety the scales, instead of being flat, form hard, conical elevations (*ichthyosis cornea*), or horny growths analogous to the spines of a

porcupine (*ichthyosis hystrix*). In these cases the skin may be as hard as that of a saurian or pachyderm.

Intra-uterine or Fœtal Ichthyosis.—Newly born infants with this affection present thickening and induration of the epidermis. The skin is of a dirty colour, hard and inelastic, and marked with deep fissures, due to stretching of the skin during growth. The child cannot close its mouth, and consequently cannot suck, so that it usually dies from inanition.

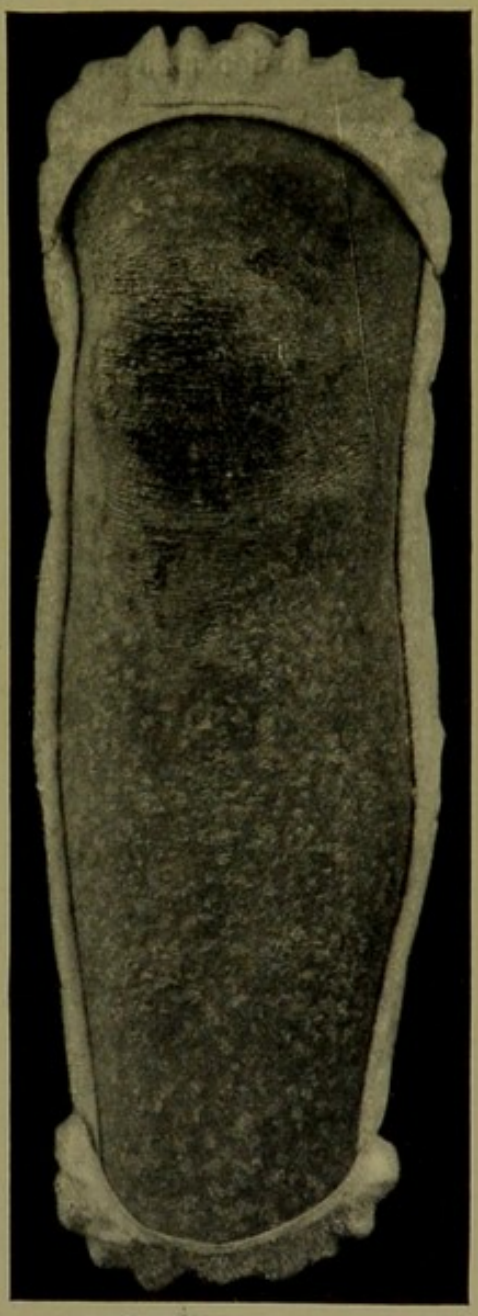


Fig. 72.—Ichthyosis.
(St Louis Hospital Museum.)

Pathological Anatomy.—The stratum corneum is hypertrophied, the stratum granulosum and stratum lucidum have disappeared, and the Malpighian layer is more or less atrophied. The papillæ of the dermis are usually atrophied, sometimes hypertrophied and infiltrated with embryonic elements. The hair follicles and sebaceous glands are atrophied and filled with horny masses. The sweat glands are partly atrophied.

Diagnosis.—Ichthyosis must not be confused with *keratosis pilaris* (sometimes called anserine ichthyosis), which is limited to the pilo-sebaceous follicles; but sometimes the two diseases coexist. Ichthyosis is easily distinguished from *pityriasis simplex*, which is not congenital, and is characterised by furfuraceous desquamation, with or without subjacent redness; and form *pityriasis rubra*, in which the skin presents red patches, and in certain places, characteristic circumpilary epidermic cones. *Palmar and Plantar keratosis* is constituted by horny thickening of these regions, which is rare in ichthyosis. In *nævus verrucosus*, the lesions are sometimes covered with squames, but the nævus is circumscribed, and sometimes situated over the course of one or more nerves.

Ichthyosis must be distinguished from the peculiar dry condition of the skin and constant epidermic desquamation observed in old

people, in tuberculous subjects, and in patients affected with chronic diseases (cachectic ichthyosis); also from the ichthyosiform desquamation seen in certain diseases of the nervous system, which must be regarded as a trophic disorder of the skin.

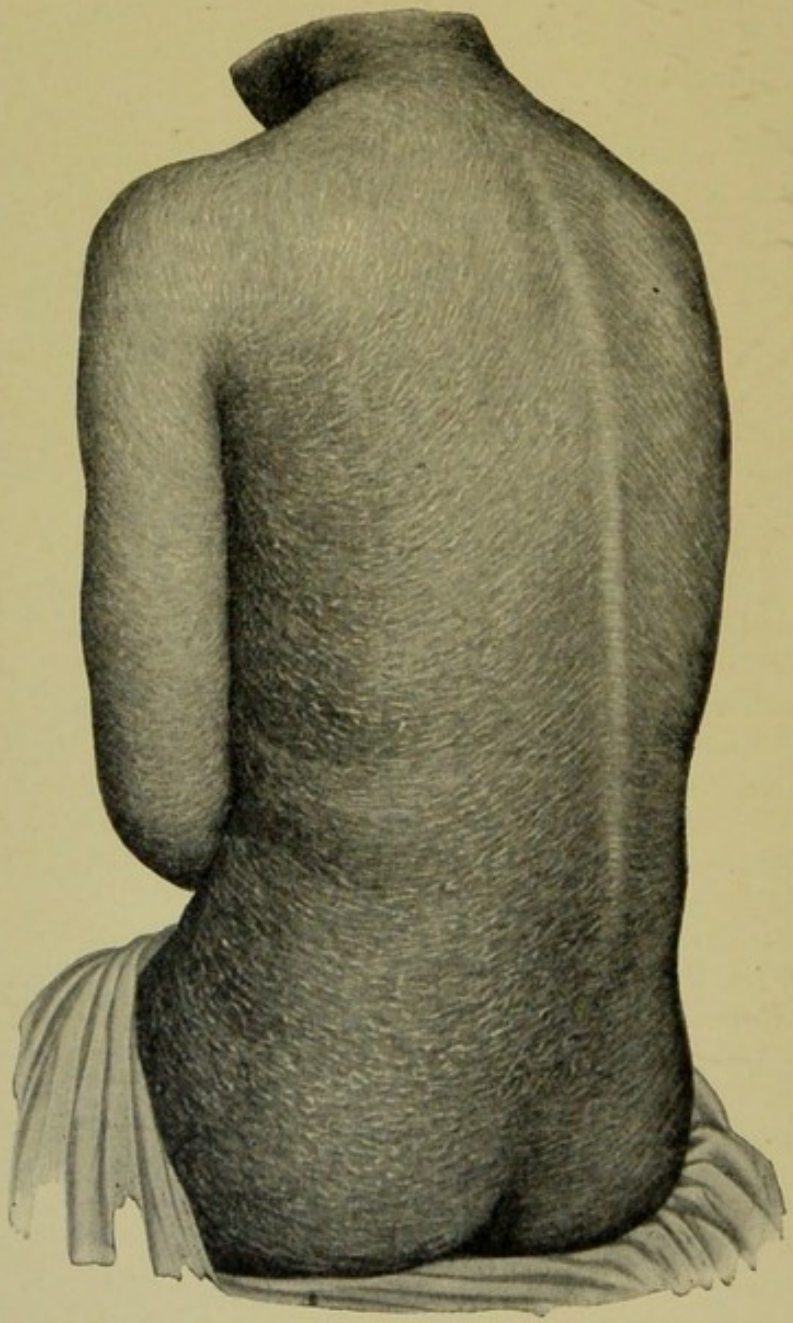


Fig. 73.—Ichthyosis cornea. (Mracek-Hudelo.)

Treatment.—The patient should take prolonged baths containing glycerine, using soft soap or pumice soap to remove the scales. Every night glycerole of starch should be applied for some time, with or without the addition of tartaric acid (4 or 5 per 100). Ichthyosis is incurable, but the ordinary form can be made almost invisible.

CORN.

This is a circumscribed thickening of the horny layer of the epidermis, presenting on its lower surface a small central core which is imbedded in the mucous layer, and which differentiates a corn from a callosity. Corns occur most frequently on the sides of the toes, especially on the outer side of the little toe, also on the sole of the foot under the heads of the first and second metatarsal bones; but they may occur over any bony projection of the foot. They are generally caused by the pressure of tight boots. The name "partridge-eye" has been given to corns situated between the toes, which have a depressed centre and raised border. Corns may become inflamed and even set up lymphangitis, which is sometimes dangerous in old people and diabetic subjects.

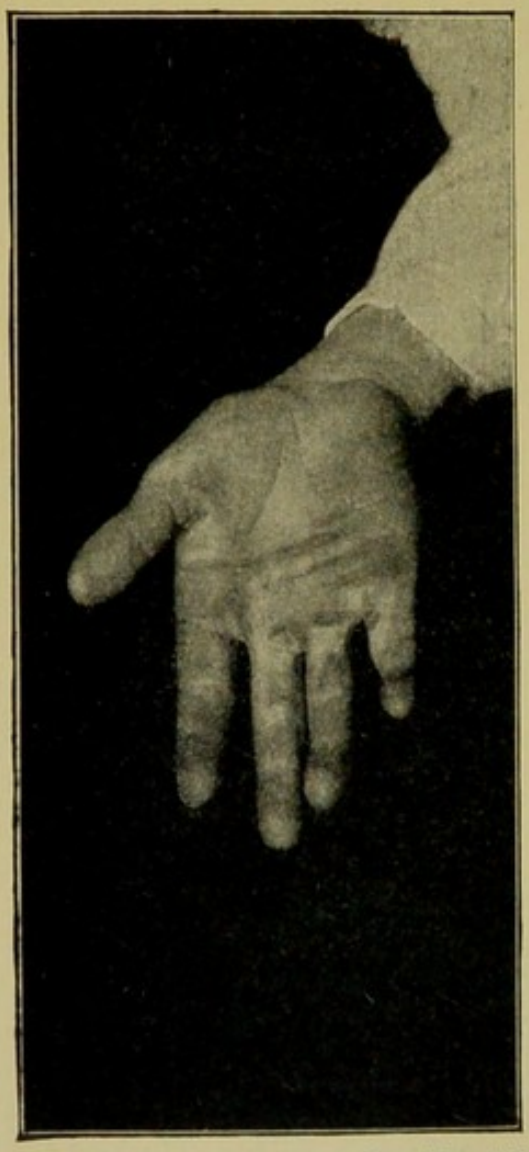
TREATMENT.—The corn should first be softened by poultices and then scraped. If this is not sufficient, salicylic acid collodion (10 per 100) may be applied every night for a week; after this, a poultice is applied, when the corn can be removed, sometimes with its root. Tincture of iodine may also be applied for several days; this softens the corn, which can then be removed by scraping.

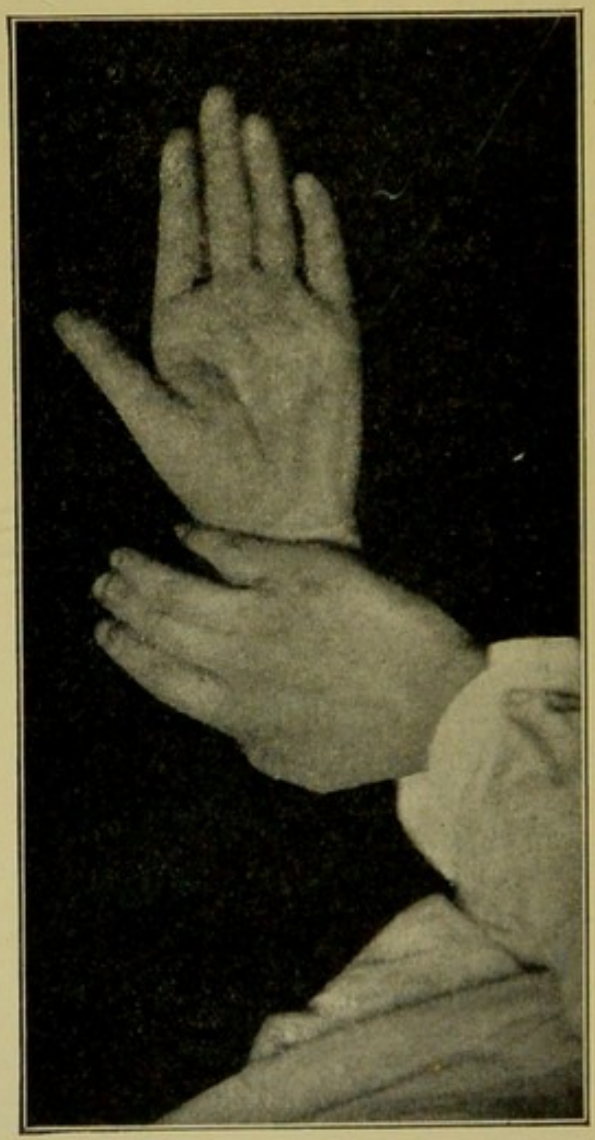
CALLOSITY.

The name callosity or *tylosis* is given to a more or less extensive thickening of the epidermis, which varies in colour from dirty white to dark yellow. This epidermic thickening is dry and hard, the normal lines of the skin are almost effaced, and cutaneous sensibility is diminished. When detached from the skin it is slightly translucent, apparently homogeneous, and thicker in the centre than at the edges. Microscopic examination shows that it is formed by layers of horny cells parallel to the surface.

ETIOLOGY.—Callosities are usually produced by prolonged pressure on the skin overlying a bony prominence. They often occur on the heels and soles after prolonged walking or standing, but their most common situation is on the hands, especially in manual workers. In this region, they vary in situation, form and extent, according to the tools employed. In carpenters, they occur on the thumb and index finger, from using the plane; in cobblers, on the palm and on the right thigh, from using the hammer; in hatters, on the thenar eminence, from rolling a cylinder with the hands; in violinists, on the tips of the fingers of the left hand, etc. In burnishers, multiple callosities occur, depending on the instruments they use. In metal-workers, callosities may be caused by mineral acids. Callosities are important from the medico-legal point of view.

Complications.—When callosities involve a considerable part of the palm, they may hinder movement. They may become complicated by painful fissures extending to the dermis. A serous bursa is often formed underneath the callosity, which may become inflamed





FIGS. 74 and 75.—Callosities in burnishers.

and form an abscess. When the cause which produced the callosity is removed, the lesion gradually disappears; this does not occur in essential keratosis.

TREATMENT. — Callosities may be destroyed by the careful application of a concentrated solution of caustic potash. After a few applications, an attempt is made to detach the callosity, after a hot bath. But the lesion recurs indefinitely as long as its cause persists.

ESSENTIAL KERATOSIS OF THE EXTREMITIES.

This affection (incorrectly called *keratodermia*, since it does not affect the dermis) is characterised by thickening of the epidermis of the palms of the hands and soles of the feet. *Secondary keratosis* may occur in the course of chronic eczema, psoriasis, pityriasis rubra pilaris, and dermatitis herpetiformis, when these dermatoses affect the hands and feet, and also in arsenical intoxication; but, apart from such cases, there is a *primary essential keratosis* of the palms and soles, which is the form we are now describing.

Varieties.—Besnier distinguishes four forms:—(1) A congenital and hereditary form, with or without nævi on other parts of the body. In one case the mother, two sisters, and three maternal uncles were also affected. (2) A form which develops in childhood (erythematous keratosis). (3) A form which develops in isolated spots on the palms and soles, sometimes at the orifices of the sweat glands, which are distended by horny cones formed by concentric layers. (4) An accidental form, which, in my opinion, is the same as callosity.

Symptomatology.—The lesions are usually symmetrical, affecting both hands or both feet, or all four extremities at once. Sometimes the whole palmar surface of the hand and fingers, or the whole plantar surface of the foot, is affected; sometimes only part of the surface, the centre of the palm, the heel, or the sole over the heads of the metatarsal bones. Sometimes there are islets of keratosis on the palmar surface of all the fingers over the heads of the metacarpal bones, and on the thenar and hypothenar eminences.

The epidermis varies in thickness in different regions; it is sometimes so thick as to hinder movement. Its colour is gray or dirty yellow. Sometimes there are painful fissures extending to the dermis. The subjacent dermis is almost normal, but sometimes presents hypertrophy of the papillæ and a certain degree of redness, which may extend beyond the keratosis and even reach the wrist. Between the islets of keratosis the skin is healthy. In some cases there is severe itching or burning sensations. When the foot is affected, walking is difficult, but not painful; when the hand is affected, movements are limited. Although the disease appears to be of nervous origin, there are no sensory disorders nor changes in the reflexes. The secretion of sweat persists. The nails are curved. The disease develops in crops, the lesions being more marked in winter, but permanent. According to Besnier, keratosis is aggravated by manual work; but, if patients are watched long enough, exacerbations are seen to occur apart from any work.

Pathological Anatomy.—In a patient of Besnier's, Balzer found

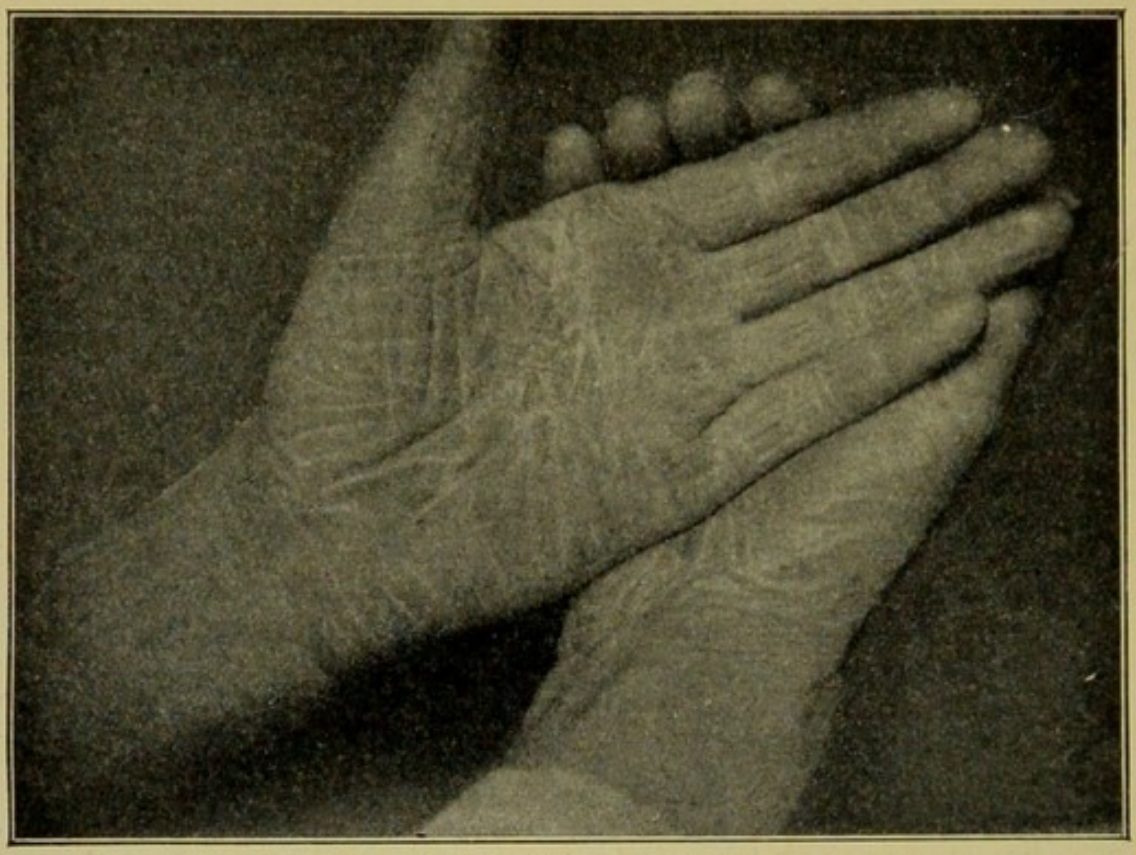


FIG. 76.—Symmetrical palmar keratosis.

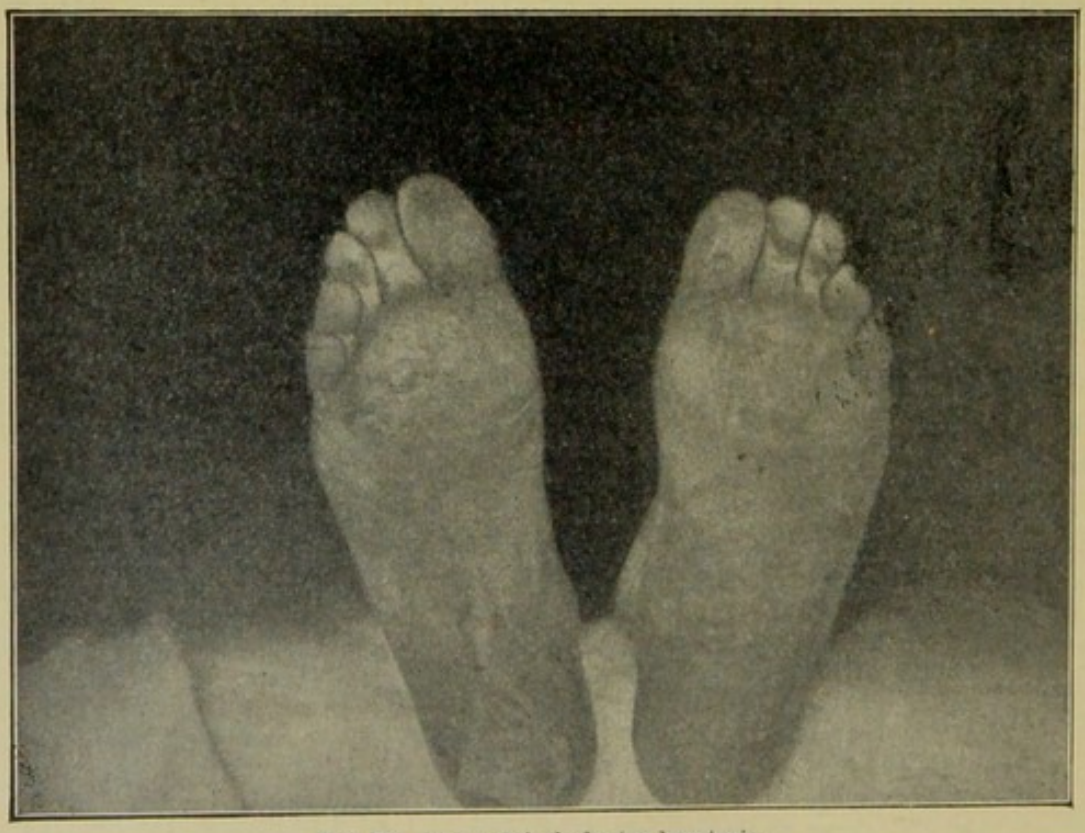


FIG. 77.—Symmetrical plantar keratosis.

that the thickened horny layer of the epidermis consisted of cells with ill-defined outlines and no nuclei, forming, in the upper layers, a refractile, homogeneous mass, easily stained by carmine. The ducts of the sweat glands were dilated, and surrounded by flattened cells.

ETIOLOGY.—This is obscure. The disease is sometimes hereditary and congenital. It occurs in persons of excitable nervous temperament, and this peculiarity, combined with the symmetrical distribution of the lesions, suggests that essential keratosis may be a tropho-neurotic lesion of central origin. The fact that it is sometimes congenital approximates it to nævi.

FIG. 78.—Plantar symmetrical keratosis. (St Louis Hospital Museum.)

DIAGNOSIS.—It is sometimes difficult to distinguish essential keratosis from *eczema*, *psoriasis*, *lichen simplex*, *lichen planus*, and *pityriasis rubra*, when one of these diseases affects the palms of the hands. These dermatoses can be recognised by the presence of characteristic elements on the hand, or on some other part of the body. The palmar or plantar syphilide, which sometimes simulates keratosis, is differentiated by the presence of round papular elements, of a coppery red colour, covered with squames, which are thinner than those of keratosis.

TREATMENT.—Keratosis continually recurs in spite of all treatment. Arsenic has been tried in large doses, but I have never seen any good results from it; moreover, such treatment is irrational, because arsenic itself may produce keratosis. Locally, the thickened epidermis may be softened by poultices and then scraped, after which the remains of the epidermis may be detached by means of soft soap plasters. After this, an ointment of chrysarobin or pyrogallic acid, or better, salicylic acid (5 per 100) may be applied. But, in spite of all treatment, the lesions always recur.

ARSENICAL KERATOSIS.

This occurs in occupational arsenical poisoning, while therapeutic arsenical intoxication usually gives rise to pigmentation.

Arsenical keratosis is observed in workmen employed in the manufacture of the arsenical green pigments used in the paper

industry. It is morphologically similar to the essential keratosis which we have just described, but the thickened epidermis is incrusted with green particles, arising from the dust of the arsenical salts. The lesions are symmetrical, and affect the whole of the plantar and palmar regions; but they are more pronounced on each side of the folds of flexion of the palm and fingers, and on the heel and anterior part of the plantar arch over the heads of the metatarsal bones.

The keratosis is associated with other cutaneous signs of arsenical intoxication; pigmentation, and ulcers between the fingers and toes, on the chin and nasal septum, and on the scrotum, where they resemble mucous patches.

The patients are also affected with *sclerodactylia;* the fingers being smooth and tapering, and their integuments thinned and fixed to the bones. The presence of sclerodactylia, which is manifestly a trophoneurosis, shows that all the cutaneous lesions are also of nervous origin. The keratosis itself is of nervous origin, and is not due to an external traumatic cause; it is simply increased by manual work.

This affection is curable, and disappears when the cause of the intoxication is removed.

EPIDERMIC AND PAPILLARY HYPERTROPHIES.

CUTANEOUS HORN.

This is a circumscribed growth of the horny layer of the epidermis, with hypertrophy of the subjacent papillæ, resembling in appearance the horns of animals. Horns of various sizes and shapes have been observed; cylindrical, conical, or rolled on themselves. They may be single or multiple, implanted in a cup-shaped depression or on a broad base. They have been seen on the scalp, eyelids, ears, nose, lips, glans penis and prepuce, trunk and limbs. They sometimes develop rapidly, persist for years, and then fall off, sometimes growing again.

They arise from hypertrophied papillæ, and are formed by columns of epidermic cells, united longitudinally, developed round the papules. Sometimes sebaceous cysts are found at their base. The base may become inflamed and ulcerate, and even undergo epitheliomatous transformation. The affection is sometimes hereditary.

TREATMENT.—This consists in extirpation of the horn with its base, followed by cauterisation with the thermo-cautery.

WARTS.

These are cutaneous outgrowths, round when single, polycyclic when several are united. They have a rough, papillomatous surface, and are usually sessile, but sometimes pedunculated. When large and agglomerated, they have a tuberous or cauliflower appearance. Their colour is yellowish gray, or blackish brown. They occur usually on the hands and face, but may be met with in any region, even on the sole of the foot. Warts may be persistent or transitory, disappearing spontaneously. They are seldom single, for they are auto-inoculable. They are generally painless, but may become fissured and inflamed, and then become painful.

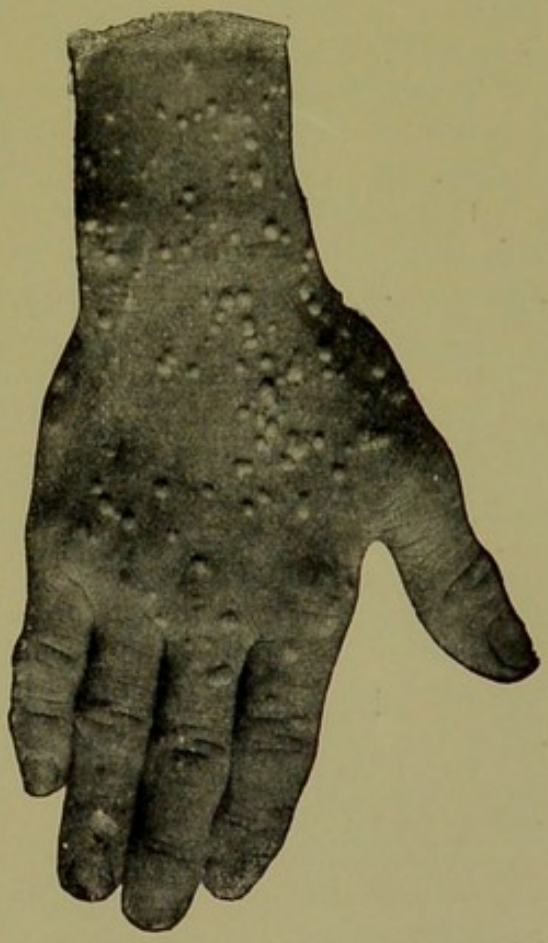


Fig. 79.—Warts. (Marwedel-Chevassu.)

A wart is a circumscribed hypertrophy of the papillary body and of the epidermis. In the centre of the papillary hypertrophy is a simple or ramified vascular loop, surrounded by embryonic cells and fine connective-tissue fibres; the Malpighian layer is hypertrophied, and covered with a thick horny layer. Majocchi discovered a bacterium in warts (*bacterium porri*), which was successfully cultivated and inoculated by Kühnemann. Variot inoculated one subject from another. Warts are therefore contagious and inoculable.

Apart from common warts, Besnier described a form of *flat juvenile wart*, which occurs on the face and on the back of the hands in young subjects. These warts are small, flat elevations, the size of a pin's head to a lentil, and usually very numerous. They develop by inoculation on excoriations caused by scratching, and disappear spontaneously in a few years. They must not be mistaken for *sebaceous adenomas*, when situated on the face.

Treatment.—Warts may be destroyed by caustics, such as nitrate of silver or nitric acid, or by the thermo-cautery or galvano-cautery. Calcined magnesia given in small doses for a long time,

sometimes causes warts to disappear. Flat juvenile warts may disappear after rubbing with salicylic acid or pyrogallic acid ointments, but it is simpler to destroy them with the galvano-cautery.

SIMPLE PAPILLOMA.

This is a circumscribed lesion, characterised by hypertrophy of the papillary layer of the dermis. It must be distinguished from secondary papillomatous lesions sometimes observed on chronic patches of eczema, lichen simplex, psoriasis, and in chronic pemphigus, sycosis, certain tertiary syphilides, and verrucose tuberculosis.

This affection occurs among coopers, rope-makers, basket-makers, petroleum refiners, those engaged in the washing of plates and dishes, and in lemonade-makers.

The papilloma is usually situated on the hands, at the ends of the fingers and thumb around the nail, which becomes detached; sometimes on the heel of the foot. It forms a patch with raised edges, irregular in size and shape. The surface is granular, rough to the touch, fissured, dry, and covered with horny tissue. The colour varies from gray to dark red. It is generally painless, but occasionally painful. It is slow and progressive in evolution, and sometimes ulcerates.

There is a special form of papilloma affecting the scalp, to which I have given the name of *penicilliform papilloma*. This form, which is fairly common, occurs as small, round, circumscribed, warty masses, covered with flexible projections, formed by elongated and hypertrophied papillæ. Papillomas have the same structure as warts, consisting of groups of papillary elevations compressed against each other.

Papillomas must be distinguished from *anatomical tubercle* and from the *verrucose tuberculide*.

TREATMENT.—Papillomas may be destroyed by scraping, under an anæsthetic, but more simply by the thermo-cautery or galvano-cautery.

GONORRHŒAL PAPILLOMA.

This special form of papilloma, described by Vidal and Chuffaard, is a rare manifestation of severe gonorrhœa accompanied by gonorrhœal rheumatism. It appears from three to five weeks after the onset of gonorrhœa.

Gonorrhœal papillomas are true papillomas, formed by groups of hypertrophied papillæ covered by hyperkeratosis and thick horny masses. They occur on the palms and soles, and on the pulp of the fingers and toes. In rare cases they have been observed on all parts of the body, even on the face and genital organs.

There are two forms of gonorrhœal papillomas, which are only two degrees of the same lesion, and are generally associated: (1) *isolated horns*, about half an inch in diameter and the same in height, of a yellow or brown colour; (2) *horny patches* of various sizes, resulting from the confluence of isolated horns; these may occupy the whole heel or the whole of the sole of the foot; they consist of a rough, horny thickening covered with hyperkeratosic projections, which reveal a red and villous dermis when they are removed.

These papillomas last for weeks or months and then disappear; but they may recur at each fresh attack of gonorrhœa. Their symmetrical distribution indicates that they are *trophic lesions*.

DERMIC HYPERTROPHIES.

SCLERODERMIA.

Sclerodermia is an affection characterised by induration of the skin, due to fibrous transformation of the dermis and subjacent tissues.

Varieties. — Clinically, there are two forms of sclerodermia, *diffuse* and *circumscribed*. With the diffuse form may be included the œdematous sclerodermia described by Hardy, regarded by Besnier and Doyen as a distinct affection, which they call *scleremia*. Circumscribed sclerodermia includes: (1) *sclerodactylia*, which may occur alone or with general sclerodermia; (2) *sclerodermia in bands*; (3) *morphœa*, or *sclerodermia in patches*.

Diffuse Sclerodermia.—This begins with sensations of numbness and tingling, cramps, and shooting pains in different parts of the body. It may be preceded by local asphyxia similar to Raynaud's disease. There is sometimes sensory disturbance and epidermic exfoliation, with vesicles or bullæ. The skin may be red at first; sometimes it presents telangiectases.

After a time, hard œdema of the integuments develops in the form of irregular patches of variable extent, or in linear bands, more or less raised, and sometimes crossing each other. The patches and bands soon coalesce to form diffuse sclerosis. The induration generally appears first on the upper limbs, on the face, or on the upper part of the trunk, and then extends to the rest of the body.

In the œdematous form, the lesion often extends with great rapidity; but in the ordinary diffuse form it takes months or years to spread symmetrically over the body. In a case observed by Nixon, the sclerodermia was limited to one-half of the body.

The patches gradually subside and the tissues become atrophied. The skin is then tense and glossy, and cannot be seized between the

fingers nor moved over the subjacent parts. The healthy parts are merged insensibly with the diseased parts. The colour of the skin is dirty white, sometimes dark brown, and pigmentary spots are observed, which may be mistaken for vitiligo or Addison's disease.

When it has arrived at this stage sclerodermia presents a characteristic aspect. The face has the appearance of a wax mask, the nose and lips are thin and drawn, the cheeks fixed to the bones, the eyelids immobile, and the ears rigid. This results in impediment to speech and mastication. The skin on the chest forms a cuirass, which causes much trouble in respiration. The skin has the same appearance on the lower limbs, which are often retracted; but the condition is more marked in the integuments of the upper limbs, especially on the fingers, where it constitutes *sclerodactylia*. The fingers are thin, rigid, bent at the terminal joint, and reduced in length by absorption of the greater part of the inguinal phalanx; the nails are altered; sometimes there is gangrene of one or more fingers, with ulceration, as in leprosy (mutilating sclerodermia).

FIG. 80.—Diffuse sclerodermia. (Audry.)

In this generalised form the lesions affect the hand, wrist and forearm, the movements of which become very limited. The mucous membrane of the cheeks and tongue may also be affected.

Sensation is usually normal on the sclerosed parts, occasionally a little diminished. There is a sensation of cold and constriction, sometimes deep-seated pain. Muscular changes are often added to the cutaneous lesions; extension of the sclerosis to the subjacent tissues producing muscular atrophy and contracture, and consequent deformity. The muscular changes may even extend to parts of the limbs unaffected by the cutaneous sclerosis. The osseous system is often affected in sclerodermia; we have already mentioned absorption of the terminal phalanx in sclerodactylia. Hallopeau reported a case of sclerodermia with atrophy of certain bones and multiple arthropathy.

The progress of the disease is variable; occasionally it may be rapid; but usually it is slow, sometimes very slow. It may remain stationary for years, and in exceptional cases may undergo spontaneous cure; but, as a rule, it has a fatal termination. The general nutrition becomes more and more affected, the appetite fails, and there is mental depression and insomnia; sloughing occurs, and the patient dies from cachexia or from some complication, such as pneumonia, tuberculosis, or extension of the sclerosis to the viscera (myocarditis, pericarditis, interstitial nephritis, etc.).

Circumscribed Sclerodermia — Sclerodactylia. — We have already mentioned that sclerodactylia often occurs in diffuse sclerodermia; sometimes the lesion remains limited to the upper extremities during its whole evolution. Sclerodactylia presents the same characters in both cases.

Both hands are always affected at the same time. The flexion of the fingers is due to muscular contractures. Thickening of the integuments, pseudo-ankylosis, and arthropathy play only a secondary part in the deviation of the fingers, for these lesions occur late; muscular contracture, on the contrary, appears very early.

Sclerodermia in Bands.—In this form the sclerosis occurs in the form of bands situated along the limbs and around the trunk, following the direction of the peripheral nerves, as in zona. Besnier observed a case of sclerodermia extending from the shoulder to the dorsal surface of the three first fingers of the left upper limb. Kaposi reported a case of sclerosis distributed along the course of the right frontal nerve; another corresponding to the cervical plexus and two first dorsal nerves; and a third in which the sclerosis followed the course of the right saphenous nerve. I have published a case of sclerodermia occurring in patches along the course of the internal cutaneous nerve of the right arm. Rosenthal observed a case of partial sclerodermia with facial hemiatrophy and alopecia. Brault saw a case limited to the forehead and scalp, with partial alopecia. Sometimes the sclerosis is annular and causes constriction of a limb or a finger, as in *ainhum*. The prognosis of sclerosis in bands is graver than in morphœa.

Morphœa, or Sclerodermia in Patches.—This form is possibly a distinct disease, and differs, nosologically, from diffuse sclerodermia. It begins by a pale red spot, which gradually extends and soon becomes deep red; then, after a few months, the spot becomes white in the centre, while the periphery assumes a characteristic lilac colour (lilac ring). The spot is round or oval, of variable size, with a lardaceous appearance; sometimes it is raised above the skin. When completely developed, the colour is pearly white, sometimes slightly pigmented in the centre; it is surrounded by a lilac-coloured ring, and an outer ring of brown or bistre colour.

Morphœa may be single or multiple, unilateral or bilateral and symmetrical. Its chief situations are the forehead, lower part of the neck, chest, breasts, abdomen and thighs. At the onset there are sometimes tingling sensations. The hairs fall off, cutaneous secretion is suppressed, and sensation is often diminished; but there is neither pain nor itching.

After a time the patch becomes vascularised afresh, the epidermis desquamates, and the dermis becomes thin and atrophied; the vessels mostly disappear, and the lilac ring gradually extends; sometimes superficial ulcers form. After several years the patch finally disappears and usually leaves no trace; but in some cases a smooth, yellow or grayish violet, depressed cicatrix is left, destitute of cutaneous secretion.

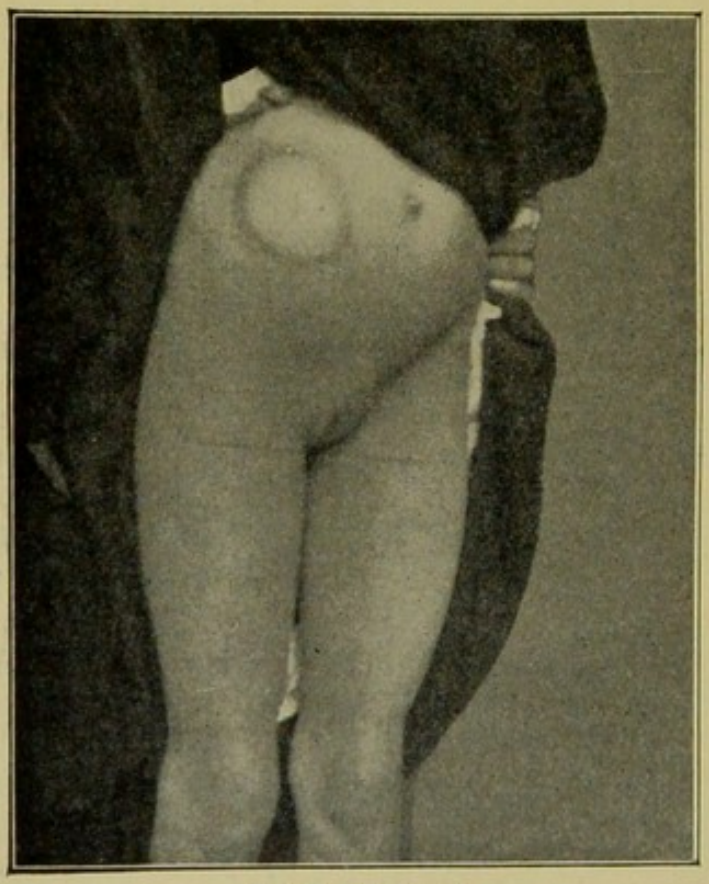


Fig. 81.—Morphœa.

In this group of circumscribed sclerosis there are no visceral lesions.

Diagnosis. — *Diffuse sclerodermia* can hardly be mistaken for any other affection. *Leprosy* differs in the presence of tubercles and disorders of sensation.

Sclerodactylia may at first simulate Raynaud's disease, leprosy, syringomyelia (Morvan's type), and chronic rheumatism. In *Raynaud's disease*, the lowering of temperature is more marked, circulatory disorder is more pronounced, the nose and ears are often affected, the integuments are not so indurated nor so adherent to the bones as in sclerodactylia, and there is no flexion of the last phalangeal joint. When the fingers are destroyed, it is by gangrene, and not by atrophy of the phalanges, as in sclerodactylia. There is, nevertheless, a rather close relationship between Raynaud's disease and sclerodactylia. In *tropho-neurotic leprosy*, the skin of the fingers is not sclerosed, pigmentary spots occur on various parts of the body, and there are cutaneous sensory disorders. In *syringomyelia*, there is

dissociation of sensation and muscular atrophy. However, it must be mentioned that Zambaco-Pacha regards sclerodermia as a hereditary, attenuated form of leprosy, and that he identifies Morvan's type of syringomyelia with leprosy. *Rheumatoid arthritis* is characterised by articular deformity of the fingers, but not by flexion of the last phalanx; the skin is not adherent to the bones, as in sclerodactylia.

Morphœa may, on cursory examination, be mistaken for *cheloid*, or for the *cicatrix* of a burn; but such errors are easily avoided. Besnier and Doyen mention a case of morphœa of the breast which was mistaken for cancer, and operated upon. In *vitiligo*, there is simply pigmentary change, without induration of the skin. The decolorised patches of *leprosy* differ from morphœa in being anæsthetic, and in the coexistence of other cutaneous leprous lesions.

Etiology.—Sclerodermia occurs most often between the ages of twenty and forty; but cases have been observed in old people and in children, six and even two years of age. Three-quarters of the cases observed occurred in women. The etiology is not well known; sclerodermia has been seen to occur after exposure to cold, after rheumatism, or after an injury; the last cause applies chiefly to circumscribed sclerodermia. In support of the arthritic nature of sclerodactylia, I have seen, in one family, the mother and daughter affected with sclerodactylia; the mother being at the same time affected with rheumatic arthritis. In another case, a patient with chronic emphysema was affected with sclerodactylia and chronic urticaria. Some authors have recently attributed diffuse sclerodermia to an internal secretion, due to changes in the thyroid or suprarenal glands (Touchard).

Pathological Anatomy.—The lesions in the skin consist in hyperplasia of the fibrous and elastic connective tissue, and subsequent contraction of this hyperplasia. The papillæ are atrophied, and in some places have disappeared; the glands and hair are atrophied; the trabeculæ of the adipose tissue and the subjacent muscles are sclerosed. In sclerodactylia, the sclerosis extends to the periosteum, and there is rarefying ostitis of the phalanges.

The cutaneous arterioles present interesting changes; the middle coat is double the normal thickness; the lumen is diminished, or even obliterated; the walls are infiltrated with embryonic cells, which may become transformed into fibro-plastic cells. There is both periarteritis and endarteritis. In fact, the sclerous process appears to commence round the cutaneous vessels, and then spreads to the surrounding tissues.

In some cases, sclerosis and degeneration of the cutaneous nerves has been found. We have already seen that the process of sclerosis is not limited to the skin, but extends to the muscles and viscera

(heart and kidney), which undergo sclerous degeneration, commencing round the arterioles.

It has been attempted to explain sclerodermia by a lesion of the central nervous system. In certain affections of the spinal cord (lateral sclerosis, tabes, and infantile paralysis), patches of sclerodermia have been found. Schultz observed degeneration in the anterior spinal nerve roots and large nerve trunks, in a case of sclerodermia with general muscular atrophy. Jacquet described small cavities in the gray matter of the spinal cord, chiefly in the lower cervical region, in a case of general sclerodermia; also granular pigmentary atrophy in the cells of Clarke's column. But the existence of these spinal lesions requires confirmation.

The initial lesions of morphœa have been well described by Radcliffe Crocker; they consist in small-celled infiltration around the blood-vessels, lymphatics, glands and pilo-sebaceous follicles, followed by the formation of connective tissue, which obliterates the vessels and glands, and then contracts to form the cicatricial patch.

PATHOGENY.—The observations in which nervous lesions were found at the autopsy are too inconclusive to prove definitely that the nervous system is concerned in the pathogeny of sclerodermia. However, the neuropathic state of many patients, and the relation of circumscribed patches of sclerodermia to the distribution of the nerves, are facts which favour a nervous origin. It is possible that the arterial lesions are the result of nerve changes, and that the latter are due to *arthritism*. Other authors consider that the arterial lesions precede the nervous disorder, and that sclerodermia is connected with the vascular areas, which, moreover, for the most part correspond to the areas of nerve distribution. We have already mentioned Zambaco's idea that sclerodermia is a degenerated form of leprosy; this requires proof.

TREATMENT.—In *diffuse sclerodermia*, hydrotherapy and spinal douches are indicated to brace up the nervous system. Tonics, such as arsenic, quinine and cod-liver oil, may also be given, and iodide of potassium for the arterio-sclerosis. Local treatment consists in massage and electricity; the latter in the form of the continuous current (the positive pole being applied to the spine and the negative pole to the affected patch), or electric baths.

Electric or hydro-faradic baths are used in my practice at the St Louis Hospital as follows: an enamelled bath is filled with saline water (10 grammes of sodium chloride to a litre); into this are plunged the two poles of an induction coil, the positive pole at the head of the bath, the negative pole at the foot. The patient remains in the bath for about half an hour every day.

Sulphur baths, vapour baths, and jaborandi sometimes cause

improvement. Lastly, the skin may be softened by oily liniments. But all these forms of treatment are very unsatisfactory.

In *circumscribed scleroderma*, Brocq obtained good results by electrolysis, practised in the following manner: one sitting is given twice a week, so as to act on each part of the patch once every two weeks; a current of 8 to 15 milliamperes is passed for twenty seconds at each puncture; the needle is attached to the negative pole, the positive pole being placed in the patient's hand (I think it preferable to connect the needle with the positive pole); the needle is introduced obliquely into the skin without piercing it, to avoid irritation of the subcutaneous tissue. Between the sittings, mercurial plaster may be applied to the patch, except when it is inflamed, when starch poultices may be used.

ELEPHANTIASIS.

The name *elephantiasis* is given to hypertrophy of the dermis and subcutaneous tissue, limited to certain parts of the body and secondary to repeated inflammation of the veins or lymphatic vessels.

Clinically, elephantiasis is characterised by increase in size and deformity of the parts affected, with thickening and induration of the skin.

True elephantiasis, which is sometimes called *elephantiasis Arabum*, must not be confounded with *elephantiasis Græcorum*, which is one of the numerous synonyms of leprosy; this term should be abolished, as it only leads to confusion.

Etiology.—Elephantiasis is met with in all countries, but is more common in tropical regions; Egypt, Arabia, the west coast of Africa, Brazil, the Antilles, the South Sea islands, Barbados, etc. In these countries, where it is endemic, elephantiasis acquires a greater degree of intensity than in temperate climates. It occurs at all ages, even in infants, but most frequently between the ages of twenty-five and fifty. With regard to *congenital elephantiasis*, this appears to exist, but some of the cases reported are doubtful, and may possibly have been *mollusciform nævi*. The disease is more common in women than men, and in the poorer classes; Creoles appear to be especially liable to it.

The predisposing causes are: damp, bad hygiene, malaria and obesity. The lesions most often affect the lower limbs and scrotum, because the circulation is more easily obstructed in these parts.

Pathogeny. — Elephantiasis may be the result of all kinds of inflammation of the dermis and subcutaneous tissue. It follows repeated attacks of cutaneous lymphangitis or erysipelas, developing around patches of eczema, lupus, syphilis, and varicose or other

kinds of ulcer. It may occur after obliterating phlebitis. In fact, it is a deuteropathic affection secondary to all venous or lymphatic lesions which cause obstruction to the return circulation of blood or lymph. In this way, elephantiasis of the scrotum may appear after bilateral excision of the glands of the groin. The vaso-motor disturbance which causes neuropathic œdema may also eventually lead to elephantiasis. To sum up, elephantiasis is only an exaggeration of lymphatic or venous œdema; the common ending of all cutaneous circulatory disorders. But most often it is due to obstruction of the lymphatic circulation, and in the majority of cases the lesions are produced either by an animal parasite or by a microbe. The animal parasite is the *filaria sanguinis hominis*, discovered by Wucherer in 1866, in the urine of a patient affected with chyluria; the microbe is the *streptococcus of erysipelas.*

In 1875 Patrick Manson and Lewis discovered the embryonic filaria in the blood of patients affected with elephantiasis, and the adult filaria in the lymphatic glands. These observers showed that the adult filaria produced elephantiasis by causing repeated inflammation of the lymphatic vessels and glands. According to Manson, the filaria gains entry into the human body through the agency of the mosquito, in the following manner: the embryonic filaria, present in the blood, is absorbed by the mosquito when it bites patients affected with elephantiasis; in the body of the mosquito the embryonic filaria develops into the larval state; the mosquito dies, the larval filaria becomes free, and lives in the water of marshes and swamps. Infection then takes place by drinking the water.

But this mode of infection is not accepted by Sommerville; neither is the filarial nature of tropical elephantiasis admitted by all. Some authors regard the presence of the filaria as only a coincidence favoured by pre-existing elephantiasis; but this view does not appear to me to be correct. It is necessary to state, however, that the filaria is not always present in tropical elephantiasis, while in some cases the streptococcus of erysipelas has been found.

Such is the pathogeny of *tropical elephantiasis,* which may be called true or primary elephantiasis.

With regard to *elephantiasis nostras*, this appears to be generally due to streptococcal infection. Richardière observed a case of elephantiasis nostras in a patient who had had three attacks of erysipelas of the face, and he suggests that the two cutaneous inflammations may be due to the same micro-organism, the *streptococcus erysipelatis.* This view is supported by the observations of Sabouraud, who obtained pure cultures of the streptococcus of erysipelas from blood or serum obtained by superficial scarification of parts affected with elephantiasis.

To sum up, the filaria and the streptococcus appear to be the two usual pathogenic agents of elephantiasis, whether it is the endemic elephantiasis occurring in tropical regions, or the elephantiasis nostras of temperate climates.

Fig. 82.—Filaria embryos and blood corpuscles. (Lancereaux.)

Symptomatology.—There is no symptomatic difference between tropical elephantiasis and elephantiasis nostras, except that the lesions are more pronounced in the tropical form.

Elephantiasis usually begins by attacks of lymphangitis in one or both of the lower limbs. The skin becomes red, smooth, tense, painful, and somewhat swollen. The lymphatic vessels of the affected region are indurated, and appear as red lines leading to the corresponding glands, which are enlarged and painful.

These attacks generally commence with a rigor, followed by fever, vomiting, and sometimes delirium. The attack lasts for several days, or at most one or two weeks, after which the general symptoms disappear and the local condition gradually improves; sometimes an abscess forms. But the important point is, that the skin and subcutaneous tissue always remain infiltrated. After a time another

attack occurs, similar to the first, but with less intense local symptoms; this leaves the integuments still more infiltrated. Finally, after several similar attacks, the condition of elephantiasis is produced.

The skin is at first smooth, tense and dry, sometimes redder than normal; after a time it becomes yellow or brown, and covered with epidermic and sebaceous debris. Eventually, papillary hypertrophies develop (verrucose elephantiasis), forming red nodosities, with fissures, from which a yellow liquid exudes, sometimes purulent and fœtid. Chronic ulcers occur, either spontaneously or after slight injuries, and become the point of origin of fresh attacks of lymphangitis and hypertrophic dermatitis. The skin is thick and hard to the touch, and fixed to the subjacent tissues, so that it cannot be pinched between the fingers; the bony eminences are more or less effaced.

DISTRIBUTION.—Elephantiasis may occur in any part of the body, but it most frequently affects the lower limbs (when both are affected, one is more so than the other), the scrotum and penis, and the labia and breasts in women; sometimes on the upper limbs, face, neck and ear.

On the lower limbs, the foot and lower two-thirds of the leg are the parts most affected; there is often, at the same time, œdema and induration of the lymphatics of the thigh; the lesion may spread to the abdomen. The lymphatic glands of the groin and popliteal space are enlarged. In tropical elephantiasis the limb is completely deformed; it becomes enlarged at its lower end, so as to resemble an elephant's foot; the leg presents bosses separated by grooves. Movement is difficult, owing to the weight of the limb, and sometimes also on account of secondary muscular atrophy. Here and there, vesicles are present, which rupture and discharge a coagulable serous fluid, which is really lymph from the lymphatic varices.

The scrotum and skin of the penis may become so enlarged as to extend to the middle of the thigh, or even to the knee (a scrotum has been seen weighing over 120 lbs.), the preputial orifice being represented by a vertical groove. In women, the labia and sheath of the clitoris are sometimes affected.

The upper limb sometimes becomes elephantiasic, as the result of lupus or syphilitic lesions.

The upper lip and lower eyelids become hypertrophied in lymphatic subjects, after repeated attacks of eczema or coryza. The forehead and cheeks sometimes remain infiltrated after recurrent erysipelas. Sessile or pedunculated appendages sometimes form on the ears.

PROGNOSIS.—In the early stages the disease may be attenuated by prolonged treatment. In the later stages it is rebellious to treatment, but is only dangerous to life in the rare cases which are

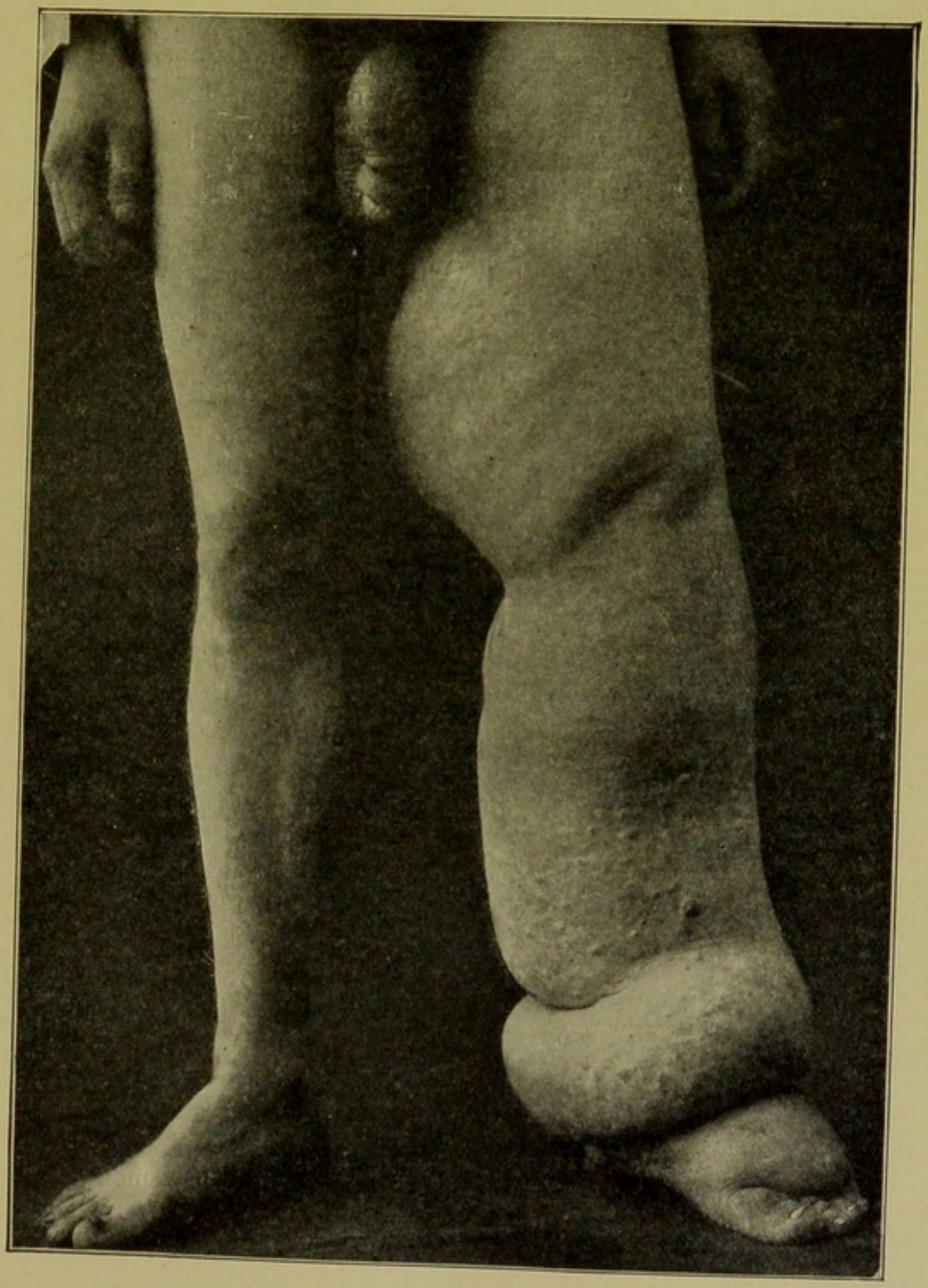


Fig. 88.—Elephantiasis.

complicated by deep phlegmonous inflammation, phlebitis, or gangrene.

Pathological Anatomy.—When elephantiasic tissues are incised, they creak under the knife, and the whole of the subcutaneous parts, down to the bone, are seen to consist of an almost homogeneous, yellowish white, fibrous mass, in which the muscles, vessels, and nerves are distinguished with difficulty. A slightly turbid, alkaline liquid escapes, which coagulates on exposure to air. The dermis is hypertrophied, and the subcutaneous tissue is several times its normal thickness; some parts are hard, others soft. The aponeuroses, intermuscular connective tissue, and sheaths of the vessels and nerves are thickened. The bones appear enlarged, or are covered with osteophytes.

Histological examination shows that the lesion originates in the lymphatic network of the dermis, the vessels of which are inflamed and surrounded with proliferating connective-tissue elements. The walls of the small lymphatic vessels are thickened; the connective-tissue fibres of the dermis are increased in size and bathed with lymph, and the subcutaneous and intermuscular connective tissue, and even the periosteum, are infiltrated by the lymphatic œdema.

Diagnosis.—This is easy. Elephantiasis cannot be mistaken for *myxœdema*, which affects the whole surface of the body; nor for *chronic œdema*, in which the skin and subcutaneous tissue are only infiltrated with serous liquid and not hypertrophied, and are not subject to repeated attacks of lymphangitis. It must be distinguished from *rheumatic* and *neuropathic œdema*, which is sometimes so intense as to simulate elephantiasis (neuropathic pseudo-elephantiasis); but it must be borne in mind prolonged neuropathic œdema may end in an elephantiasic condition.

When filariosis is suspected, the blood should be examined at night, as the larvæ are only present in the blood at this time.

Treatment.—This includes prophylaxis and curative treatment. In countries where the disease is endemic, all causes which may produce lymphangitis, exposure to cold and traumatism of any kind, should be avoided. All wounds should be treated with strict asepsis. In regions where filariosis exists, water should be boiled before drinking, and bathing in marshy places should be avoided.

When the attacks of lymphangitis occur, the treatment consists in rest in bed with elevation of the limb, hot boracic fomentations, tepid baths, and sulphate of quinine internally.

When the stage of elephantiasis has become established, attempts should be made to reduce the infiltration by means of hot sulphur douches, massage and compression. Compression is applied after the lymphangitis and any ulcers present have been cured; the limb is enveloped in wool kept in place by a bandage; over this an elastic

bandage is applied from the foot upwards, taking care not to apply it too tightly, to avoid gangrene. The elastic band is reapplied every morning, the effect being carefully watched.

Ligation or compression of the femoral artery have been tried without success, and such measures are not to be recommended. Silva Araujo and Moncorvo of Brazil recommend electrolysis, the positive pole being used in the diseased parts; continuous currents soften the indurated tissues, and intermittent currents promote absorption of the œdema; but this method requires much care. Lastly, in elephantiasis of the scrotum or labia, good results may be obtained by partial excision.

CUTANEOUS ATROPHY.

This may be local or general. The type of general atrophy is *senile atrophy*, the characters of which are known to all.

Local cutaneous atrophy includes several varieties: (1) idiopathic local atrophy; (2) local atrophy due to a central or peripheral nerve lesion; (3) atrophy observed in *xerodermia pigmentosum*.

Idiopathic Local Atrophy.—This is a rare affection, usually confined to limited parts of the skin, in the form of round white patches, varying in size from a two-shilling to a five-shilling piece, or oval patches which may be about two inches in length. These patches vary in number, and occur in subjects of apparently good health. The affected skin is thin and flexible, and has sometimes a bluish gray colour, due to the subjacent vessels seen through it. In some cases larger areas are affected; the thighs and knees, or even the whole body, except the buttocks.

Symptomatic Local Atrophy.—This may occur in various pathological conditions. It has been observed in infantile paralysis. Vidal reported a case which coincided with weakness of the right arm and leg, following a febrile disease. In this group may be included the disease named *facial trophoneurosis* by Romberg, and *progressive facial hemiatrophy* by Grasset, which belongs to the domain of neuropathology.

We do not include *vergetures* in this group, nor the cicatricial conditions of the skin left after syphilides, favus and chronic eczema.

XERODERMIA PIGMENTOSUM.

This is a diffuse atrophy of the skin accompanied by pigmentary spots, which, after a time, usually become complicated by epitheliomatous growths.

It is unnecessary to enumerate the different names which have been given to this dermatosis; that of *xerodermia pigmentosum*, given to it by Kaposi, who first described this affection in 1870, appears to be sufficient. Since this date about sixty cases of this peculiar affection have been published.

The disease begins at an early age, sometimes in the first year of life, in the form of red patches of variable extent, situated on the

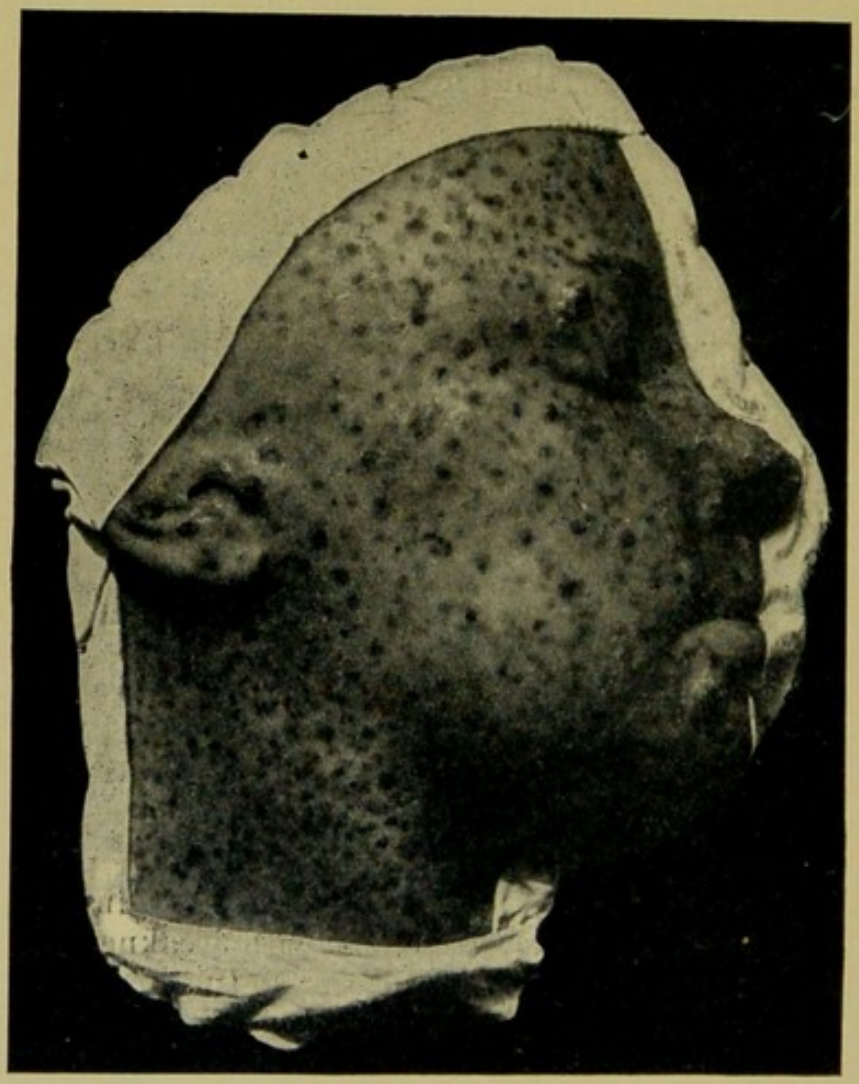


Fig. 84.—Xerodermia pigmentosum. (St Louis Hospital Museum.)

face, ears, neck, nape, shoulders, chest, arms and back of the hands; in fact, on the parts of the body most often uncovered; in people who walk barefoot, it may affect the feet and legs. The red patches, after they have disappeared, leave pigmented spots similar to lentigo; these may also appear from the first. Soon afterwards, vascular dilatations appear between the spots; these telangiectases gradually disappear, and are followed by *diffuse atrophy of the skin*. The epidermis becomes thin, dry and fissured, and sometimes raised

in places in the form of thin lamellæ. The subjacent dermis also loses its vitality and presents the same characters as in senile atrophy, becoming stiff and retracted over the subjacent tissues. Here and there are superficial depressions similar to cicatrices; these are the remains of the vascular dilatations. Secondary cutaneous lesions may also develop; vesicles, pustules, fissures and superficial ulcerations.

The retraction of the skin soon produces deformities. The mouth and nostrils become constricted, and there is ectropion of the lower eyelids, which may be followed by lesions of the cornea. Sometimes blepharitis develops, with loss of the eyelashes and destruction of the follicles. The skin of the rest of the body is normal.

Xerodermia pigmentosum never undergoes resolution. In many cases epithelioma develops on one or more parts of the face; Vidal reported a case in which there were eighteen epitheliomas. The epithelioma arises on a pigmented spot, first of all as a warty growth. It may ulcerate and become spontaneously detached and then heal or recur in the same or some other place. Pick and Elsenberg have described cases in which sarcomas developed instead of epitheliomas. In any case, whatever the form of tumour observed, visceral metastasis is rare.

Pathological Anatomy.—The pathological process appears to commence with proliferation of the connective tissue of the papillæ and of the endothelium of the vessels, followed by atrophy of the papillæ. At certain points there is a formation of new vessels (telangiectases). Later on, the interpapillary epidermic prolongations assume exaggerated development and undergo epitheliomatous transformation. Sebrazes described coccidial bodies in the epithelioma of xerodermia pigmentosum, but these were probably products of cell degeneration, like those observed in Paget's disease of the nipple.

Etiology.—This is unknown; but the solar rays may be an exciting cause in predisposed subjects. The disease generally occurs in young people between the ages of three and twenty-two; but Schwimmer saw a case commence at thirty-five. It has often been observed in brothers and sisters, and the influence of consanguinity is manifest. Barré remarks on the frequency of cancerous heredity in the parents.

Prognosis.—This is unfavourable, because of the frequent termination in epithelioma; but this complication does not occur in all cases. Riehl observed a case in which a woman aged sixty-one, who suffered from xerodermia since infancy, was free from epithelioma.

Diagnosis.—Xerodermia pigmentosum is easily distinguished

from *lentigo* and *pigmentary nævi*. In *sclerodermia*, the patches of sclerosis are more definite and there are no lentiginous spots. *Macular leprosy* is accompanied by anæsthesia, and has a different evolution.

TREATMENT.—Exposure to the sun's rays should be avoided. The skin should be kept as aseptic as possible. Epitheliomatous complications require the usual treatment for epithelioma.

NEOPLASTIC DERMATOSES.

XANTHELASMA. XANTHOMA.

THIS dermatosis is constituted by yellow patches or nodosities, which are usually situated about the eyelids, but may be disseminated over the body. The name of *xanthelasma* is applied more especially to the form which occurs in patches; that of *xanthoma* to the nodosities. Xanthelasma is occasionally met with on mucous and serous membranes.

SYMPTOMATOLOGY.—**Xanthelasma, or Xanthoma Planum,** consists of small patches, generally situated symmetrically on the eyelids, in the direction of their folds, most commonly towards the inner canthus; more rarely on the cheeks, nose, ear, neck and nape. They are often observed on the genital organs. The patches are of a chamois yellow or sulphur colour, smooth and soft to the touch, and have no raised borders like flat pigmentary nævi.

Similar lesions may occur on the mucous membrane of the gums, cheeks and soft palate. Wickham Legg and Chambard have seen patches of xanthelasma on the laryngeal and bronchial mucous membrane; Hilton Fagge, on the peritoneum, endocardium, and internal tunic of the aorta and pulmonary artery; Virchow and myself, on the cornea; Pye-Smith, on the œsophageal mucous membrane under the capsules of the liver and spleen, and in the biliary canals.

Xanthoma Tuberosum occurs in the form of tubercles or nodosities of a yellowish white colour, isolated or confluent, in the form of bands or raised patches. The nodosities have the size of a pea to that of a walnut; they are white at the apex, and often red at the base. They are rare on the face, and are usually met with on the joints of the fingers and toes, on the elbows and knees, on the palms and soles, on the trunk, in the folds of flexion of the skin, and sometimes on the scalp and penis.

They occur especially on regions which are subject to frequent friction and irritation. I have seen a case of chronic icterus which presented nodosities of xanthoma on the cicatrices left by wet

cupping for pneumonia. In this case the former traumatism of the skin determined the localisation of the xanthoma.

Xanthoma may also occur in the buccal cavity, pharynx, trachea and bronchi, and on the labia and vaginal mucous membrane.

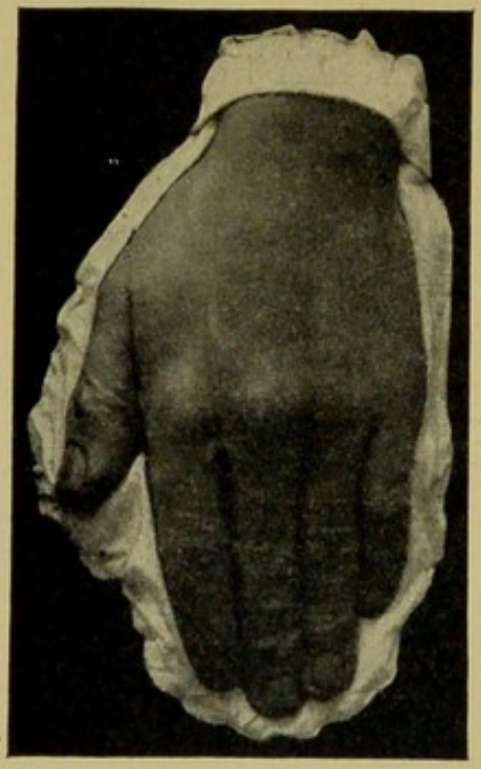


FIG. 85.—Xanthoma on the phalangeal joints. (St Louis Hospital Museum.)

FIG. 86.—Xanthoma on the elbow. (St Louis Hospital Museum.)

Xanthelasma and xanthoma sometimes coexist in the same subject, so that these two forms must not be regarded as distinct lesions. Xanthelasma is neither painful nor pruriginous; xanthoma is slightly painful on pressure.

In half the cases the affection is preceded or accompanied by hypertrophy of the liver and jaundice, sometimes by attacks of biliary colic. However, hypertrophy of the liver does not seem to be the cause of xanthoma, as was formerly believed. Besnier and Torok suggested that it was due to the formation of adipo-xanthomatous tissue in the liver. Pye-Smith found white patches on the surface of the liver, cream-coloured foci in its interior, and similar spots on the capsule of the spleen. Moxon found dilatation of the biliary passages, which were studded with xanthomatous spots, and constriction of the hepatic duct by fibrous tissue. "Unfortunately in most autopsies, a diagnosis of hypertrophic cirrhosis has been

made without a proper histological examination. We know that obstruction of the biliary passages produces hyperplasia of the intraglandular connective tissue; therefore, xanthomatous changes in the walls of the biliary passages may, on account of the obstruction they cause, lead to secondary hypertrophic cirrhosis of the liver. . . . We know that, even in the skin, the formation of fibrous tissue may preponderate over the production of xanthelasmic cells. The question of xanthofibromatosis of the liver is therefore obvious, and it is to this point that future researches should be directed. . . . But it does not follow that every change observed in the liver in the course of xanthoma is due to such a process; in a case of Murchison's, an alcoholic with xanthelasma of the eyelids, typical hypertrophic cirrhosis was found; and in a case of Wickham Legg's, there were hydatid cysts compressing the hepatic duct" (Torok).

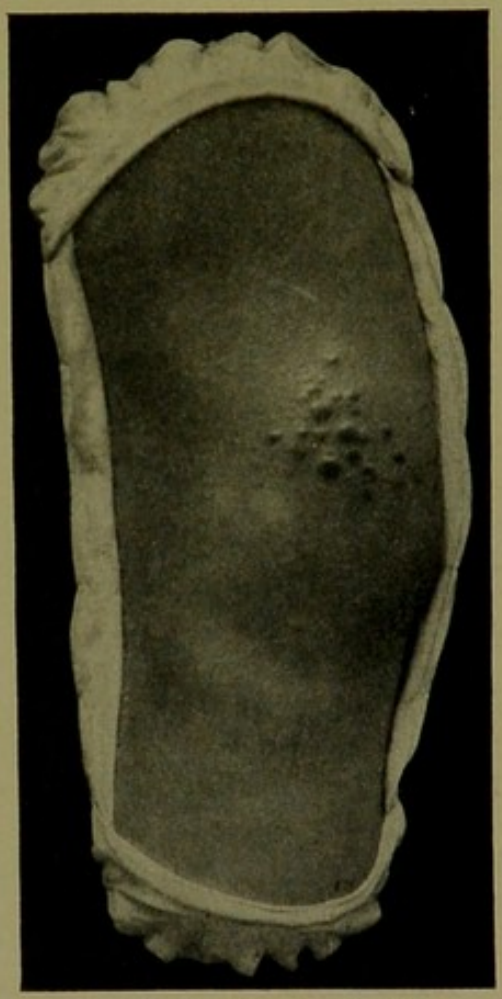


Fig. 87.—Xanthoma on the knee. (St Louis Hospital Museum.)

To sum up, the relation of xanthelasma to hypertrophy of the liver, with jaundice, requires futher investigation. Besides the icteric coloration, the skin may have a special yellow colour (*xanthochromia*); the urine does not then give the reaction for biliverdin.

Xanthoma usually pursues a slow and indefinite course, but in exceptional cases it may develop rapidly. It usually persists during life, but in rare cases disappears spontaneously. Xanthelasma of the eyelids is an isolated lesion of benign prognosis. If xanthelasma becomes generalised, if it is accompanied by fever, or if it occurs in connection with diabetes, the prognosis is more grave.

Varieties.—There are two varieties of xanthoma which require special mention, *diabetic xanthoma* and *juvenile xanthoma*.

Diabetic Xanthoma.—This differs from ordinary xanthoma in its rapid development, its temporary disappearance and reappearance, the hardness of the nodosities and their perifollicular situation, and the presence of pruritus or burning sensation; but it must not be regarded as a distinct disease. In a case of Hillairet's the

xanthelasma disappeared whenever the patient was taking diabetic diet. This form may also occur in subjects who are not actually diabetic, but predisposed to diabetes (Besnier). Hallopeau explains the glycosuria by localisation of the lesions in the pancreas, but this hypothesis has not been confirmed by autopsy.

Juvenile Xanthoma. — This develops before puberty. The lesions, instead of being yellow, are sometimes reddish.

PATHOLOGICAL ANATOMY.—The patches and nodosities of xanthelasma and xanthoma are constituted by a neoplasia, formed by connective tissue and large cells called *xanthelasmic* cells. The proportion of these elements varies in xanthelasma and xanthoma; in the former the cells predominate, in the latter the connective tissue, in various stages of fibrous transformation.

The xanthelasmic cells are large, mulberry form, multinuclear, and filled with fatty granules. They develop like fat cells, but incompletely; instead of the single drop of fat which occurs in fat cells, there are several isolated droplets. They are, in a way, fat cells which have been arrested in their development. According to Torok, this incomplete development is due to the situation of these cells in the dermis, which is not a normal situation for fat. In the subcutaneous tissue, a normal situation for fat, fatty neoplasms are formed of perfectly developed fat cells; for instance, lipoma, which is a *homœotopic* tumour; while xanthelasma is due to a *heterotopic* development of fat cells.

Chambard has described periarteritis and endarteritis obliterans, indicating that the lesion commences around the vessels. Quinquaud found an increase in the fat in the blood of xanthelasmic patients. Balzer has described a variety of xanthoma (*elastic xanthoma*), in which swollen elastic fibres are found along with scanty xanthelasmic cells, some of the fibres being in process of disintegration.

DIAGNOSIS. — Confluent granules of *milium* can easily be distinguished from xanthoma by incising one of the lesions, when the contents can be extracted; this is impossible in xanthoma. *Urticaria pigmentosum* is somewhat similar in colour, but can be diagnosed by the presence of typical elements. Politzer has seen multiple dermoid cysts which simulated xanthoma.

ETIOLOGY.—The causes of xanthelasma are obscure. It occurs at all ages. It sometimes appears to be hereditary, and congenital cases have been observed. The relation to arthritism and its various manifestations is frequent. According to Torok and others, it is a benign neoplasm of embryonic origin, due to the persistence and proliferation of the embryonic cells which form fat (Cohnheim's theory).

TREATMENT.—Besnier advises alkalis and turpentine, but I have never seen any benefit from these drugs. In diabetic xanthoma, an appropriate diet is required. Locally, the patches may be scraped;

but in the case of the eyelids, there is danger of causing ectropion by scraping the tissues too deeply. The application of perchloride of mercury collodion (10 per 100) has been recommended. Small patches may be destroyed by the galvano-cautery, but care must be taken not to produce cicatrices.

CHELOID.

Cheloid is a fibrous tumour, of variable form, which develops in the dermis, either spontaneously or around a cicatrix. There are thus two forms; *spontaneous* or primary cheloid, and *cicatricial* or secondary cheloid, the latter being more common.

Cicatricial cheloid must not be confounded with *hypertrophic cicatrix;* the former is a tumour developed around a cicatrix; the latter is a simple cicatrix, in which the cicatricial tissue has proliferated without extending beyond the original solution of continuity. The difference is easily seen by histological examination.

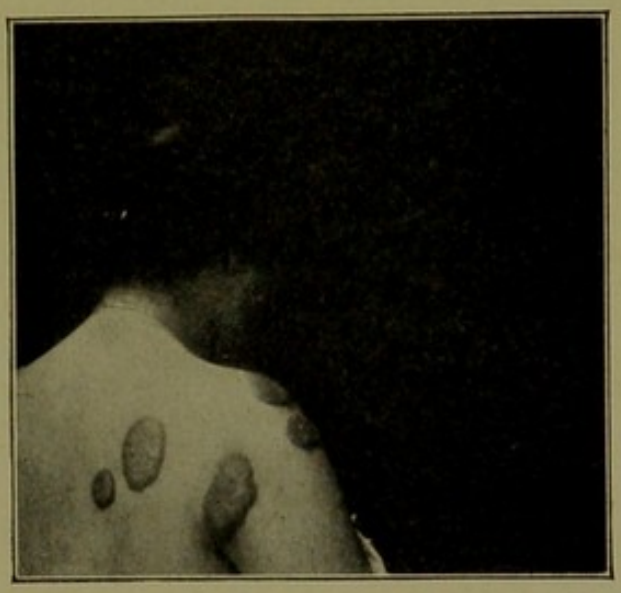


Fig. 88.—Cheloids on the back and shoulder.

Pathological Anatomy. — In a section of *hypertrophic cicatrix* the epidermis is thinned, and the papillæ of the dermis entirely absent; there are no hair follicles nor glands; the dermis contains numerous bundles of fibrous tissue. In a section of *cicatricial cheloid* the above changes are found in the centre of the tumour, on the original cicatrix; but beyond this, in the part which forms the cheloid tumour, the epidermis and papillæ are almost normal, and hair follicles and glands are present; in the dermis the bundles of fibrous connective tissue are continuous internally with the connective tissue of the cicatrix, and externally send out prolongations of variable extent.

In *spontaneous cheloid,* the epidermis and interpapillary prolongations of the Malpighian layer are normal, and the papillæ of the dermis intact; at a certain distance from the papillary layer is found the tumour, formed by bundles of connective tissue, some in the direction of its long axis, others oblique or perpendicular to it; between the bundles are cells and nuclei, especially abundant round the vessels. In both varieties the blood-vessels are numerous, with here and there lymphatic spaces.

It is sometimes difficult to decide whether a cheloid is spontaneous or cicatricial, as the differential characters are not always clearly defined.

ETIOLOGY.—*Cicatricial cheloid* is often secondary to the cicatrices of wounds and burns, to cicatrised lupus or syphilis, to the cicatrices of variola or vaccinia, to the pustules of acne, to leech bites, to perforation of the lobule of the ear, and to the application of nitric acid, croton oil, or other irritating applications.

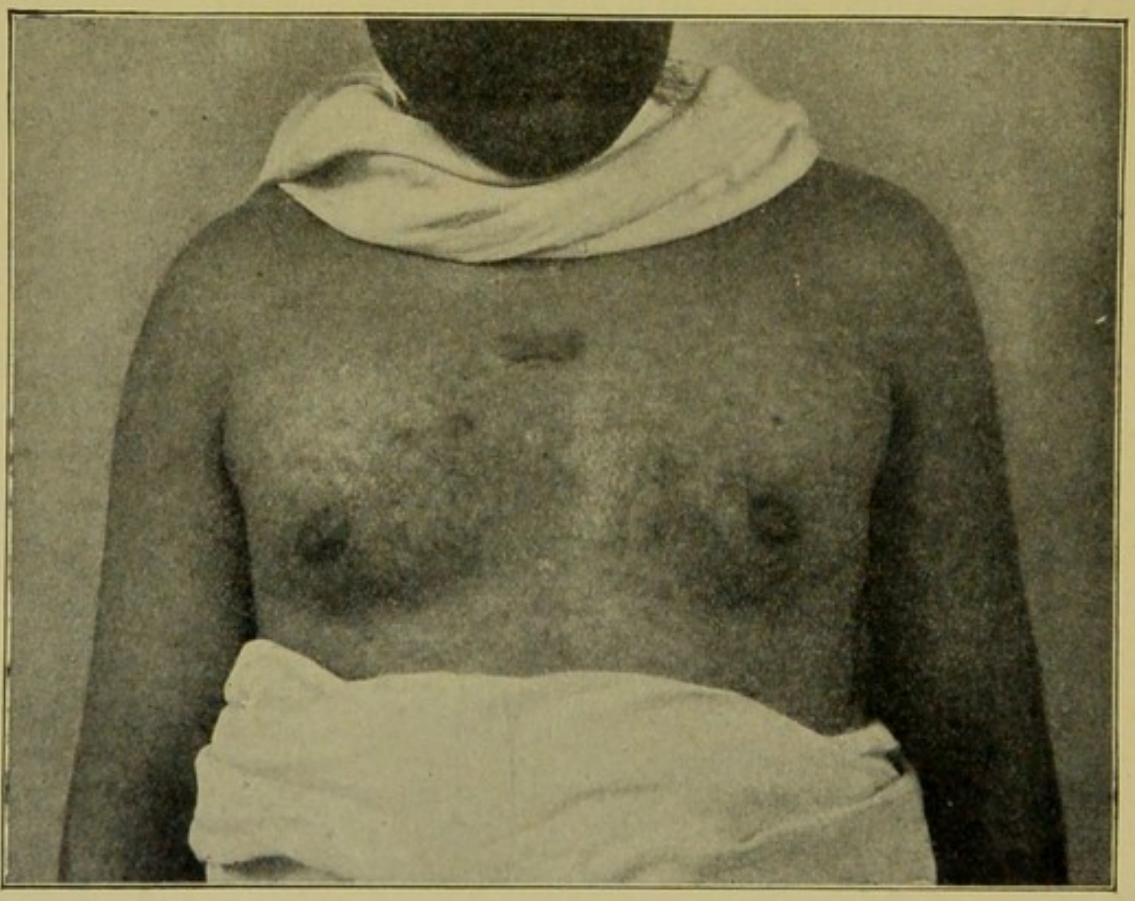


FIG. 89.—Spontaneous cheloid on the sternum.

Some authors deny the existence of *spontaneous cheloid*, and hold that every cheloid results from a wound (Barthélemy); others (Hardy, Vidal and myself) maintain that cheloid may develop in an absolutely healthy skin without any solution of continuity. In a case of Marie's, treated by scarification, an eruption of small tumours of the same kind appeared on parts which had never been the seat of any wound or cicatrix; and he suggested that the lesion might be due to an infectious agent; but in spite of repeated researches no microbe has been found (Vidal and Leloir, Gaucher and Sergent). Even if a microbe was discovered, the etiological problem would still not be solved, for the predisposition of certain persons to develop

cheloids must be taken into account. Kaposi has seen individuals who were free from cheloids after traumatism up to thirty or forty years of age, and then became subject to them.

Cheloid occurs in youth and adult age, and is more common in negroes. Heredity and consanguinity appear to have some influence in its production.

SYMPTOMATOLOGY. — In *cicatricial cheloid*, the cicatrix becomes enlarged and sends out processes into the neighbouring tissues. Nodules develop around it, which usually coalesce with the main tumour. The cheloid then forms a hard, projecting tumour with well-defined borders, smooth or nodular, sometimes round, but more often elongated in the form of a band or bridle, sending out fibrous

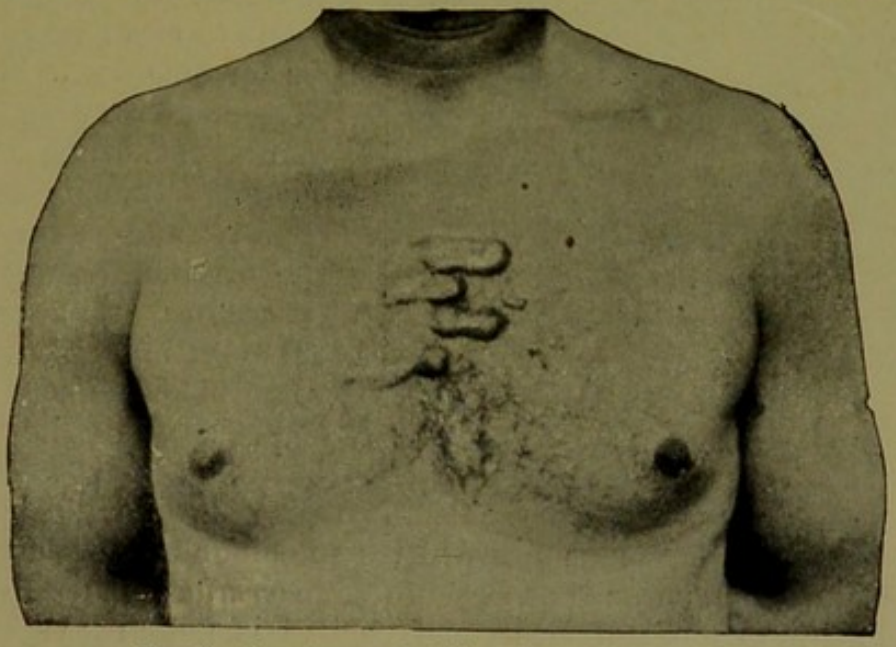


FIG. 90.—Spontaneous cheloids. (Marwedel-Chevassu.)

processes like the claws of a crab (hence the name cheloid). The colour of the tumour varies from dull white to red or purple, with peripheral telangiectases. When kneaded with the fingers, cheloids become turgid. They often have a tendency to disappear after a few months or years.

Spontaneous cheloid is at first a small induration, which develops very slowly. When single, its most common situation is over the sternum; but sometimes several cheloids are found in the same subject, appearing almost simultaneously and often placed symmetrically, especially on the chest, back, sides of the thorax, and upper limbs. In a case reported by de Amicis, there were 318 cheloids and also a cicatricial cheloid on the right arm, which developed on a wound made for the biopsy of a tumour.

Spontaneous cheloid is round or oval in form, less irregular than cicatricial cheloid. The skin over the cheloid is of normal appearance,

and presents hairs and glandular orifices. Spontaneous cheloid does not extend beyond the thickness of the skin, and remains mobile over the subjacent parts. Its other characters are the same as those of cicatricial cheloid. Sometimes the tumour is painless, sometimes very sensitive; sometimes there is violent itching, or shooting pains.

PROGNOSIS.—In rare cases, spontaneous cheloid undergoes resolution, terminating in a thin white cicatrix. It often remains stationary, but may increase; in the latter case fresh tumours may appear around the first.

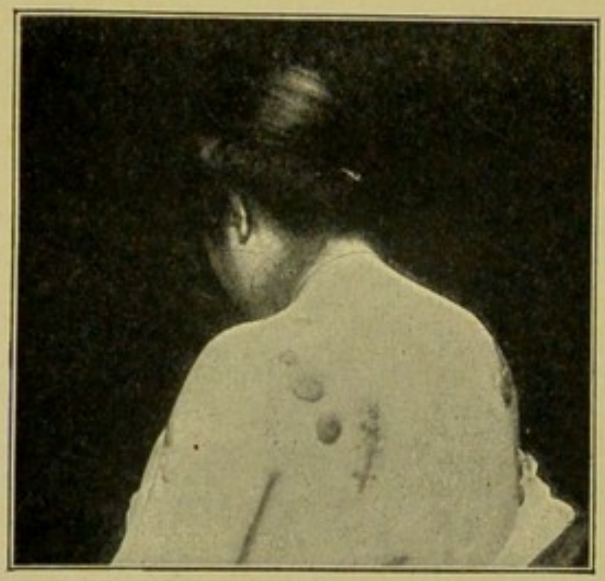


FIG. 91.—Cicatricial cheloid recurring after removal.

Cheloid never becomes inflamed or ulcerated. It tends to recur when cauterised or removed by the knife, in the cicatrix and in the points of suture. The recurrent tumour develops rapidly, and is larger than the original lesion, and an eruption of secondary tumours may occur around it. Cheloid causes no disturbance in the general health.

DIAGNOSIS.—Spontaneous cheloid must be distinguished from *fibroma* and *myoma*, which are whitish in colour and of larger size; and also from localised patches of *sclerodermia*. The distinction between spontaneous and cicatricial cheloid is not always easy, and depends chiefly on the history. *Hypertrophic cicatrix* differs from cheloid in being more or less raised above the surface of the skin, never extending beyond the limits of the original loss of tissue, and in showing no tendency to extension. It occurs frequently in scrofulous subjects after abscesses, and is often situated below the jaw. It sometimes ends in resolution, and never recurs when removed.

TREATMENT.—Removal by the knife and destruction by caustics are contra-indicated, because of inevitable recurrence. Scarification, recommended by Vidal, is an excellent method of treatment, but must be repeated for a long time to give any result. The prolonged application of mercurial plasters and hot sulphur douches are sometimes successful. Injections of oil of creosote (20 per 100) have been tried, but I have never obtained good results from this method.

Brocq recommends *electrolysis*, using platinum-iridium needles thrust deeply into the tumour, and a current of 5 milliamperes passed for thirty seconds. This gives rise to a white zone from

3 to 5 millimetres wide around the point treated; another point is then treated about half an inch from the first, and so on, so that the white zones touch each other, and the whole tumour is submitted to the action of electrolysis. The operation is repeated once a week. In my opinion the negative pole should be connected with the needle. However, electrolysis does not give such good results as scarification, and sometimes causes the tumour to increase in size.

Browning claims good results for sublimate collodion (1 in 30) applied every five days. Sevestre recommends arsenic internally.

More recently *radiotherapy* has been tried with considerable success. I have obtained very favourable results by this method, and am of opinion that radiotherapy is preferable to scarification. The application of *radium* is as efficacious as the X-rays, and is less liable to cause radiodermatitis. Radium will no doubt eventually supplant the X-rays in the treatment of cheloid.

FIBROMA MOLLUSCUM.

This is a round tumour, sessile or pedunculated, hard or soft, of variable size, from that of a pea or nut to that of a fœtal head.

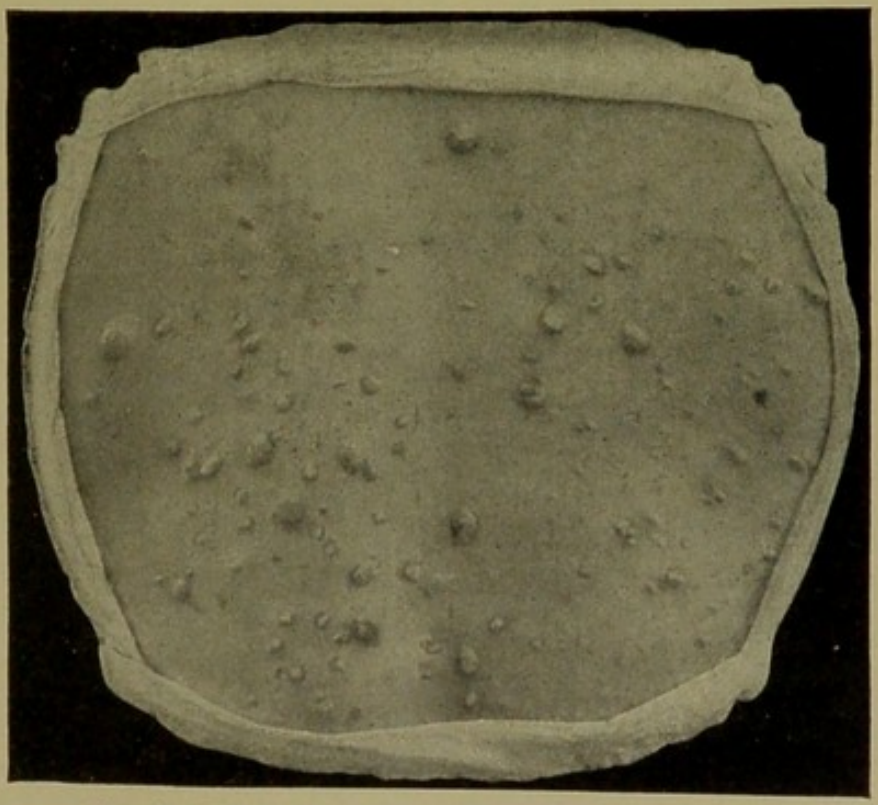


FIG. 92.—Fibroma molluscum. (St Louis Hospital Museum.)

The tumour can be moved with the skin over the subjacent parts. It is often pedunculated and pear-shaped (*molluscum pendulum*).

Sometimes it seems to be formed by a simple fold of skin (*dermatolysis*),[1] but a fibrous cord representing the remains of the fibroma can be felt by palpation. The skin may be pale or violet, and presents dilated vessels and sometimes comedones.

Fig. 93.—Generalised cutaneous fibromatosis. (Marwedel-Chevassu.)

In some cases there are hundreds of tumours present. This form of *generalised cutaneous fibromatosis* is often associated with pigmentary nævi and deficient mental development. It is sometimes known as *Recklinghausen's disease.*

Some of the tumours often shrink and resemble raisin seeds, while others develop enormously. They occur on the face, eyelids, scalp, trunk, genitals, and on the palms and soles. They may also occur on the mucous membrane of the hard palate and pharynx.

The tumours may cause trouble by their weight, owing to their situation. They are liable to inflammation and gangrene, and may become the seat of epithelioma. They may disappear spontaneously, often incompletely, leaving

[1] True *dermatolysis* consists in a looseness of the skin, which forms folds, and falls by its own weight over the parts situated beneath. It is observed chiefly on the eyelids, neck and abdomen. It is a congenital lesion, and must not be confused with the dermatolysis of fibroma.

a cutaneous fold or an atrophic spot; but as a rule they persist indefinitely.

PATHOLOGICAL ANATOMY.—The tumours consist of cellular tissue, which becomes more fibrous, according to the age of the tumour. According to Rokitansky, this tissue develops from the deeper layers of the dermis; according to Virchow, from the cellular envelope of subcutaneous fatty lumps; according to Recklinghausen, from the nerve sheaths; according to Fagge and Howse, from the hair follicles. In reality, it may arise from all parts of the connective tissue of the skin and its appendages. The pedicle contains blood-vessels.

ETIOLOGY.—Molluscum fibrosum is often congenital, or it may appear some time after birth, owing to hereditary predisposition. It often coexists with various kinds of nævi, especially pigmentary nævi.

DIAGNOSIS.—Molluscum is easily distinguished from *lipoma*, *mollusciform nævus*, and *sebaceous cyst*.

TREATMENT.—This consists in surgical removal of the tumours.

DERMATOMYOMA.

Besnier distinguishes two forms of dermatomyoma: *simple myoma* and *dartoid myoma*.

Simple myoma develops on the trunk and upper limbs, in the form of red spots the size of a lentil, round or oval, and very slightly raised; or, in the form of small red tumours, smooth and hard, of the size of a lentil to that of an almond, the colour disappearing under pressure. The tumours are painful when compressed. They develop slowly and last indefinitely. They are benign tumours, and do not recur when extirpated. They generally appear at an advanced age, without any appreciable cause; in Besnier's case, uterine myomata were also found at the autopsy. Balzer has shown that dermatomyomata consist of bundles of unstriped muscular fibres and elastic connective tissue.

Dartoid myoma is met with on the scrotum, nipple and labia majora. It may be sessile or pedunculated, and has the size of a nut or almond. It belongs rather to the domain of surgery than to dermatology.

ADENOID EPITHELIOMA.

This rare affection was first described histologically by Balzer, under the above name. It has also been described by Quinquaud as *eruptive epithelial celluloma*, and by Darier and Jacquet as *eruptive hydradenoma*, or *sudoriparous adenoma*.

It is characterised by small, pale red, flat, papular elevations: appearing first on the chest, where they may remain localised; or they may become disseminated over the trunk and limbs, seldom on the face. They vary in size from a millet seed to a lentil. They never develop into large tumours, even after persisting for twenty years. Their number varies, but may reach several hundreds. They

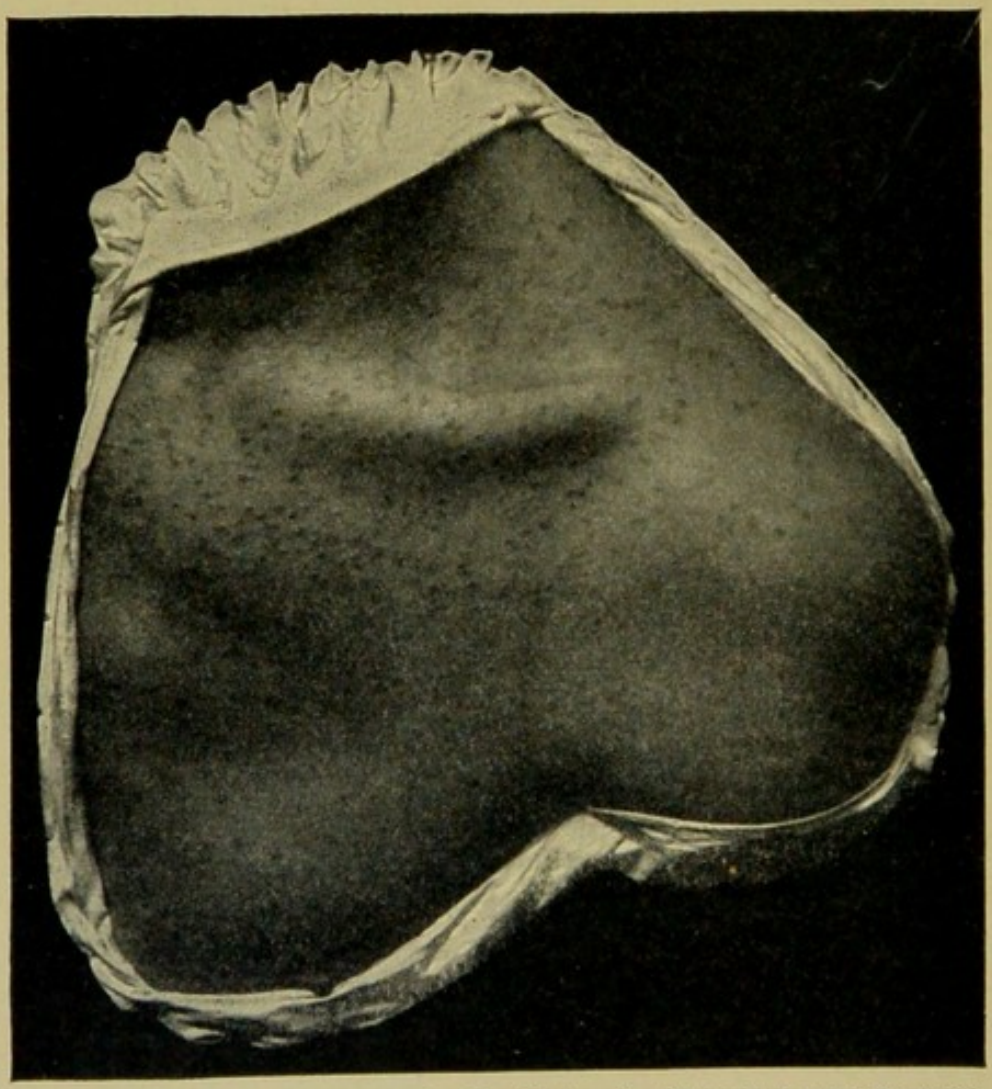


Fig. 94.—Eruptive hydradenoma (adenoid epithelioma). (St Louis Hospital Museum.)

are painless, never ulcerate, and develop in successive crops. The general health is unaffected.

Etiology.—This is unknown. In Quinquaud's patient, the lesions appeared to have commenced at the age of thirteen or fourteen years, and it is probable that their first appearance is not noticed.

Diagnosis.—This affection must be distinguished from the various forms of *acne*, from *molluscum contagiosum*, *flat juvenile*

warts, sebaceous adenoma, hydrocystoma, vascular and *warty nævi, colloid degeneration of the dermis,* and the *acneiform syphilide.*

PATHOLOGICAL ANATOMY.—Balzer concluded, from histological examination, that the lesion was an adenoid epithelioma with colloid cysts, originating in the sweat glands. Jacquet and Quinquaud regard it as an aberrant development from para-epithelial remnants. Torok, who gives it the name of *syringo-cystadenoma,* attributes it to abnormal development of the embryonic germs of sweat glands.

TREATMENT.—When the affection causes disfigurement, the lesions may be destroyed with the galvano-cautery.

SCLEROMA, OR RHINOSCLEROMA.

This is a morbid growth chiefly affecting the mucous membrane of the anterior nares and nasal fossæ; hence the name *rhinoscleroma;* but, as the affection may occur elsewhere, the name *scleroma* is

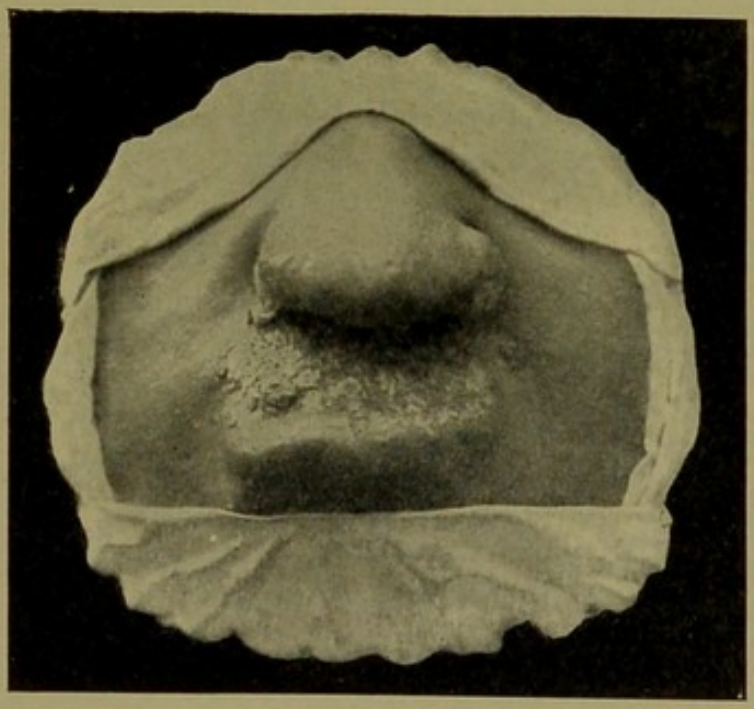


FIG. 95.—Rhinoscleroma. (St Louis Hospital Museum.)

perhaps preferable. It was first described by Hebra and Kaposi in 1870.

Rhinoscleroma has only been observed in subjects of ten to fifteen years of age, chiefly of the poorer classes, inhabiting the eastern provinces of Austria, the south-west of Russia, Italy, Central America, Brazil, Egypt and India. Four cases have been seen in the United States.

Rhinoscleroma is characterised by the formation of isolated or

confluent nodosities, of a bright red or brown colour and of almost cartilaginous hardness, which develop symmetrically inside the nostrils, on the nasal septum and on the adjacent part of the upper lip, giving a characteristic aspect to the lower part of the nose.

The first signs of the lesion are nasal intonation and a fœtid discharge from the nose. After a time, the nostrils and nasal fossæ become so constricted as to obstruct the passage of air. There is never ulceration, but rhagades may occur, exuding liquid which dries into crusts. The pharynx and soft palate then become the seat of nodosities, and the uvula disappears in the tumour. The pillars of the fauces are white and rigid, and sometimes contract adhesions with the posterior wall of the pharynx, as in pharyngeal syphilis. This results in difficulty in swallowing. The lesion may extend to the larynx and trachea, and may cause so much obstruction as to necessitate tracheotomy. After some time superficial ulcers occur in the pharynx, but they never extend deeply.

Scleroma may also extend to the bones, and invade the hard palate and alveolar border of the upper jaw, causing loss of teeth. It may spread to the lachrymal duct and produce a lachrymal tumour. In rare cases it spreads to the skull and causes cerebral compression. Invasion of the tongue and lower lip has also been observed.

The growth may commence in the pharynx, palate or larynx, independently of the nose. Potiquet saw a case which began in the ear.

Prognosis.—The evolution is very slow; eighteen years in a case of Vidal's. The growth never disappears spontaneously; at the most, it may shrink in parts, with the formation of fibrous cicatrices. It always has a tendency to spread, and recurs inevitably after all attempts at extirpation. However, Lubliner reported a case which disappeared after typhus. The lesion is never accompanied by glandular enlargement or signs of general infection, but it causes serious disturbance of the general health by obstructing respiration and deglutition. The prognosis is still graver when there is laryngeal obstruction or extension to the skull.

Pathological Anatomy.—By its histological structure, scleroma belongs to the group of granulomata, or chronic inflammatory neoplasms (lupus, leprosy, syphilis). Large cells are found as in leprosy and lupus, enclosing micro-organisms; discovered by Frisch in 1882. These are capsulated bacilli (2 μ by 0·5 μ), isolated or in chains, and very similar to the bacillus of Friedlander. Besides these large cells, hyaline bodies are present in great quantity.

Etiology.—The bacillus of Frisch is so frequently found that it has been regarded as the pathogenic microbe of rhinoscleroma; but inoculations have proved negative, so that the presence of the bacilli may be a secondary phenomenon; in fact, Netter and Thost have

shown that the bacillus of Friedlander, which resembles in many points the bacillus of Frisch, may be present in the saliva and nasal mucus in the normal state.

DIAGNOSIS.—Rhinoscleroma cannot be mistaken for *lupus*. It differs from *tertiary syphilis* by its hardness and slow growth, and by the symmetry of the lesions. Antisyphilitic treatment has no effect on it. *Epithelioma* differs by the presence of pain and ulceration.

TREATMENT.—Scraping is useless unless the whole lesion is removed, for scleroma is very liable to recur. Lang obtained some result by injections of salicylic acid (1 per 100), made every day, combined with naso-pharyngeal douches with salicylate of soda and the internal administration of salicylic acid (8 grains), thrice daily, for two months; later on, injections of carbolic acid (1 per 100) were substituted. Kaposi succeeded in almost completely destroying a rhinoscleroma by injections of salicylic and osmic acids. Stoukowenkoff cured a case with 222 injections of Fowler's solution (12 per 100). Koehler had good results with the galvano-cautery.

If the case is beyond radical operation, treatment must be directed towards re-establishing the passage of air through the nasal fossæ by means of chloride of zinc bougies and nasal sounds.

MYCOSIS FUNGOIDES.

This is a chronic dermatosis, which usually commences in the form of eruptions of various kinds; eczematiform, erythematous, urticarial or lichenoid. These are followed by the development of dermic tumours of a red colour, formed by reticular connective tissue and embryonic cells or fibroplasts. The disease was first described by Alibert, and afterwards by Bazin.

SYMPTOMATOLOGY.—Bazin described three periods: an eczematiform period, a lichenoid period, and a period of tumours. But this division does not apply to all cases; for, in the first place the eruption may be erythematous, or may even consist of more or less extensive infiltration; secondly, the order of appearance is not constant, the lichenoid condition may be the first. It is, therefore, more rational to divide the disease into two periods: (1) that of *premycosic eruptions*; (2) that of *tumours*.

First period.—The eruptions consist of patches resembling simple urticaria or erythema, or dry eczema, bright red or purple in colour, irregular in form, and varying in size and number. They disappear partially on pressure, and are sometimes slightly raised. There is much itching, and often furfuraceous desquamation.

In rare cases the premycosic eruption is psoriasiform, and for a

long time may resemble true psoriasis, for which it is usually mistaken.

The erythema is sometimes very extensive, and may be almost or completely generalised (*premycosic erythrodermia*). This eruption may precede the formation of tumours by several years, or it may follow their appearance. It consists of large red patches, similar to those of scarlatina or scarlatiniform erythema, which gradually spread, but usually leave some areas of skin unaffected. These

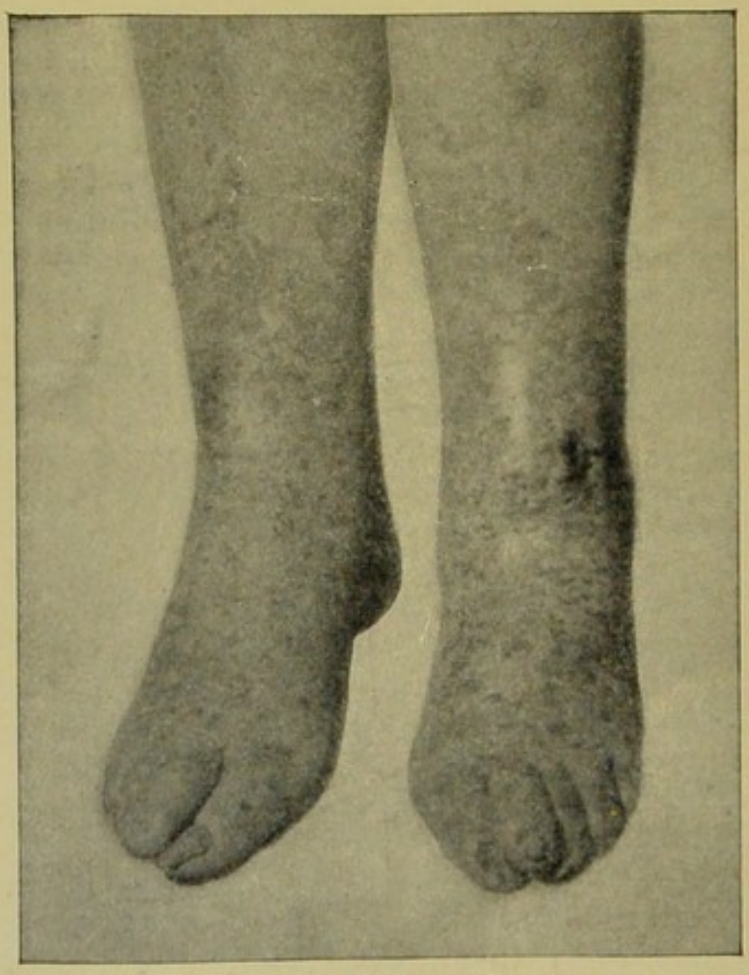


Fig. 96.—Premycosic erythrodermia. (Audry.)

patches are often accompanied by thickening of the skin; on the face, by œdema. Desquamation is sometimes hardly noticeable, sometimes abundant. Pruritus is intense. The lymphatic glands are more or less enlarged, and sometimes there is elevation of temperature, with profuse sweating. The erythrodermia presents alterations of increase and decrease, but seldom disappears altogether. Decolorised patches appear here and there, which after a time become pigmented.

All the premycosic eruptions are mobile and transitory, and recur at variable intervals for long periods. The itching may be

paroxysmal or continuous, and gives rise to scratching, excoriation, and exudation, which forms small crusts. Erythematous eruptions are caused by secondary inoculation of the excoriations. In proportion as the eruptions continue to recur, the lesions become deeper and more fixed, and form large lichenoid patches, consisting of small red acuminate papules, or of larger papules with flat and shining surfaces, like those of lichen planus, or of large broad papules.

FIG. 97.—Mycosis fungoides. (St Louis Hospital Museum.)

Later on, in the centre of the eruption or on the healthy skin, appear infiltrations, similar to patches of *erythema nodosum*, of a red or brick-red colour. On the face, these infiltrations cause considerable deformity; they may contract the palpebral fissure, enlarge the nose and deform the mouth. On the fingers, they form nodosities. In a case of Hallopeau's, one of these patches was situated on the prepuce, and simulated an indurated chancre with phimosis. In a case of Besnier's, the infiltration was accompanied by bullous and

vegetating patches, simulating pemphigus vegetans. Sometimes the patches are annular, or in the form of a semicircle.

These patches of infiltration may diminish or even disappear; but while some disappear, others appear elsewhere. Those which persist may become ulcerated. The premycosic period may last for ten or even twenty years before the tumours appear.

Second period.—Part of an erythematous or lichenoid patch becomes swollen and forms a tumour. More rarely, the neoplasm develops on a part of the skin which appears healthy. The tumours vary from a nut to an egg in size. They are generally hemispherical, but sometimes horse-shoe shaped; irregular in form when several tumours coalesce. The colour may be bright red, like a ripe tomato, or dark red, brown or purple. The base is usually broad, and sometimes surrounded by a furrow; the epidermis is smooth, and has a varnished appearance. The surface of the tumour is sometimes lobulated. The tumour can be moved with the skin over the subjacent tissues; it is painless on pressure, generally firm and often elastic, sometimes soft and even partially reducible.

There are two varieties of mycosis: one in which the tumours remain for a long time limited to one part of the body, and another in which they are generalised. They have even been observed on the palate, uvula and pharynx.

At this stage of the disease, tumours and eczematoid or lichenoid eruptions are present together. The tumours may remain without change for several months, and may then undergo absorption, or become ulcerated. Sometimes a tumour disappears in a few days, leaving no trace, or only a slightly depressed reddish spot: but fresh tumours always appear. Some of the tumours become ulcerated; the ulcerations are often superficial, bleeding easily and discharging a non-fœtid liquid, which forms brown crusts; in other cases, a slough is formed, which is eliminated by suppuration; in other cases, again, but more rarely, the whole tumour undergoes softening, and discharges a fœtid liquid through one or more openings.

The *mycosic ulcer* has a fungoid appearance; it is sometimes covered with crusts, and suppurates freely. Its borders, according to Hallopeau, often present a regular round swelling from 5 to 10 millimetres wide, abrupt internally and rounded externally, which undergoes necrosis at its inner border, while the external part, which is red and infiltrated, extends at the periphery and forms the zone of invasion of mycosis. This zone of invasion may cease to extend; the ulcer then cicatrises, completely or partially; in the latter case, extending on one side and healing at the other. Sometimes the zone of invasion reappears, and the lesion progresses afresh. The ulceration may extend in depth and destroy all the soft parts (ear and eye) till it reaches the bone.

When the hairy regions of the body are attacked, there occur patches of alopecia. The nails are altered, but present no characteristic lesions.

The viscera are never affected, except the spleen, which is sometimes enlarged. There is often, but not always, general glandular enlargement, but the glands do not suppurate, and may subside

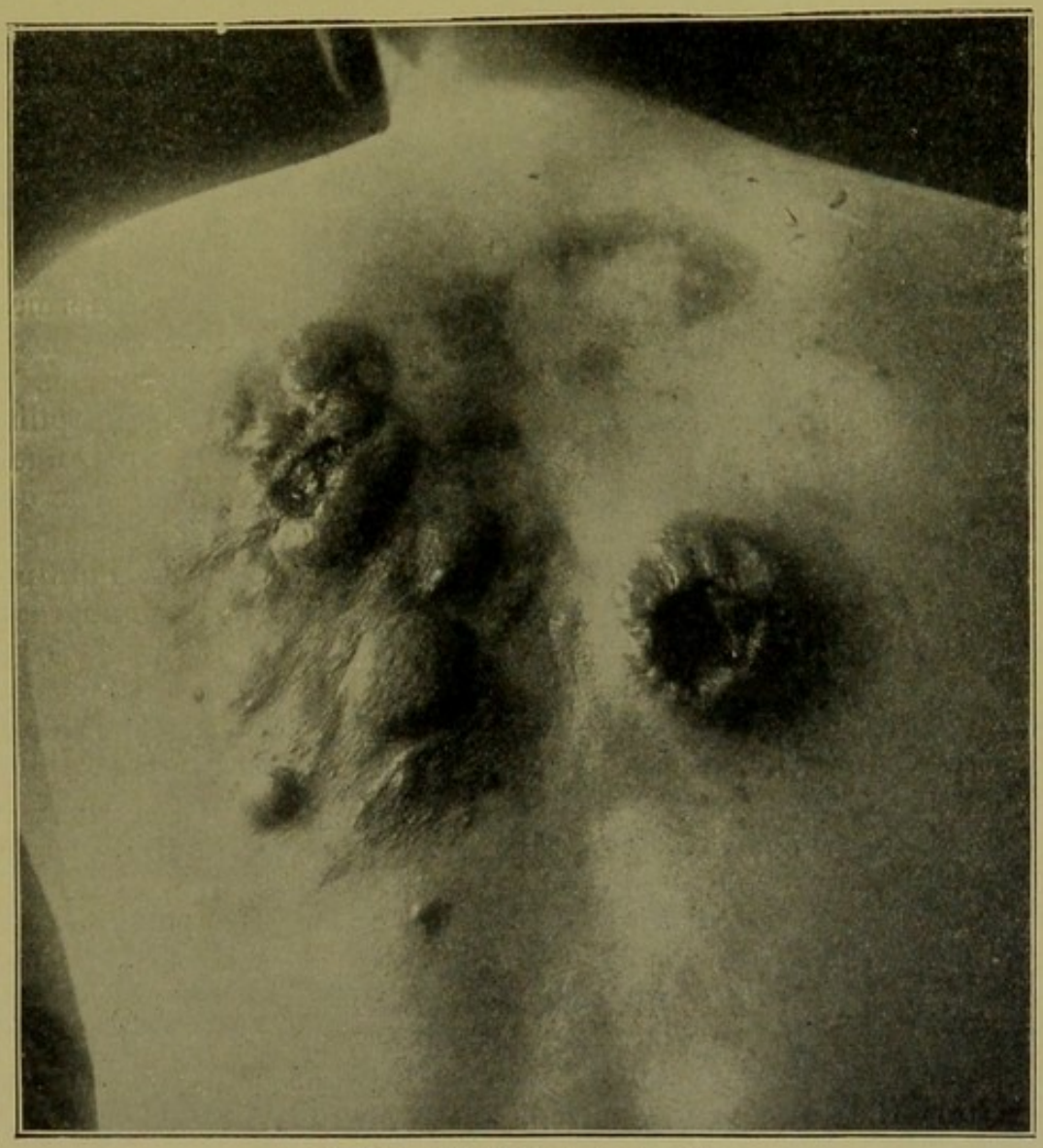


Fig. 98.—Ulcerated mycosis fungoides.

like the eruptions. Besides this general adenopathy, there is often enlargement of the glands corresponding to the tumours. Leucocytosis may be present, but this is rather rare.

Prognosis.—The evolution of mycosis fungoides is always slow, and presents remissions of variable length. The lesions may suddenly disappear for several months or even a year, to reappear later on in the form of eruptions or tumours. The disease usually lasts from

three to twelve years, but some cases have been fatal in six months. As the lesions progress, the patient becomes weak, emaciated and anæmic, and suffers from diarrhœa; sometimes there is fever and delirium. Death occurs either from exhaustion or from some complication, such as pleuro-pneumonia; however, Bazin has reported an authentic case of recovery.

VARIETIES.—Vidal and Brocq have described cases in which there were no premycosic eruptions, the tumours appearing from the first. I have also seen cases of this kind. The tumours are then more localised; they develop in the same way, but seem to have a more rapidly fatal termination.

Sometimes the tumours appear first, and are followed by the eruptions already described; in other cases, tumours and eruptions appear simultaneously.

PATHOLOGICAL ANATOMY.—The *premycosic eruptions* were formerly regarded as common eruptions on which the tumours became grafted; but Phillipson showed that they have the same structure as the tumours. The lesion occurs chiefly in the papillæ and subpapillary layer; it is constituted by round cells with easily stained nuclei, large cells with much protoplasm, sometimes giant cells. The deep connective tissue of the dermis appears normal. In the prickle-cell layer of the epidermis there are sometimes foci which Phillipson regarded as masses of fixed or embryonic cells, others as migratory cells.

In the *mycosic tumours*, the principal charges occur in the dermis, not in the papillary layer. Numerous embryonic cells are seen occupying the meshes of a fibrillary network. These cells constitute the characteristic element; they are extremely numerous, often round, sometimes fusiform like those of sarcoma, sometimes stellate and anastomosing with each other, as in myxoma.

In fact, mycosis fungoides, from the histological point of view, resembles sarcoma so closely that some authors regard the two diseases as identical; Sireday regards it as a form of *lympho-sarcoma*. But the clinical characters of mycosis and sarcoma differ too widely for the two diseases to be identical.

After the discovery of micrococci in mycosic tumours by Rindfleisch and Auspitz, it was thought that mycosis might be an infective disease, as Bazin formerly believed. I had already (1880) found micrococci in mycosic tumours, before the above-mentioned authors; but inoculations in the peritoneum of guinea-pigs with blood or fragments of diseased skin always gave negative results. However, the hypothesis of the infective nature of mycosis fungoides is admissible, but requires proof. The idea that it is a cutaneous lymphadenia cannot be maintained; for the conception of a lymphogenous diathesis which would connect such different diseases as

lymphadenia, leucocythemia, and mycosis fungoides is a pathological error which we need not discuss. From the histological point of view, mycosis fungoides should be placed among the embryoplastic tumours; a neoplasm closely allied to the sarcomas. This view, which I have held for a long time and which is now almost universally accepted, is supported by the researches of Dominici, who gives the following description:—

Examination of fragments of skin from mycosis fungoides reveals two modifications, apparently heterogenous: an adenoid condition, and inflammatory reaction of the skin.

The *adenoid condition* is constituted by infiltration of embryonic lymphatic cells in the interstices of the vascular connective tissue, and reticulation of this tissue.

The *inflammatory reaction* is constituted by: (1) transformation of some of the fixed cells into small epithelioid elements, or into giant cells, few in number and not very large; (2) hypertrophy of the vascular epithelium and cells of the walls of the blood-vessels; (3) sclerosis, affecting both the intervascular connective tissue and that of the walls of the vessels.

Of these three manifestations of the histological process of mycosis, one only is generally regarded as essential, viz., the immigration and multiplication of embryonic lymphatic cells or lymphocytes in the skin. The reticulation of the connective-tissue stroma appears to be a purely passive phenomenon, due to mechanical dissociation of this tissue by the embryonic lymphatic cells. The inflammatory reaction is only the result of secondary infection.

As the embryonic lymphatic cell is formed, according to the classical view, away from the skin, which is only a temporary resting-place for it, it follows that the mycosic neoplasm is fundamentally constituted by elements which were originally foreign to the skin. This has led to the hypothesis that mycosis fungoides is a *cutaneous lymphadenia*. This hypothesis has been contested by some authors, especially by Professor Gaucher, who regards mycosis fungoides as an embryoplastic tumour, arising from embryonic retrogression of the connective tissue of the skin, more or less related to sarcoma, and considers that some of the inflammatory manifestations form part of the histological process of this affection.

This view is confirmed by my researches on the pathological anatomy of the connective tissue, for the following reasons:—

(1) The development of the reticulum which encloses the lymphatic cells of mycosis is as important as the presence and multiplication of the latter elements. In fact, the reticular condition of the connective tissue of mycosic tumours does not result simply from dissociation of this tissue by the lymphatic cells, but from its *embryonic transformation*. This takes place in the dermis in the following way: (*a*) absorption of some of the bundles of fibrous and elastic tissue; (*b*) increase in the protoplasm of the fixed cells and multiplication of their nuclei; (*c*) transformation of the fibrous tissue into plasmodia, *i.e.*, protoplasmic masses with many nuclei; (*d*) decomposition of the plasmodia into stellate cells, which remain connected by their extremities so as to form a network of embryonic cells, the meshes of which afterwards enclose the lymphatic cells.

(2) The transformation of connective tissue into a network of embryonic fixed cells is, at least in part, a primordial phenomenon, both from the chronological and the evolutionary points of view. It precedes the immigration of lymphatic cells, so that the framework in which the latter become lodged is already formed for them. Again, a portion of the lymphatic cells are formed

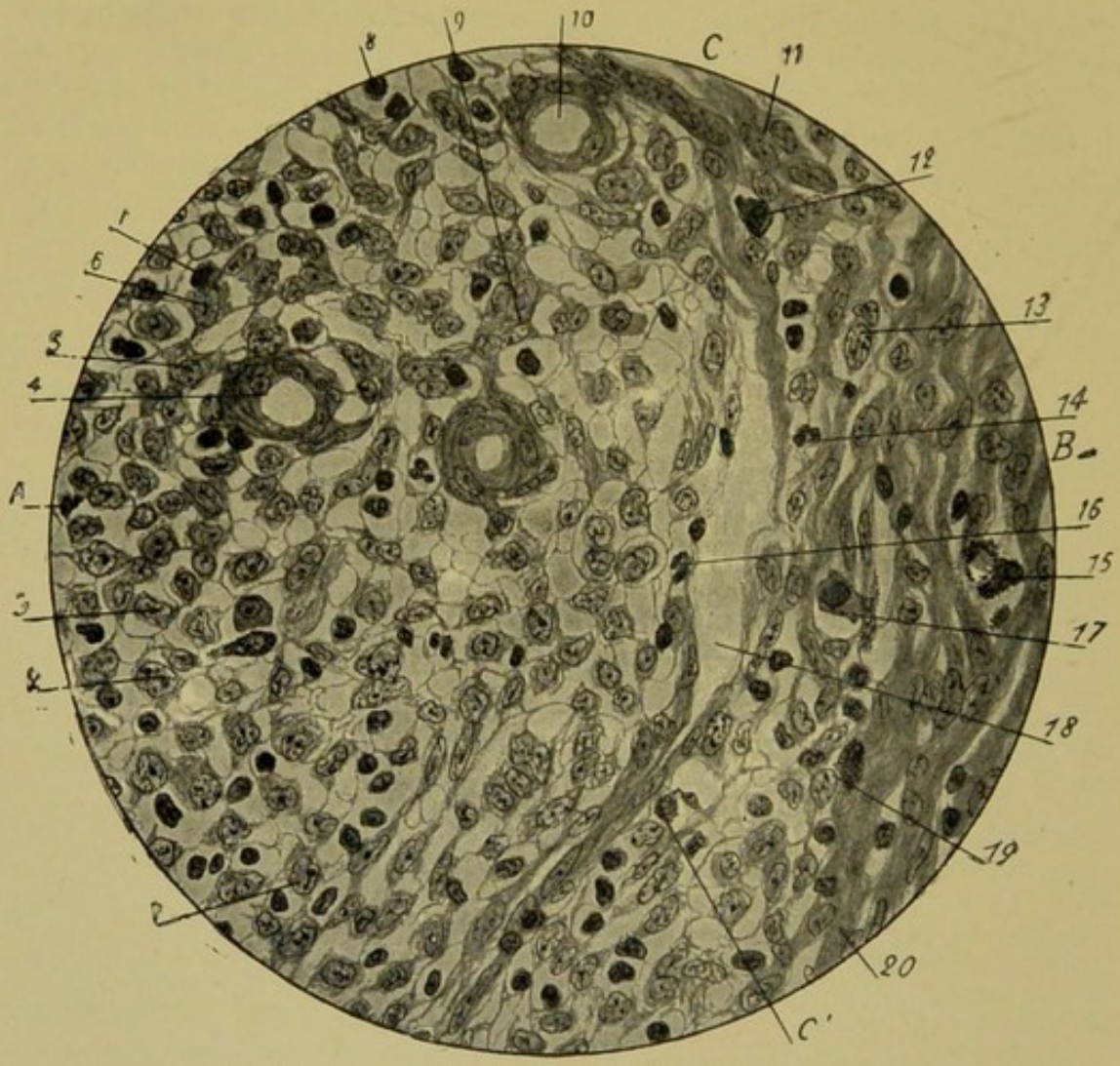


FIG. 99.—Section of skin from mycosis fungoides. (Dominici.)

This represents a part of the dermis where the embryonic reticular connective tissue predominates over the lymphatic cells.

On the left. A. Portion of dermis transformed into embryonic reticular tissue and formed by: (1) small stellate cells with large nuclei, united by their extremities into a continuous network; (2) embryonic plasmodia or embryonic giant cells with several nuclei. Diminution of connective-tissue bundles. A few free embryonic cells in the lymphatic spaces.

On the right. B. Portion of skin where the dermis preserves its fibrous structure. Immediately to the left of B is a zone between C and C', where the dermis is transformed into embryonic reticular tissue. The connective-tissue bundles and elastic fibres become absorbed, while the fixed cells multiply and become transformed into small stellate cells with large nuclei, united together by their protoplasmic processes.

1, 2, 5, and 6. Small stellate fixed cells of the embryonic connective-tissue network. 3. Small embryonic lymphatic cell or lymphocyte with clear nucleus, arising from the liberation of a fixed cell of the embryonic syncytium. 4. Capillary blood-vessel, the adventitial cells of which have anastomosed with the cells of the embryonic syncytium. 7 and 8. Embryonic lymphatic cells or lymphocytes with dark nuclei. 9. Small plasmodium, representing several embryonic cells fused together to form a protoplasmic mass with several nuclei. 10. Capillary blood-vessel with plasmodial or embryonic endothelium, the adventitial cells of which are united to the embryonic cells of the reticulum. 11. Portion of fibrous tissue with proliferation of cells, but absorption of collagen and elastine. 12. Plasma-cells. 13. Fibrous tissue of the dermis with commencing embryonic retrogression: a few lymphatic cells are seen. 14. Polynuclear cell. 15. Mast-cell. 16. Endothelium of a dilated lymphatic vessel. The endothelium is traversed by leucocytes with compact nuclei. The cavity of the vessel is destitute of lymphatic cells in this part of the section; this corresponds to the rarity of these elements in the surrounding connective-tissue stroma; in the zones where lymphoid cells are abundant, the lymphatic vessels are crowded with lymphocytes, emigrating from the lymphadenomatous regions towards the corresponding glands. 17. Plasma-cells. 18. Mast-cells. 19. Lymphocyte charged with eosinophile granules, which become transformed into polynuclear eosinophiles. 20. Polynuclear lymphocyte.

from the reticulum, by some of the cells of the latter becoming separated from their fellows and liberated in the form of small, free, embryonic cells.

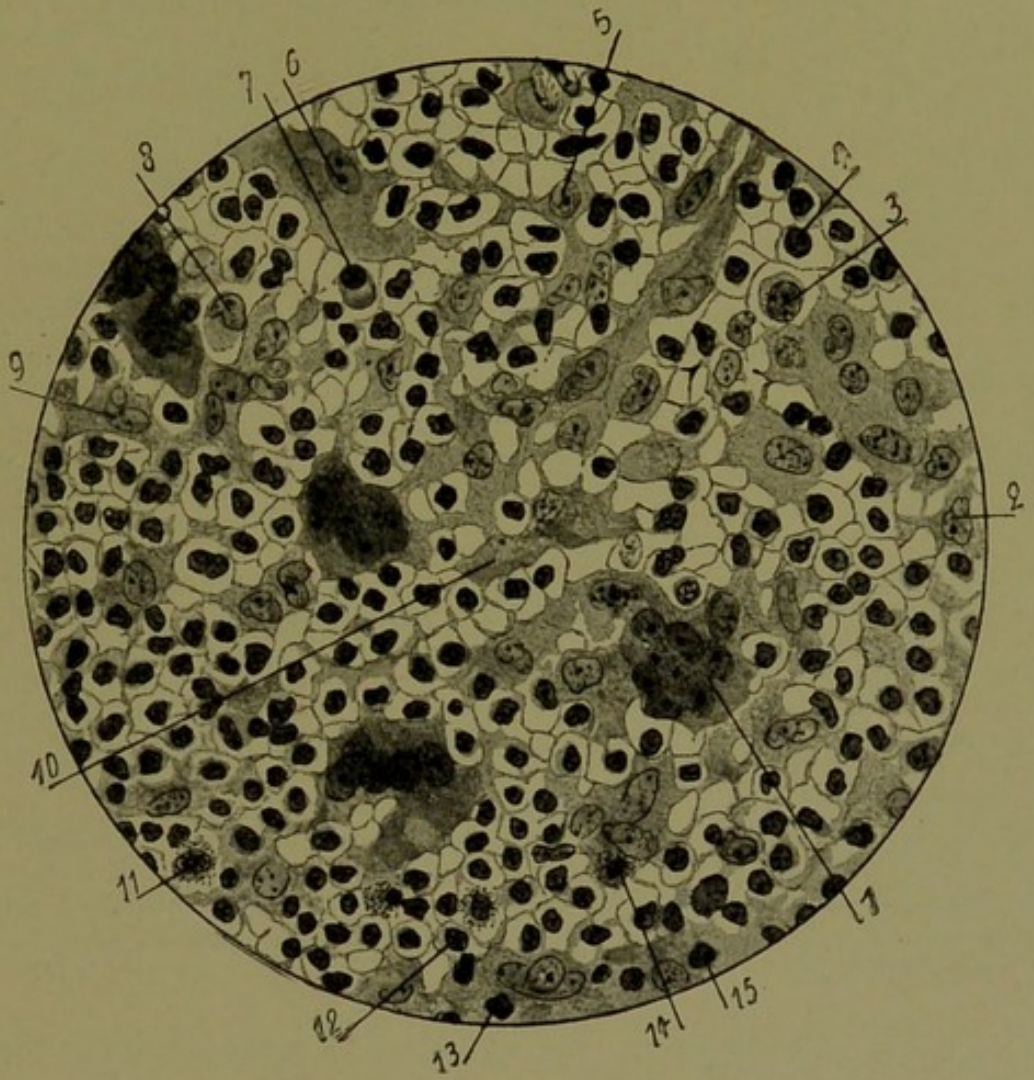


Fig. 100.—Section of skin from mycosis fungoides. (Dominici.)

This represents a part of the dermis where the inflammatory nature of mycosis can be recognised by :—

I. The transformation of part of the plasmodia and embryonic cells of the reticulum into epithelioid cells. Epithelioid transformation of the cells of the reticulum is characterised by : (*a*) increase in protoplasm ; (*b*) affinity of protoplasm for acid stains ; (*c*) clear colour of nucleus, the chromatin of which is very delicate.

II. The transformation of embryonic lymphatic cells (free cells) into macrophages, plasma-cells or eosinophiles. The free cells undergo the same evolution as in Fig. 99.

1. Plasmodium remaining embryonic in its lower part, and commencing to disintegrate on the right and upper part into cells of the reticulum, which immediately become epithelioid. 2 and 5. Stellate cells of the syncytium of embryonic type. 3. Embryonic lymphatic cell with clear nucleus, approaching the type of large mononuclear macrophage. 4. Embryonic lymphatic cell or lymphocyte with dark nucleus. 6. Large epithelioid cell with two nuclei. 7. Lymphatic cell of the type of plasma-cell. 8. Macrophage developed from such a cell as 3. 9, 10, and 13. Fixed cells of the syncytium undergoing epithelioid transformation. 11, 12, and 14. Lymphatic cells containing eosinophile granules. 15. Small epithelioid plasmodium with three nuclei, situated below and to the right of 14 (the indicating line is too short).

(3) The network of fixed embryonic cells is ready to undergo sarcomatous evolution. This process takes place at the period of tumours. The latter are partly formed by blocks of atypical sarcomatous tissue, in which the lymphatic spaces become obliterated, and from which the free embryonic cells

are excluded. On the other hand, these sarcomatous zones of mycosis are often infiltrated by plasma-cells, mast-cells, polynuclear and eosinophile cells, due to the inflammatory reaction concomitant with the process of tumour formation.

(4) The inflammatory reaction of mycosis forms part of the histological process of this affection. Among the inflammatory manifestations may be mentioned: (*a*) the local outbreak of plasma-cells, mast-cells, and eosinophile polynuclear cells; (*b*) the formation of epithelioid cells, simple or giant; (*c*) sclerosis.

I attribute these phenomena to a histological process connected with the mycosis for the following reasons: (1) they appear in the mycosic nodules, where the continuity of the epidermis prevents secondary infection; (2) the production of granular leucocytes, epithelioid cells, and sclerosis results from the evolution of the formative elements of the mycosic neoplasm, that is, from embryonic lymphatic cells and from fixed cells of the reticulum. The plasma-cells, mast-cells, and polynuclear eosinophiles are nothing more than the products of transformation of the lymphoid cells; (3) the epithelioid cells, simple or giant, are formed by the embryonic cells and plasmodia of the reticulum, the protoplasm of which is increased and becomes acidophile; (4) the sclerosis results essentially from the transformation of fixed embryonic cells into fibroblasts, which form dense fibrous bundles, and whose development is accompanied by obliteration of the lymphatic meshes and disappearance of the free embryonic cells.

Conclusions.—Although the lesions of mycosis fungoides may be essentially represented by a lymphoid or lympho-sarcomatous transformation of the skin, this affection, nevertheless, presents the histological characters of an infective disease. In fact, the lymphoid tissue of mycosis agrees in structure and evolution with that which is developed in the course of all chronic inflammatory conditions of the vascular connective tissue, and only differs from this in its diffuseness and power of growth. With regard to the sarcomatous metaplasia which sometimes complicates mycosis, this enters into the category of those lympho-sarcomas which recent research authorises us to class among the inflammatory processes.

ETIOLOGY.—This is unknown. As already mentioned, the infective nature of mycosis fungoides is probable, but not proved. The affection was rare formerly, but seems to have become more frequent lately, possibly because it has become better recognised, especially in its premycosic state. It generally occurs after the age of forty; but Landouzy observed a case in a child of seven months. It is more frequent in the male sex. It is not hereditary, and does not appear to be contagious.

DIAGNOSIS.—This is fairly easy in the second period when tumours are present, but very difficult in the first period. A premycosic eruption may be mistaken for *urticaria, dry eczema, lichen planus, psoriasis* or *scarlatiniform erythema.* But the premycosic eruption is not transitory like *urticaria,* and the colour of the patches is vermilion red; the skin is more infiltrated than in *eczema;* lichenoid premycosic patches are darker red than *lichen planus,* and the skin is more thickened; *psoriasis* is much less pruriginous, as are all other affections which may be confounded

with premycosic eruptions; in *scarlatiniform erythema* there is more abundant desquamation, and the lesion is more benign and of shorter duration. As Besnier remarks: "In every doubtful case of pruriginous dermatosis of prolonged duration and rebellious to treatment, whether it assumes the form of erythrodermia, psoriasis, diffuse or circinate squamous eczema, chronic urticaria or lichenoid prurigo, the possibility of a premycosic eruption of mycosis fungoides must be borne in mind."

Mycosic infiltrations sometimes simulate *tertiary syphilides;* but the presence of eczematoid eruptions and pruritus, the absence of syphilitic antecedents, and the failure of antisyphilitic treatment will decide the diagnosis. The infiltrations may also be mistaken for *leprosy;* but in the latter the patches are anæsthetic, of a brown colour, and more permanent. Premycosic erythematous patches differ from *lupus erythematosus* by the intensity of the itching, the presence of glandular enlargement, and the coincidence of eczematoid eruptions.

At the *tumour period* the diagnosis is easier, and the differential diagnosis chiefly concerns *cutaneous lymphadenia* and *sarcoma.* In mycosis there is less enlargement of the lymphatic glands, excepting those corresponding to ulcerated tumours; the spleen is not always enlarged; leucocytosis is not constant; the tumours are always cutaneous, while those of lymphadenia are at first subcutaneous; and the tumours may disappear, an event which never occurs in lymphadenia. Moreover, it must be admitted that the nature of the affection called "cutaneous lymphadenia" is not quite definite; it was called *pernicious lymphodermia* by Kaposi.

Telangiectasic sarcoma differs from mycosis in nearly always commencing in the extremities, in its violet colour, in the absence of premonitory eruptions, and in scarcely ever disappearing spontaneously. The diagnosis between certain forms of *generalised sarcoma* and mycosis, when it begins in the form of tumours, is sometimes difficult; but the mycosic tumours have a brick-red colour, and a rapidity of appearance and disappearance which is not observed in sarcoma; also there are no visceral lesions.

TREATMENT.—Iodides have been tried without effect. Arsenic has been given both internally and by subcutaneous injection, the latter method being successful in a case of Koebner's. Brocq treated mycosic ulcers successfully with camphorated naphthol, and injected the same into the tumours; this produced sloughs, which were also dressed with camphorated naphthol.

Radiotherapy has been much employed in mycosis fungoides. There is no doubt that it causes absorption of the tumours, but it does not cure the disease; in fact, severe symptoms have been produced, fatal in one case, after the rapid absorption of mycosic tumours under the X-rays.

However, radiotherapy is the best form of treatment at present known; but it must be used with great care, and should not be applied to too large areas at a time. But in spite of radiotherapy, fresh tumours often appear near those which have been cured by X-rays.

CUTANEOUS SARCOMATOSIS.

This disease occurs in two forms: *melanotic* and *non-melanotic*. In the latter form, the tumours may be generalised, primary and telangiectasic; or the lesions may consist of a single tumour, which may become the point of origin of secondary generalised tumours. There are thus four distinct types to be studied separately: (1) primary generalised telangiectasic sarcomatosis; (2) common localised sarcoma; (3) secondary cutaneous sarcomatosis; (4) melanotic sarcoma.

Primary Generalised Telangiectasic Sarcomatosis.

This form was described by Kaposi under the name of *pigmentary sarcoma*, but this denomination causes confusion with melanotic sarcoma; the names *telangiectasic sarcoma*, or *hæmorrhagic sarcoma*, given to it by Tanturri and Koebner respectively, are preferable, as the colour is not due to pigment but to blood colouring matter.

Symptomatology.—This affection always begins in the extremities. On the hands and feet, on the dorsal surface or on the palm or sole, a kind of hard œdema develops, which causes painful tension or pricking sensations. Bluish spots then appear, and in the middle of these, hard isolated bluish nodosities. Sometimes the blue spots are absent, and the nodosities appear from the first; they give a peculiar mammillated appearance to the hands and feet, and a characteristic purple colour; the fingers and toes are swollen.

There are two periods: a period of *infiltration*, and a period in which the nodosities become *tumours*, but this order of appearance is not constant; sometimes the disease appears first as an isolated tumour, followed later on by manifestations on the limbs; sometimes as multiple lesions on the trunk or face.

The tumours are generally round in form, sometimes flat, such as those situated on the plantar region. Their colour is dark red at first, but soon becomes purple, sometimes brown. They are very numerous, from 30 or 40 up to 1000, and vary from a pea to a hen's egg in size. They may be sessile or pedunculated, isolated, or grouped to form patches, the centre of which is atrophied; they are often covered with epidermic scales, sometimes with horny stratified elements. They are of only moderately firm consistence, sometimes soft and compressible when they are very vascular; they bleed freely

when punctured. When the vessels rupture, the tumours may acquire a firm consistency due to coagulation of the blood (erectile sarcoma). Most of the tumours are situated in the dermis, some in the subcutaneous tissue.

This form of sarcomatosis may occupy all regions of the body. In spite of the multiplicity of the tumours, the general health remains good for a long time; the lymphatic glands are never affected, and there is no leucocytosis.

Fig. 101.—Telangiectasic sarcoma.

Evolution.—The tumours may remain stationary, or may disappear spontaneously in a few weeks, or even in a few days, leaving a yellowish gray cicatricial spot, which may cause retraction of the skin, especially on the palms. But other tumours appear during retrogression of the first; tumours recur after surgical removal, and the disease may invade the soft palate, fauces, hard palate, etc. There is sometimes fever, with anorexia and diarrhœa, and the patient becomes gradually exhausted.

In the last stage of the disease, tumours develop in the nasal, laryngeal, and tracheal mucous membranes, in the lung and pleura, on the surface of the stomach and intestine, and in the liver, spleen, and muscles, causing symptoms corresponding to these different localisations. A curious point is that the lymphatic glands never become sarcomatous. The patient succumbs to visceral sarcomatosis, or to septicæmia, when some of the tumours become ulcerated; but the latter event is rare.

Sarcomatosis develops slowly, generally lasting two or three years; sometimes ten to eighteen years. I have seen a case in which the lesions were confined to the hands and feet for eight years. In other cases, especially in young subjects, death may occur in a year, or even in a few months.

Pathological Anatomy.—The tumours present a purple or

orange-green colour on section, due to changes in the hæmoglobin deposited in the tissues. There is no sharp limit between the healthy and diseased tissues. Histological examination shows the presence of cells, blood-vessels, blood pigment, and intercellular substance.

The cells are those of sarcoma, and form islets of different sizes, situated in the dermis around the glands and hair follicles. They are round and fusiform; the round cells vary in size, but are always nucleated; the fusiform cells become connected to form a fibrillary tissue. They appear to be connected by an amorphous substance full of blood pigment.

The vessels appear as lacunæ of various shapes, the walls being formed by sarcomatous elements; the lacunæ are lined by swollen epithelium covered with blood corpuscles, coloured or decolorised (Tanturri). This vascular new formation appears to be primary, and not secondary to the formation of sarcoma; possibly it plays the principal part in the production of the disease. These newly formed vessels have weak embryonic walls, which are easily ruptured; the blood effused in the tissues undergoes changes; the blood pigment accumulates between the cells and even inside them, around the nucleus.

The hair follicles are never altered as in mycosis, and when the scalp is affected there is no alopecia. The epidermis may be thinned or thickened; there is often pigmentary infiltration of the Malpighian cells.

ETIOLOGY.—This is very obscure. Sarcomatosis is met with more often in men than in women, between the ages of forty and sixty; some cases have been seen in children. In most cases there is no evidence of heredity, but in some cases there has been a history of cancer. The disease is rare in France; it occurs chiefly in Hungary and the countries bordering on it, but there is no explanation of this distribution. It is possible that sarcomatosis is an infective disease, but this remains to be proved.

DIAGNOSIS.—Sarcomatosis may be mistaken for *cysticercus* of the skin and subcutaneous tissue, but this is settled by puncture. *Fibroma molluscum* is quite a different formation. *Myoma* of the skin occurs in the form of small, hard, smooth, red tumours, painful on pressure, which remain for a long time stationary. In *tubercular leprosy* the tubercles are yellow, red, or brown, and never form real tumours; there are also disorders of sensibility. For the diagnosis between sarcomatosis and *mycosis fungoides*, see page 259.

TREATMENT.—Koebner and Shattuck have obtained good results with subcutaneous injections of Fowler's solution. Injections of four, six, or nine drops, with an equal quantity of water, were injected every day for several months; the treatment being

suspended from time to time. I prefer the following preparation of arsenic:—

Arsenious acid	1 part
Carbonate of soda	10 parts
Distilled water	100 „

The injections are made in the buttock, or in the tumour itself. Radiotherapy or radiumtherapy may be employed, as in other malignant growths, but I have not tried these methods in telangiectasic sarcoma.

Primary Localised Sarcomatosis. Common Sarcoma.

This form of sarcoma is not telangiectasic, and is quite different, clinically, from the preceding disease. It occurs especially in women. It sometimes develops on a nævus. Its most common situations are the dorsal surface of the foot, the index finger, and the palpebral region. It arises in the dermis or hypodermis, and forms a tumour of variable size, never exceeding the size of an orange, of hard consistence, and the same colour as the normal skin.

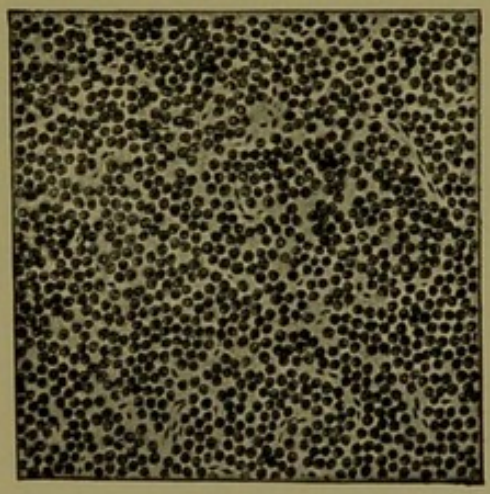


Fig. 102.—Section of round-celled sarcoma.

Fig. 103.—Section of spindle-celled sarcoma.

The tumour remains stationary for a long time (three years in one case); it then ulcerates, and the lymphatic glands become enlarged, but not always sarcomatous. In a case of Shepherd's, amputation through the thigh led to rapid diminution of the glands, which were only inflamed. This distinction is important as regards surgical intervention

According to Hallopeau, "the sarcomatous tumours extend along the course of the lymphatic vessels; they become the seat of ulcerations, which may persist or may undergo cicatrisation, generally partial and not durable; in their mode of distribution, and in their

characters and evolution, they present a great analogy to nodular tuberculous lymphangitis, but they differ from this in the constant hæmorrhages which are produced, either in the interior of the neoplastic tissue, or externally after ulceration of the tumour."

Generalisation may take place in the viscera (liver, etc.), or in the skin (secondary cutaneous sarcomatosis).

PATHOLOGICAL ANATOMY.—The tumour is formed of round or fusiform embryonic cells. In a case of Koebner's, telangiectases and blood pigment were present.

PROGNOSIS.—This is not so grave as in general sarcomatosis. The tumour may be cured temporarily by complete removal, but it almost always recurs. When it becomes generalised, death is inevitable.

Secondary Cutaneous Sarcomatosis.

This may be secondary to primary cutaneous sarcoma, or to visceral or glandular sarcoma. In the published cases, the secondary cutaneous tumours appeared from eighteen months to two years after the appearance of the primary tumour; their number varied up to a hundred. The tumours occur chiefly on the trunk, rarely on the head or limbs; they vary in size, and are quite painless. Some develop in the skin, others in the subcutaneous tissue. The glands corresponding to the regions in which the tumours are situated are not always affected. Some of the tumours may undergo retrogression, but others always appear. They are liable to recur after removal. Death takes place in six months to two years after the onset, from visceral metastases.

Melanotic Sarcoma.

This is a form of sarcoma which is infiltrated with a special colouring matter, *melanine*. It often develops on a pigmentary nævus. It occurs between the ages of twenty-one and fifty-six. The tumour is always single at first; generalisation in the skin and viscera is secondary. The situation of the tumour is variable. It is small at first, and never exceeds the size of a walnut. Its shape is oval or spherical; its base usually sessile, rarely pedunculated. Its colour is very dark, like that of black ink. It is always of firm consistency. It is at first movable, but afterwards becomes fixed to the subjacent parts.

Melanotic sarcoma remains stationary till it becomes generalised, as the result of unsuitable treatment or irritation, or without any appreciable cause.

Generalisation takes place either by the lymphatics or by the blood-vessels. In the former case, the corresponding lymphatic glands become sarcomatous; the lymphatics between the tumour and

the glands may be the seat of sarcomatous deposits; the remote glands are affected later.

In the second case, generalisation takes place in the viscera and in the skin. In the skin, fresh tumours may appear round the first or in other regions. They may be situated in the dermis or hypodermis, or in both. Their number may be considerable; their colour is black, and their structure the same as that of the primary tumour. Some tumours may disappear entirely, leaving a black spot, or even no trace of their existence; but they may also recur in the same place. Sometimes the tumours ulcerate, and then present a black, vegetating base surrounded by an indurated border. The ulcer discharges a little pus and a thick blackish liquid; it is rarely

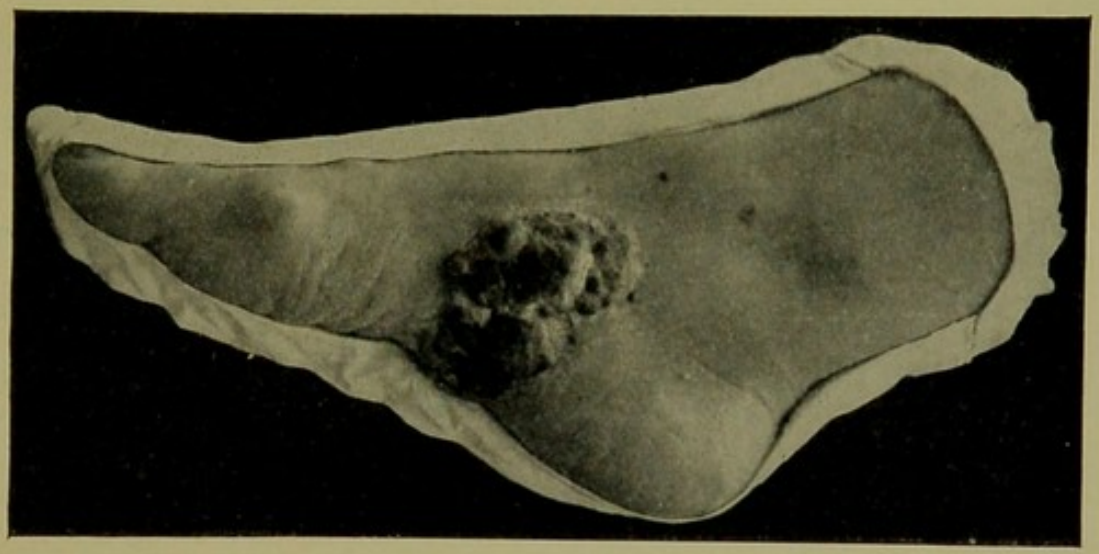


Fig. 104.—Melanotic sarcoma of the foot. (St Louis Hospital Museum.)

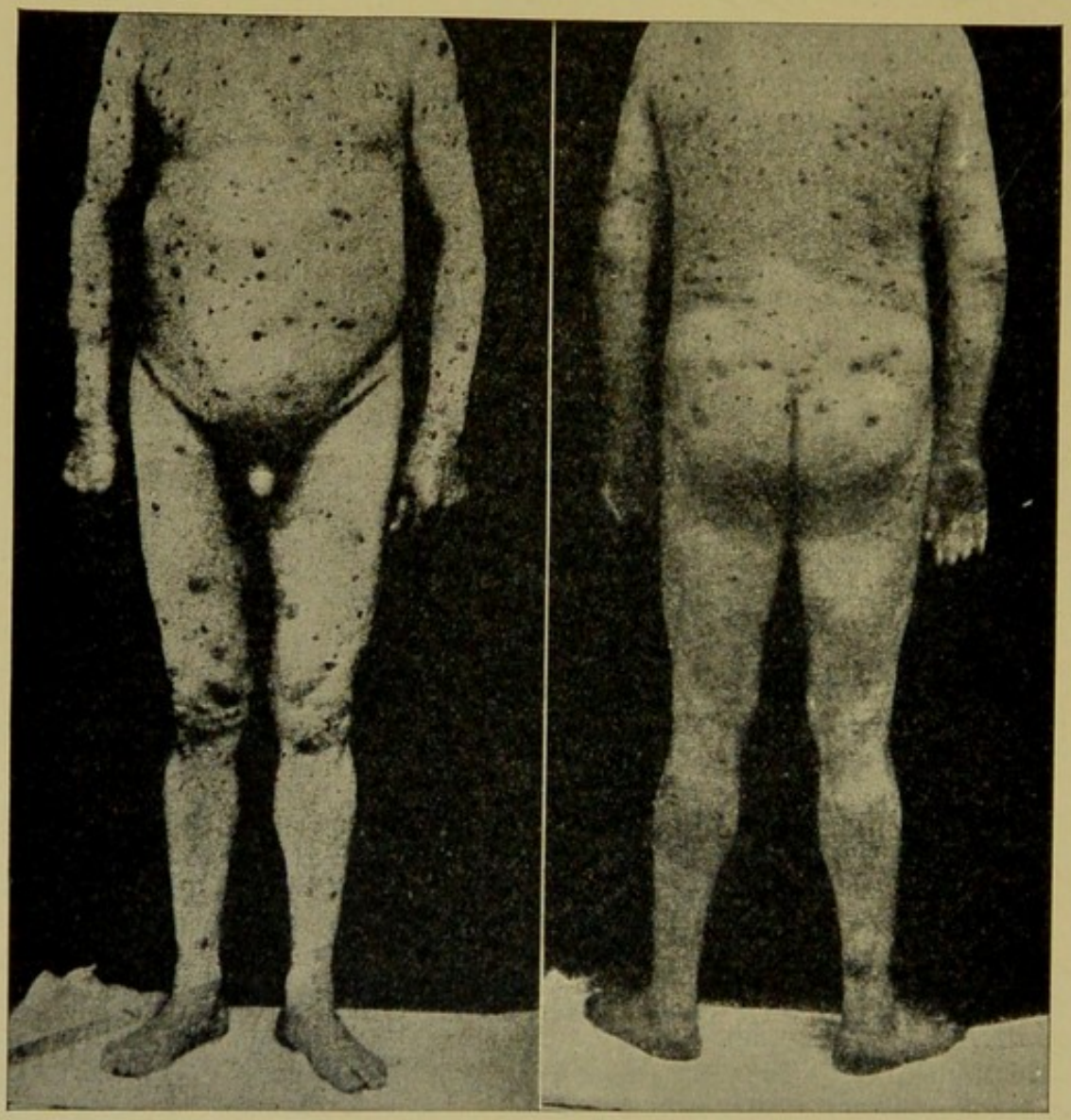
hæmorrhagic. At this period the patient becomes cachectic, the skin is of a blackish colour, and the urine becomes dark on the addition of nitric acid. The termination is always fatal, death occurring in two or three years.

Cutaneous melanotic sarcoma may also be *secondary*, to a primary sarcoma of the choroid, for example.

Diagnosis.—It is sometimes difficult to diagnose between melanotic sarcoma and *pigmentary nævi;* but the latter are not so dark in colour, and have quite a different evolution. It must be remembered that black spots sometimes appear on the skin after the ingestion of *antipyrin.*

Pathological Anatomy.—Melanotic sarcoma consists chiefly of fusiform cells, but round cells are also sometimes present. There are very few vessels. The pigment is deposited in the cells around the nucleus, but it is absent in some cells; it is also present in the intercellular substance. The epidermis is always normal. In

general melanotic sarcomatosis, melaniferous leucocytes are found in the general circulation—an important point in prognosis.



FIGS. 105 and 106.—Generalised cutaneous melanotic sarcomatosis.

TREATMENT.—Melanotic sarcoma should never be touched with the knife. Although an attempt may be made to extirpate a simple sarcoma, operative interference in melanotic sarcoma, even when skilfully performed, can only end in disaster.

However, I have devised a method of operation on melanotic sarcoma which appears to be free from the disadvantages of surgical excision. After having determined that there are no melaniferous leucocytes in the blood, and that, consequently, generalisation of the disease is not imminent, the tumour is widely removed with a thermo-cautery knife; after this, the surface is treated by fulguration

by currents of high frequency and high tension, according to the method of Keating-Hart (see p. 295). I have performed this operation on a melanotic sarcoma of the foot; six months after the operation the tumour had not recurred, and the patient was in good health.

Radiotherapy.—The X-rays have been tried in all forms of cutaneous sarcomatosis. The mode of application is the same as for epithelioma (p. 284).

At first there were great expectations from this mode of treatment, and remarkable cures were published; but now that the enthusiasm of the early days has calmed down, favourable results have become more and more rare, and, inversely, unfavourable results have become more numerous. Radiotherapy should, therefore, be used with a certain amount of scepticism in sarcoma. This treatment may be useful sometimes, but it often fails to prevent the malignant evolution of sarcoma; in fact it may even precipitate it.

Treatment by *radium* may also be tried, but its value remains to be proved. I have under observation a case of melanotic sarcoma of the internal angle of the eye, which was treated by radium by Dr Domenici; it appears to be improved, but is not yet cured.

EPITHELIOMA.

We shall only deal here with epithelioma of the skin (*cancroid* of the older authors), leaving aside epithelioma of the mucous membranes. There are three varieties: (1) a superficial form, characterised by the presence of ulceration without any prominent tumour (*rodent ulcer*); (2) a form resembling rodent ulcer in its superficial situation, but differing from it by its papillomatous appearance (*superficial papillary epithelioma*); (3) deep epithelioma with an obvious tumour (*cutaneous cancer*).

Rodent Ulcer.—This appears in the form of one or more small, hard, shining granulations, the size of a pin's head and of a pale red colour. These coalesce to form an indurated patch which eventually ulcerates, either spontaneously or after some traumatism, and is covered with a crust formed of dried blood. After a time, sometimes several years, the primary lesion extends by the peripheral development of fresh granulations. The lesion most often commences at the internal angle of the eye, on the nose, or on the forehead, more rarely on some other part of the face. The ulcer is superficial, round or irregular, with sharply cut indurated borders, and a reddish brown, granular base, secreting a viscid liquid. There is often a cicatrix in the centre of the ulcer, which then forms a peripheral furrow surrounding the cicatrix. Sometimes the forma-

tion of granulations at the borders of the ulcer ceases, and the lesion heals spontaneously after some years, sometimes fifteen or twenty years. But in most cases the lesion gradually extends, and in severe cases may destroy a large portion of the face, including the muscles and bones, producing a vast cavity which may extend to the pharynx. The ulceration is usually accompanied by itching; pain is only

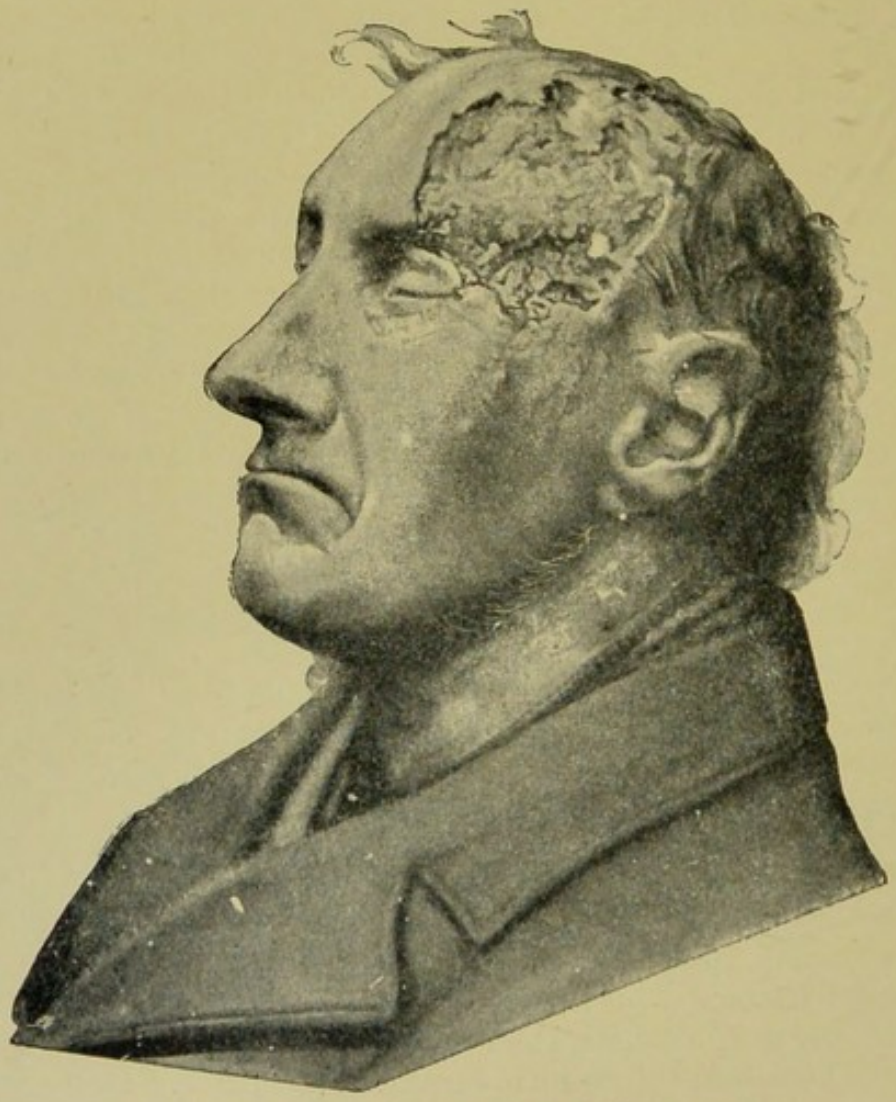


Fig. 107.—Rodent ulcer. (Sultan-Kuss.)

severe in very extensive cases. Whatever the duration or extent of the disease, the lymphatic glands are hardly ever affected, and there is no cachexia nor visceral metastasis. Recurrence after operation is common.

According to Dubreuilh, rodent ulcer is an epithelioma, formed by small cells grouped in round or angular lobules, at the periphery of which the cells are arranged radially, like a layer of cylindrical epithelium. These cells have no prickle processes, they do not

undergo keratinisation, and never form epidermic globes. But there is often vacuolar degeneration in the central cells of the lobules. On account of these characters rodent ulcer constitutes a special morbid entity, although it is included in the group of epitheliomas.

Rodent ulcer may easily be mistaken for *lupus* or *tertiary syphilitic ulceration*.

Papillary Epithelioma.—This form, which is very common, at first resembles simple hypertrophy of the papillæ, or common papilloma; later on, either spontaneously or from repeated irritation, it grows, and becomes indurated at the base and bleeds easily. It often undergoes exuberant growth, and develops vegetations which sometimes become covered with blackish crusts or horny formations.

This form of epithelioma, contrary to rodent ulcer, may occur on any part of the face; on the chin, on the nose, where it often assumes the vegetating form, on the upper and lower lip, on the temples and cheeks, on the forehead and scalp, especially in bald subjects. When situated on the eyelid, it may involve the conjunctiva; when situated on the nose, it may invade the vomer, perforate the cranium, and affect the meninges; when situated on the upper lip, it may invade the buccal mucous membrane, the alveolar border of the maxilla, and the hard palate.

Besides the face, papillary epithelioma may occur on any part of the body, especially the genital organs. It may develop on the glans penis, around the meatus, or on the prepuce. After a time, the tumour increases in size, the dorsal lymphatics of the penis become indurated, the inguinal glands are affected, and death supervenes in two or three years. It is more rare on the labia majora, still more rare on the trunk, umbilicus and nipple. On the limbs, it may develop on a patch of lupus.

After a variable duration, papillary epithelioma forms an ulcer with punched-out, everted, indurated borders, from which caseous masses enclosing epidermic globes can be expressed. In a few months or years, sometimes much more rapidly, the ulcer causes destruction of the subjacent tissues: muscles, cartilages and bones. In rare cases the whole growth is eliminated by necrosis. Sometimes cicatrisation takes place at one part, while the growth extends in other directions.

After a few months, or several years, the corresponding lymphatic glands become enlarged. Sometimes visceral metastases occur, followed by cachexia and death.

Deep Epithelioma.—This is rather rare, if we consider only those epitheliomas which develop from the glands in the deeper part of the skin. It forms a tumour of rapid growth, over which the superficial part of the skin eventually ulcerates. The evolution is the same as in the preceding form, but more rapid.

Pathological Anatomy.—According to Cornil and Ranvier, superficial epithelioma of the skin has the characters of pavement-celled lobular epithelioma, and deep epithelioma of the skin those of tubular epithelioma. In the first case, the growth arises from the deep layers of the epidermis; in the second, from the sebaceous

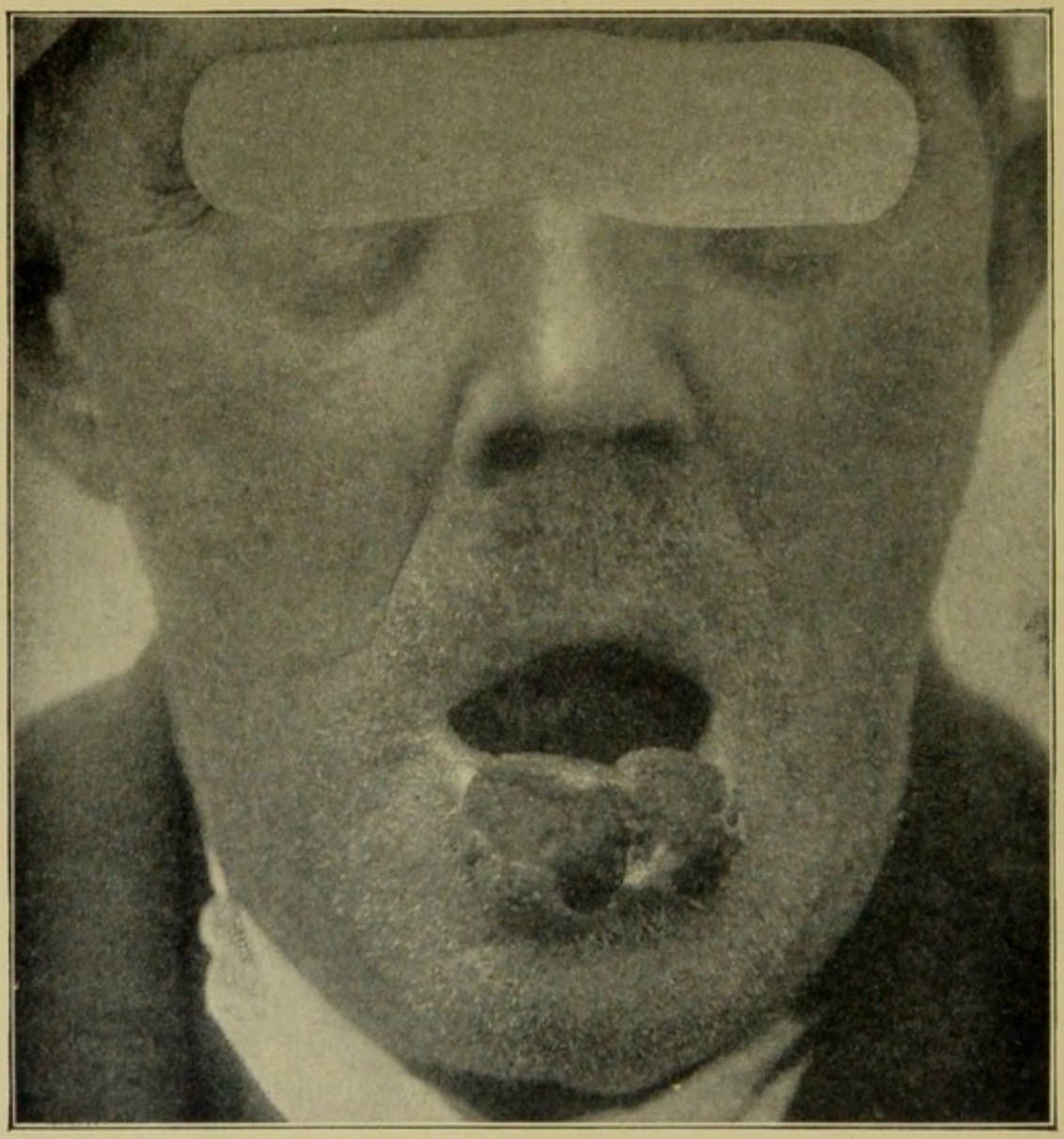


Fig. 108.—Epithelioma of the lower lip.

glands, hair follicles and sweat glands. Cornil has observed some cases of melanotic epithelioma, but this form is exceptional; melanotic tumours are generally sarcomas.

Diagnosis.—*Lupus* differs from epithelioma in the presence of small yellow tubercles situated around the ulceration; the latter does not bleed so easily, and there is less induration of the tissues.

The *ulcerated syphilide* is characterised by its slow evolution and the presence of other signs of syphilis. However, the vegetating form of epithelioma may easily be mistaken for vegetating lupus or a

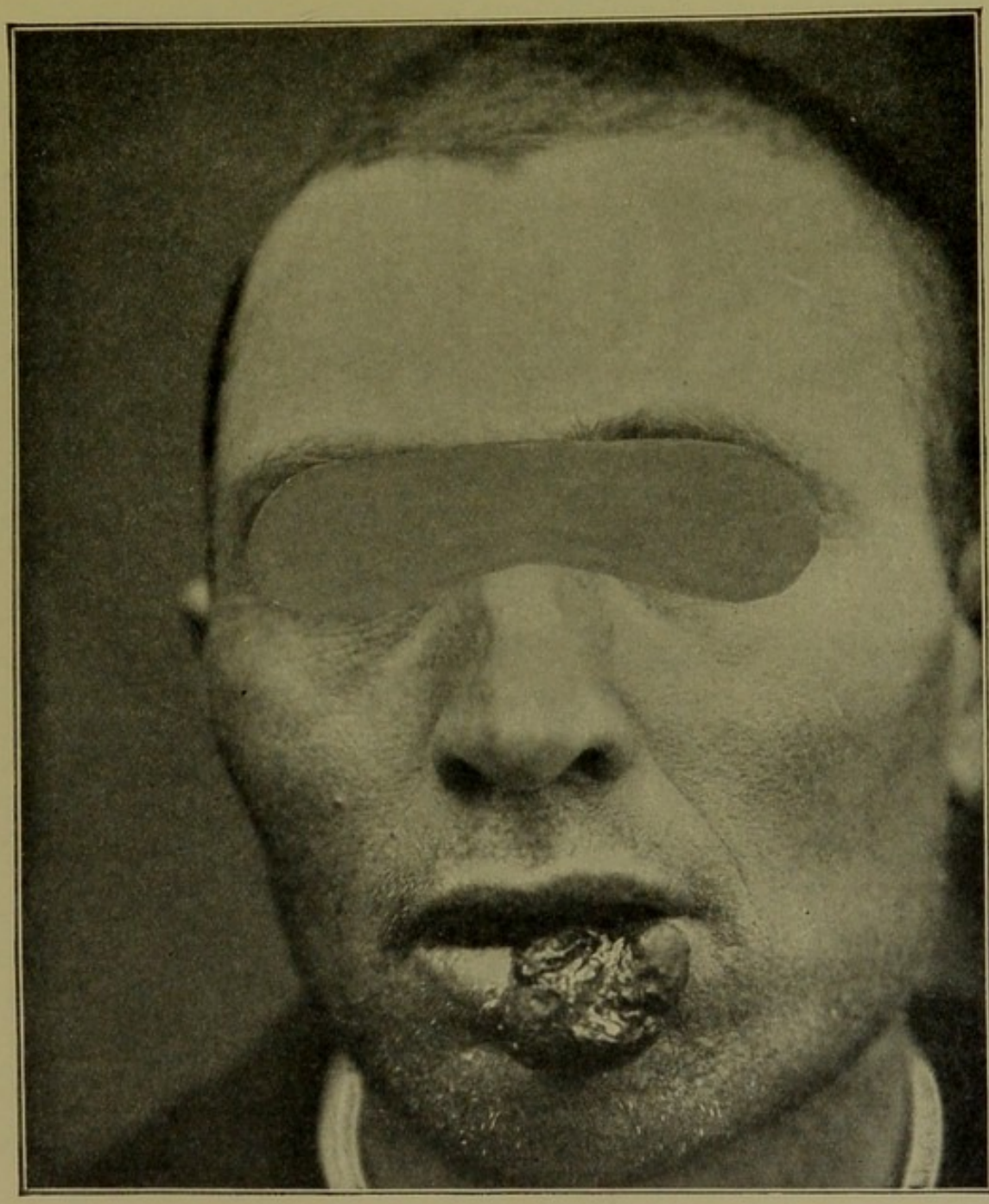


Fig. 109.—Ulcerated vegetating epithelioma.

vegetating syphilide. In its early stage, a papillary epithelioma of the penis might be mistaken for a chancre, a syphilide or lupus. It is sometimes difficult to distinguish acne sebacea, in process of epitheliomatous transformation, from simple seborrhœa.

Etiology.—Epithelioma is generally observed after the age of forty; it is more common in men than in women. The lesion may be single or multiple. The influence of heredity is probable.

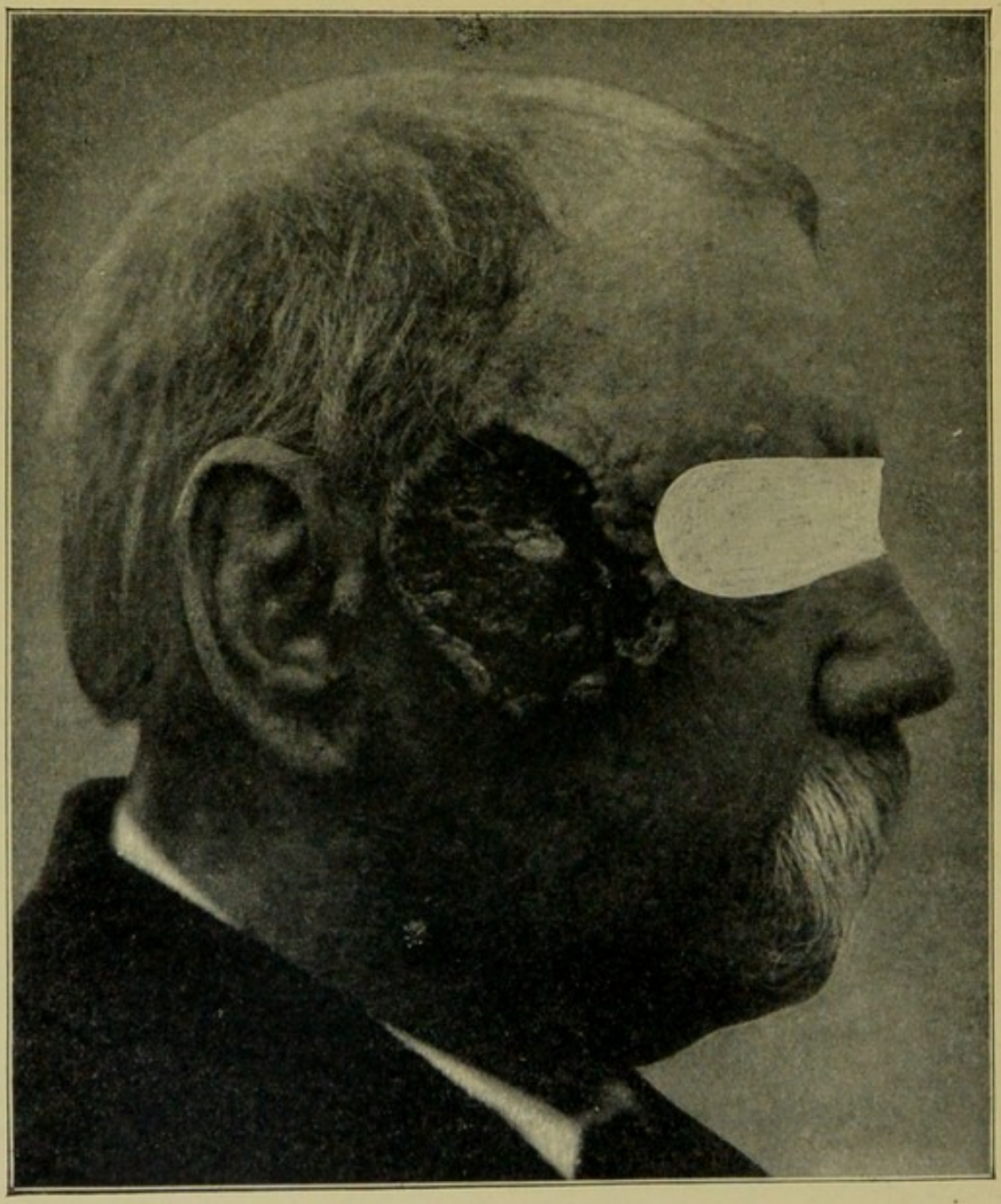


Fig. 110.—Epithelioma.

Frequent local irritation, the abuse of tobacco and the use of pipes with short stems, in the case of the lips, and certain occupations such as the manufacture of coal blocks and the purification of petroleum, seem to favour the development of epithelioma in pre-

disposed subjects. Cancer of the scrotum in chimney-sweeps has long been known.

It is not uncommon to see epithelioma develop on a nævus, or on a chronic varicose ulcer or patch of psoriasis. The sebaceous acne

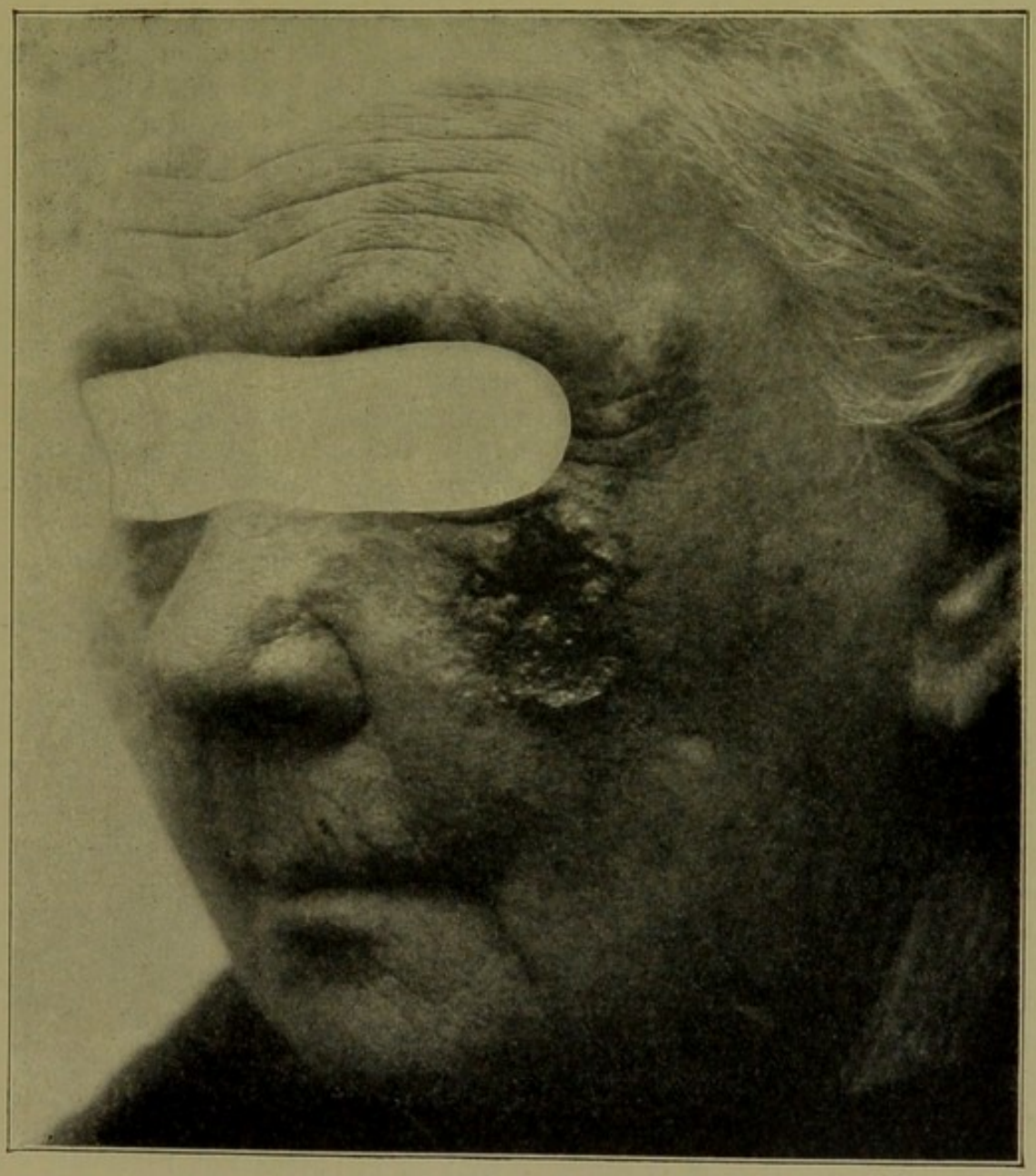


Fig. 111.—Ulcerated epithelioma.

of old people is only an epithelioma developed on a senile seborrhœic skin; underneath the adherent fatty crusts is formed a characteristic epitheliomatous patch, which bleeds when the crusts are detached. Epithelioma may also develop on senile seborrhœic warts. Sebaceous cysts (wens) may also undergo epitheliomatous degeneration. All

affections of the sebaceous glands predispose to epithelioma. *Xerodermia pigmentosum* is often complicated by epithelioma.

Epithelioma may develop on the cicatrices of ulcers, such as those of syphilis or lupus. This is not transformation of one diseased tissue into another tissue, as was formerly believed, but implantation of a new lesion on a pre-existing one. This is proved by the fact that one of the lesions can be cured, while the other continues to develop; for example, in the case of a hybrid lesion formed by cancer and syphilis, the syphilitic element of the lesion can at first be improved by antisyphilitic treatment, without modifying the evolution of the cancer.

Epithelioma may also develop on the cicatrices of burns and wounds; its progress is then slower than on the healthy skin, and the ulcerative form predominates over the papillomatous. It is sometimes very difficult to distinguish between a simple and epitheliomatous ulceration in a cicatrix.

We have already mentioned that epithelioma may develop on an old patch of lupus; in this case it is difficult to say whether the epithelioma developed on the lupus itself or on a cicatrix.

The transformation of buccal leucoplakia into epithelioma is well known; but this does not concern us here, as we are only dealing with cutaneous epithelioma.

The *nature of epithelioma* is still very obscure, in spite of numerous researches. Some observers have found corpuscles similar to those found in Paget's disease, and have regarded them as parasites (psorosperms); but more recent researches have shown that the corpuscles described as parasites in cancer and in Paget's disease are nothing more than products of cell degeneration.

Prognosis.—This is not so grave in rodent ulcer as in the other forms. The prognosis is grave in papillary epithelioma and still more so in the deep form, on account of the liability to recurrence, which is almost inevitable after removal by the knife. Epithelioma is more torpid in old people. In rare cases cutaneous epithelioma may be complicated by visceral deposits.

Treatment.—If we took the old name for epithelioma, *nole me tangere*, literally, we should never touch this lesion. This prohibition should only be applied to surgical excision, which is always followed by recurrence. But it is evident that it is better not to touch an epithelioma, even by caustics, than to destroy it incompletely and leave a part of the tumour, which will form a focus for recurrence. There are also cases of rapid evolution with early glandular invasion, in which all treatment is more or less illusory. Lastly, in persons of very advanced age, it is often wise not to touch an epithelioma. With these exceptions, every epithelioma should be operated upon, not by the knife, but by one of the methods to be described.

In old people with seborrhœa, prophylactic measures should be taken against the development of epithelioma, by attention to the hygiene of the skin and the use of lotions, such as an alcoholic solution of boric acid.

There are seven principal methods of treatment which may be used in cutaneous epithelioma: (1) cauterisation; (2) caustics; (3) curetting; (4) the electric spark; (5) radiotherapy; (6) radium; (7) fulguration.

Cauterisation.—This may be done either with the galvano-cautery or the thermo-cautery. The best method is to destroy the whole tumour and the parts for some distance beyond it with the blade of the thermo-cautery. A local anæsthetic may be used in nervous patients, but is not necessary. After stopping hæmorrhage by aseptic wool, the wound is covered with powdered chlorate of potassium, which is left on as long as the patient can endure it, and is then replaced by ordinary moist dressings. As this application is painful, cocaine may be applied beforehand. During the next few days the wound is bathed several times a day with a solution of chlorate of potassium (6 per 100), or chlorate of magnesium (10 to 20 per 100), and then covered with an ointment of chlorate of potassium (20 per 100) or chlorate of magnesium (10 or 20 per 100), which is left on permanently; or with powdered chlorate of potassium, covered with wool, which is only left on for an hour or two, on account of the pain. When chlorate of potassium cannot be endured, the wound may be treated by tepid sprays of boiled water and moist dressings. When the wound begins to granulate, chlorate of potassium may be replaced by aristol, but the continuation of the former is preferable. If any of the tumour remains after this treatment, it must be cauterised again and dressed with chlorate of potassium as before. This mode of treatment, consisting in the combination of cauterisation with chlorate of potassium, which I have employed successfully during the last fifteen years, is, in my opinion, the best treatment for cutaneous epithelioma. In cases of small superficial epitheliomas, I use the thermo-cautery only, without chlorate of potassium, followed by daily spraying with boiled water and moist dressings.

Caustics.—One of the best of these is the following:—

Arsenious acid	2 parts
Sulphide of mercury	6 „
Calcined sponge	12 „

The crust is first removed with a poultice, and the tumour covered with a little of this caustic made into a paste with water, and a piece of amadou (German tinder) applied over the paste. In a week or a fortnight the tumour comes away with the amadou, and the wound

is dressed with chlorate of potassium. In large tumours this caustic is dangerous, on account of possible arsenical poisoning.

Other caustics which have been used are Vienna paste, chloride of zinc, lactic acid, acetic acid, resorcin, pyoctanin, and picric acid. But none of these caustics are as good as the above, or cauterisation combined with chlorate of potassium.

Curetting.—This method is very inferior to cauterisation, and more liable to be followed by recurrence; it is also very painful. All friable tissue is removed by the curette, hæmorrhage is stopped by wool pressure, and chloride of zinc is applied to the wound. In a few days the wound is dressed with wool soaked in chlorate of potassium.

The Electric Spark.—This method has been successfully employed in small cutaneous epitheliomas, especially in the papillary form. Apart from its destructive action, this method has the advantage of promoting cicatricial repair. The procedure consists in riddling the diseased tissue or ulceration with small, very short sparks. A naked electrode is connected with a high frequency resonator regulated to near the minimum. The application is not very painful, and the first sparks produce slight anæsthesia.

In *rodent ulcer*, cicatrisation can sometimes be obtained with chlorate of potassium alone, but only in the case of ulceration. The crusts are first removed with a concentrated solution, and the ulcer is then covered with chlorate of potassium, in the form of ointment or powder. When this causes much irritation, it may be replaced for a time by boric acid dressings. But in most cases this treatment by itself is insufficient, even in rodent ulcer, and it is generally necessary to use the thermo-cautery.

To sum up, the best treatment for cutaneous epithelioma is *cauterisation*, whenever this is possible. When cauterisation is impossible, on account of the extent or depth of the epithelioma, much improvement can be obtained with chlorate of potassium; before using this it is well to cauterise the exuberant parts of the growth.

The internal administration of chlorates of potassium or sodium has been tried without any benefit. I have tried chlorate of magnesium, the use of which is more logical, but without any definite result.

In the treatment of extensive or deep epitheliomas, *radiotherapy*, *radiumtherapy* and *fulguration* must now be discussed.

Radiotherapy.—It is necessary to deal with radiotherapy at some length, because it is the treatment in vogue, but it does not appear to me to be notably superior to the older methods of treatment for cutaneous epithelioma.

The X-rays possess a destructive action on superficial epitheli-

omas of small extent; they cure in the same way as cauterisation, but much more slowly, and with the danger of radiodermatitis. They have no curative action on deep tumours; sometimes they even lead to more rapid growth of the neoplasm.

Having made these reservations, I shall now describe (1) the principles and general technique of radiotherapy in cutaneous therapeutics; (2) the treatment of cutaneous epithelioma by the X-rays.

Principles and technique of radiotherapy.—The X-rays are not only invisible rays capable of acting on photographic plates, of making fluorescent bodies luminous, and of traversing opaque bodies; they also have a stimulating and destructive action on the tissues. The effects on the skin do not appear during exposure to the rays nor immediately afterwards; but a week or a fortnight, sometimes several months, after exposure.

Physiological researches have proved:—

(1) That the X-rays in small doses cause stimulation, and in large doses destruction of cells; (2) that they have an elective action on cellular elements, principally on the Malpighian layer and the horny layer of the epidermis; (3) that they are capable of diminishing the virulence of certain cultures of microbes, and even of arresting their development.

The apparatus necessary for the production of X-rays includes (1) a source of electricity; (2) a Crooke's tube.

The source of electricity, which must be of high tension, may be either (1) a powerful static machine worked by an electric motor; (2) a powerful induction coil, which may be worked by a battery, by accumulators, or from the electric-light supply; (3) or a transformer with closed magnetic circuit transforming the current of 110 to 220 volts, used for house illumination, into currents of high voltage.

The *Crooke's tube* is a glass bulb, in which a vacuum is made, containing two electrodes; one, concave, made of aluminium, from which the cathode rays are emitted; the other, flat, made of platinum, which receives the cathode rays and transforms them into X-rays; this is called the anticathode.

The cathode rays which strike the anticathode are not all transformed into X-rays; a good many are reflected by the platinum plate on to the glass of the tube, which becomes fluorescent under the action of these rays. This reflection of cathode rays takes place in all directions, but only below the level of the anticathode, so that only one-half of the tube is illuminated. Every point of the tube struck by the reflected cathode rays gives rise in its turn to X-rays; hence the source of X-rays in the tube is twofold, viz., the cathode and the wall of the tube.

The rays emitted by a tube differ in power of penetration according to the intensity of the vacuum, which renders the tube

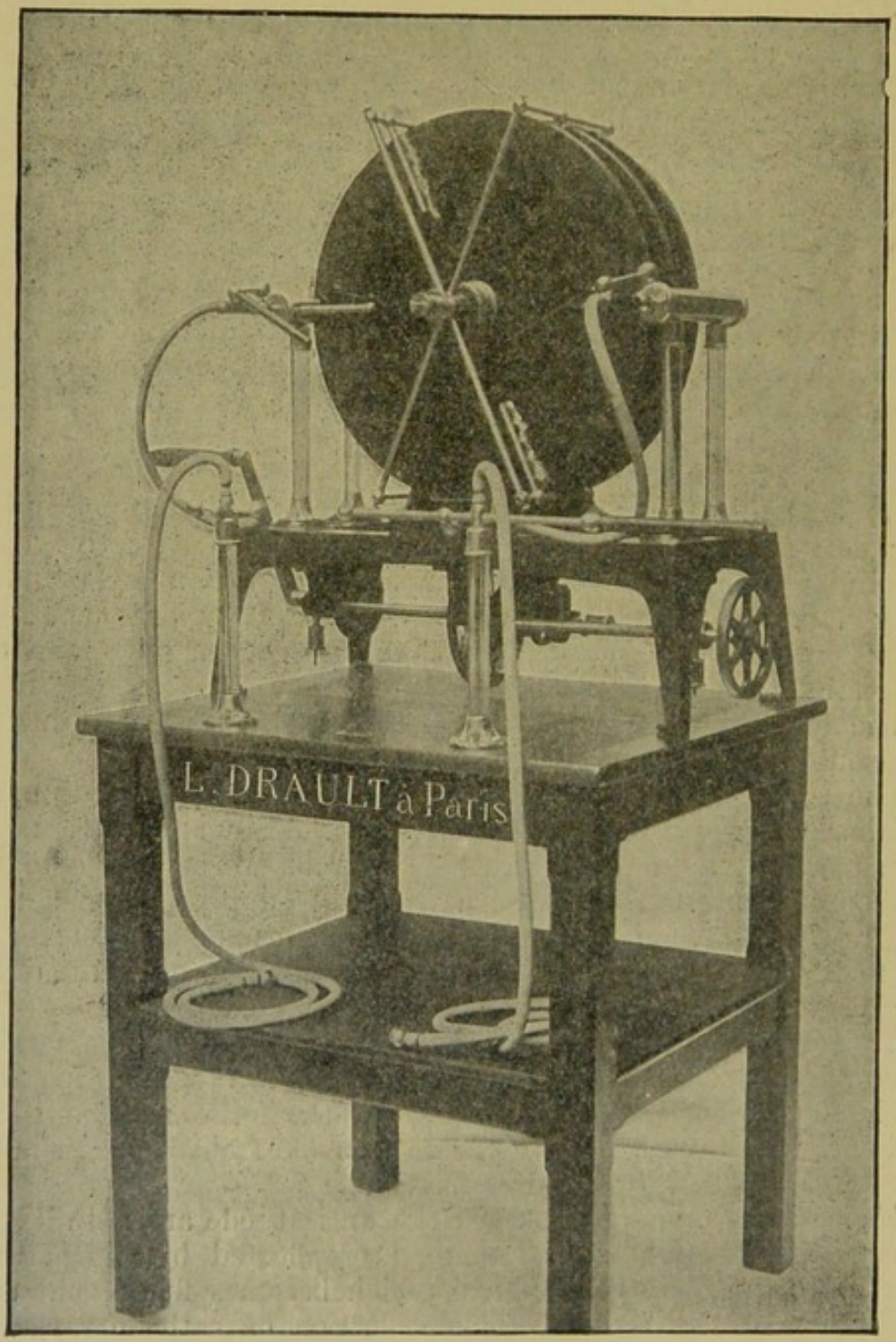


Fig. 112 – Static machine with multiple plates.

more or less resistant to the passage of the electric current. Tubes which give X-rays of little power of penetration are called *soft tubes;* those which give X-rays of moderate power of penetration

are called *medium tubes;* those which give rays of great power of penetration are called *hard tubes.*

The feebly penetrating rays are almost entirely absorbed by the first layers of tissue which they encounter, and consequently have a harmful action on the healthy skin. The strongly penetrating rays, on the contrary, are scarcely at all absorbed by the tissues they traverse, and have very little action on the skin.

The fact that the strongly penetrating rays traverse the skin without absorption, does not imply that a hard tube is harmless for

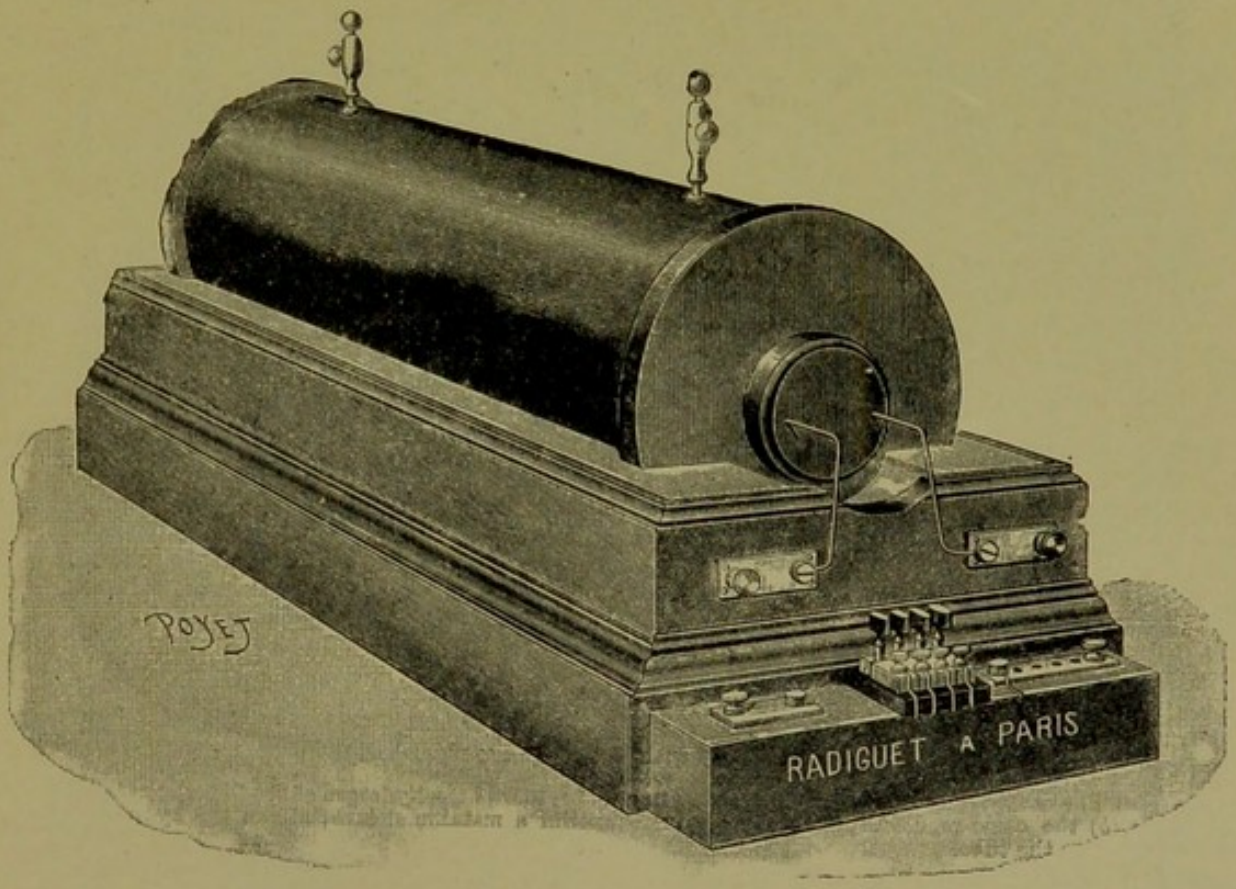


FIG. 113.—Induction coil.

the skin. In fact, a hard tube, besides the strongly penetrating rays, always gives out a small quantity of less penetrating rays, which are absorbed by the skin, and may ultimately have the same effect as rays from a soft tube.

In radiotherapy, medium tubes are often used; *i.e.*, tubes which give out rays capable of affecting all the layers of the skin.

Originally, it was necessary to use a different quality of tube for each kind of rays; it was also necessary to renew the tubes, for they become hardened by use, and consequently give out rays of greater penetrative power. But, thanks to the invention of the osmo-regulator by Villard, we can now vary the degree of vacuum

in a tube, and make it soft or hard at will. The osmo-regulator consists of two parts: (1) a thin platinum tube sealed to the Crooke's tube, with its lumen opening into the cavity of the latter; (2) a movable platinum sheath by which the fixed platinum tube can be covered. If the platinum tube is heated without its sheath, the hydrogen of the flame penetrates the Crooke's tube by osmosis, and renders it softer. If the platinum tube is heated while covered with its sheath, till the latter becomes red hot, the hydrogen escapes from the Crooke's tube, and renders it hard. In practice, it is rarely

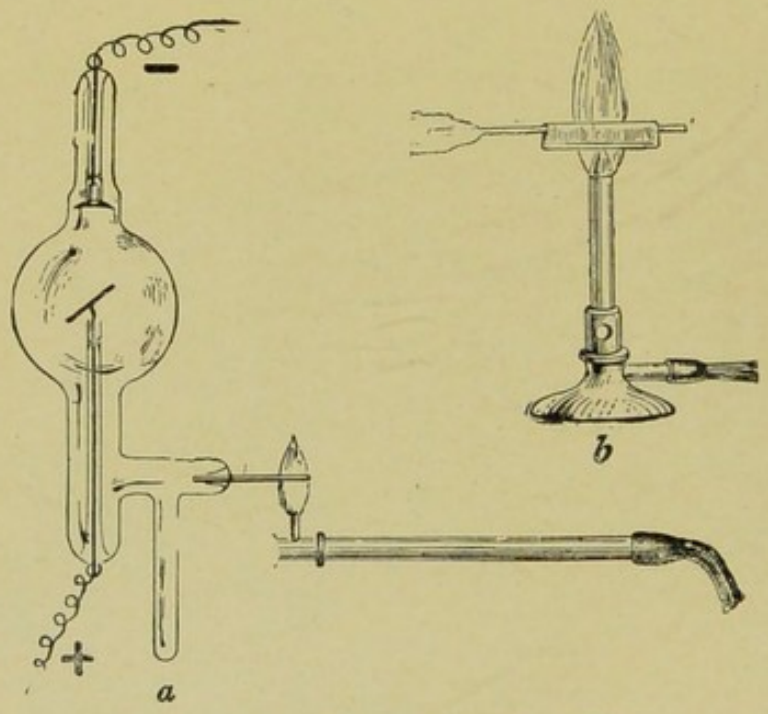


FIG. 114.—Tube with Villard's osmo-regulator.

(*a*) The osmo-regulator, heated to redness in a flame, allows the hydrogen of the flame to enter the tube; (*b*) the osmo-regulator, heated to redness within a metallic sheath, allows the hydrogen to escape from the tube.

necessary to harden the tube, but nearly always necessary to soften it, as it hardens by use.

In order to determine the *quality of the rays*, or their power of penetration, in other words, to determine the degree of hardness or softness of the tube, two instruments have been devised: the *spintermeter* of Béclère, and the *radiochromometer* of Benoist.

The *spintermeter* indicates the degree of hardness or softness by measuring the length of spark passing between two conductors placed in the circuit of the Crooke's tube. This spark is called *equivalent*, for its length is equivalent to the resistance offered by the tube to the passage of the current.

The spintermeter is nothing more than a discharger, one of the electrodes of which is movable and slides in a short tube, and can

be placed at various distances from the other electrode. When the spintermeter is interposed in the circuit of the Crooke's tube, the current can pass by one of two ways, either through the tube in the form of a vacuum discharge, or through the air in the form of a spark.

To determine the quality of a Crooke's tube by means of the spintermeter, the two points of the discharger are first put in

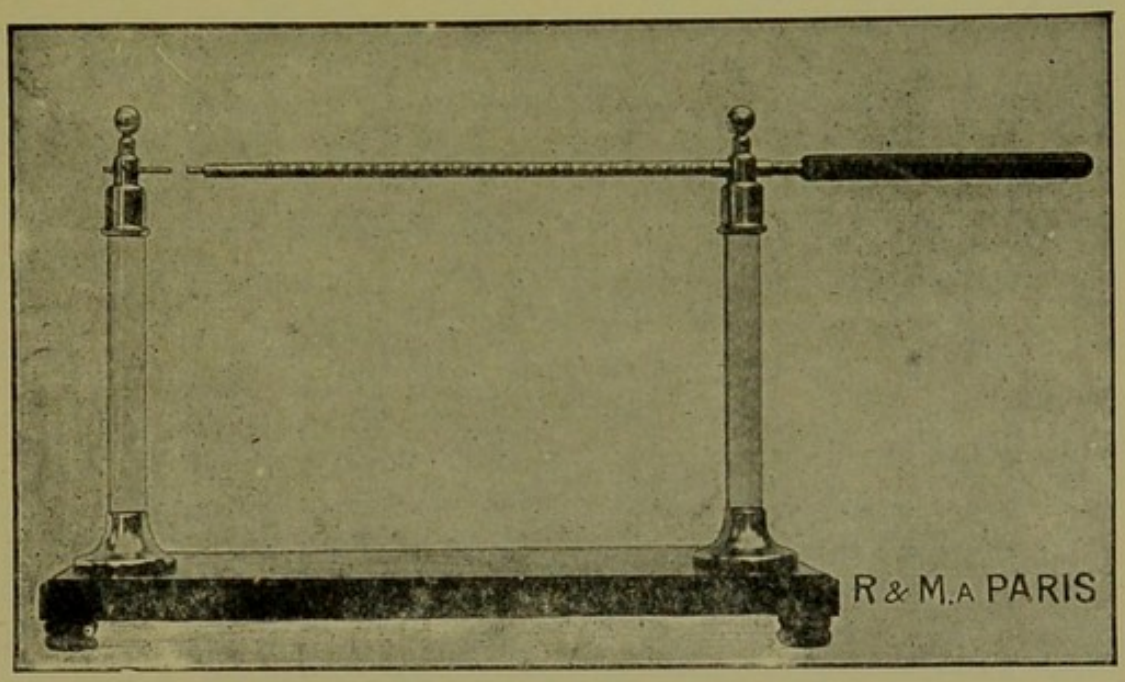


Fig. 115.—Béclère's spintermeter.

contact, and then slightly separated. A continuous spark is then produced, because the current always chooses the path of least resistance, which in this case is the air. As the extremities of the conductors are more and more separated, a point is reached when the spark becomes intermittent, or is no longer produced, while the tube becomes illuminated, indicating that the current finds less resistance in the tube than in the air between the conductors. The harder the tube, the longer is the spark, the resistance of the tube being greater than that of a thick layer of air. Inversely, the softer the tube, the shorter is the spark. At the point when the spark is no longer produced, the number of degrees marked on the movable rod is read; this number measures the distance the movable rod has been withdrawn, and the length of the equivalent spark.

The *radiochromometer* of Benoist is based on the unequal transparency of different metals to rays of the same power of penetration. If a metal of known density and thickness is taken as a standard, it is clear that another metal of different density must be of a certain

thickness to be traversed in the same way, and give the same shadow on a fluorescent screen as the metal used as a standard.

The radiochromometer consists of a disc, four or five centimetres in diameter, the centre of which is formed by a circular plate of

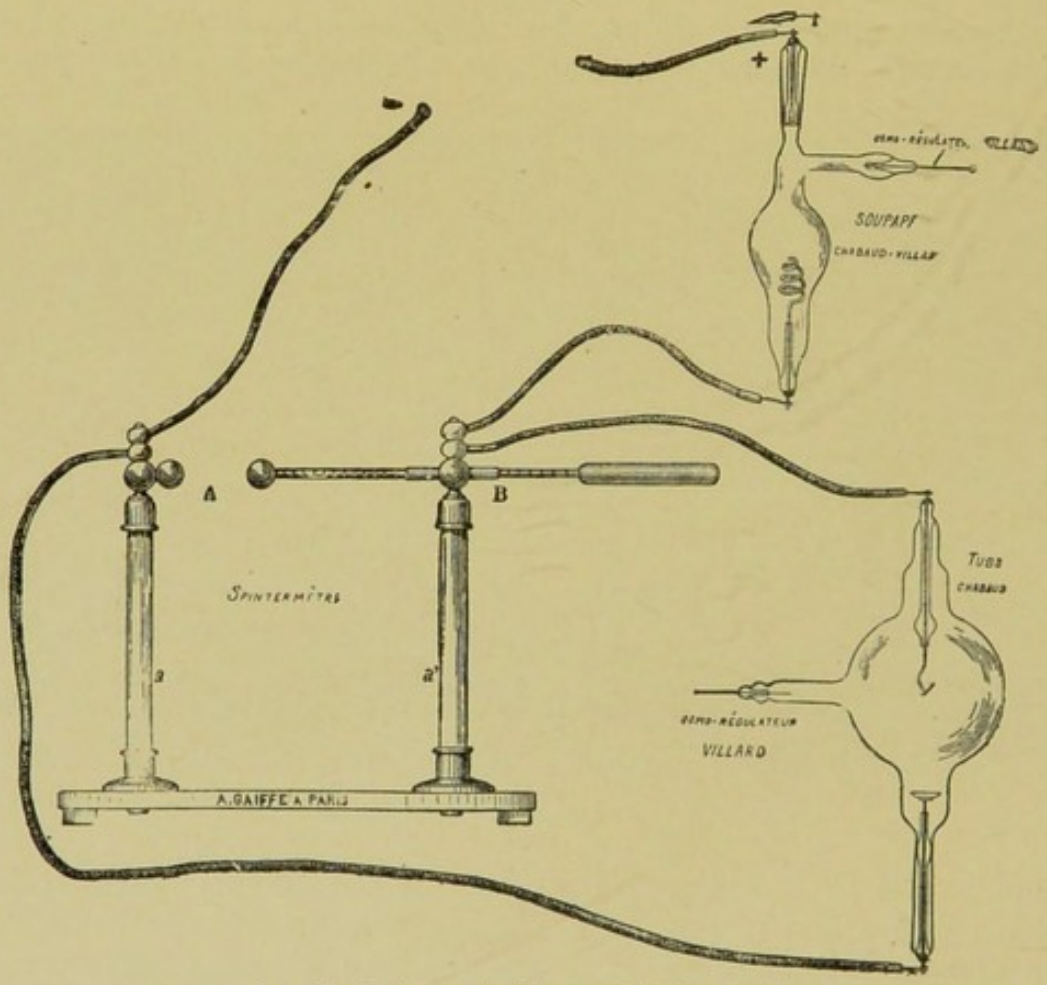


FIG. 116.—Spintermeter interposed in the circuit.

silver $\frac{1}{10}$ of a millimetre thick. This silver plate is surrounded by a series of twelve aluminium sectors, arranged in a scale, increasing in thickness from 1 to 12 millimetres (Fig. 117). The instrument is applied behind the black surface of a fluorescent screen placed before the tube to be tested. It is then determined which sector gives the same shadow as the central silver disc on the fluorescent screen. If, for example, it is found that sector No. 4 gives the same shadow as that of the silver disc, the rays furnished by the tube are called No. 4 rays. Nos. 4 and 5 correspond to soft tubes; 6 to medium tubes; 7 and upwards to hard tubes.

It is equally important to know the *quantity of rays* absorbed in a given time. This may be estimated in two ways.

The first, or indirect method, consists in interposing a *milliampere-*

meter in the circuit of the tube. If the number of milliamperes and the time during which the rays have acted are known, we have an approximate estimate of the radiation of the tube, for a given distance from the skin, and for rays of known quality.

But the activity of a tube, all other conditions being the same, varies with its resistance, that is, with its degree of hardness; the indications of the milliamperemeter are only comparable for rays of the same quality. Again, varieties in the construction, form and dimensions of tubes render these indications only of service for the comparison of absolutely identical types of tubes. Thus, ever since radiotherapy was instituted, attempts have been made to measure the X-rays directly, instead of measuring the electric elements used to produce them.

For this purpose, three kinds of apparatus have been devised. Some interpose the variations of resistance of selenium under the

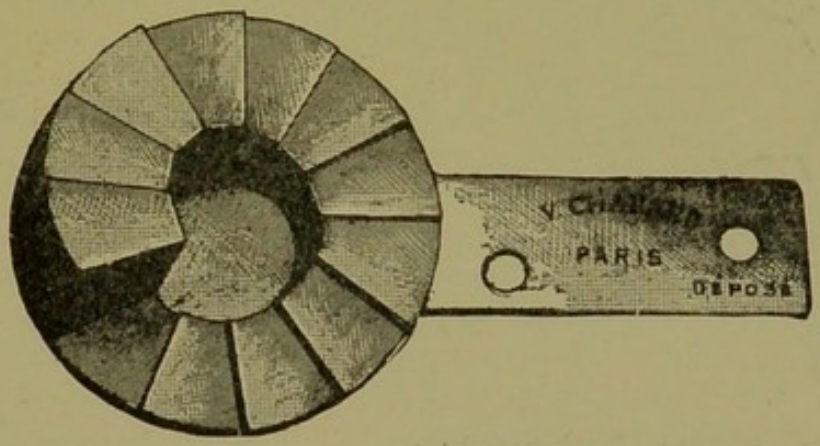


Fig. 117.—Benoist's radiochromometer.

influence of the rays; others, called fluorometric apparatus, compare a plate made fluorescent by the X-rays to be measured, with a standard fluorescent plate illuminated by a fragment of radium salt; others again, which are most generally used, are based on the well-known phenomenon of the change in colour of certain substances under the action of the X-rays.

The first apparatus of this kind was Holzknecht's *chromoradiometer*. This is now seldom used, for its indications are uncertain; but it has served to establish a term of comparison, incorrectly called a unit—*the unit H of Holzknecht*—which, in the absence of a unit of physical measure, gives a rough posological estimate. I shall not describe Holzknecht's apparatus (Fig. 118), nor those of Freund or Schwarz, because in France and Germany at any rate, the *Sabouraud-Noiré reaction* is used in preference.

This reaction is the practical application of what is known as Villard's reaction, this physician being the first to show the changes

in colour of *platino-cyanide of barium* under the influence of the X-rays. This salt, when exposed for a sufficient time to the X-rays, changes to bistre, then to light brown. This action seems to be proportional to the quantity of rays absorbed. Sabouraud and Noiré made use of this property to fix a tint in the scale of colour-changes of platino-cyanide which cannot be exceeded without risk of producing radiodermatitis. The maximum tint determined by Sabouraud and Noiré corresponds to about 5 of Holzknecht's units.

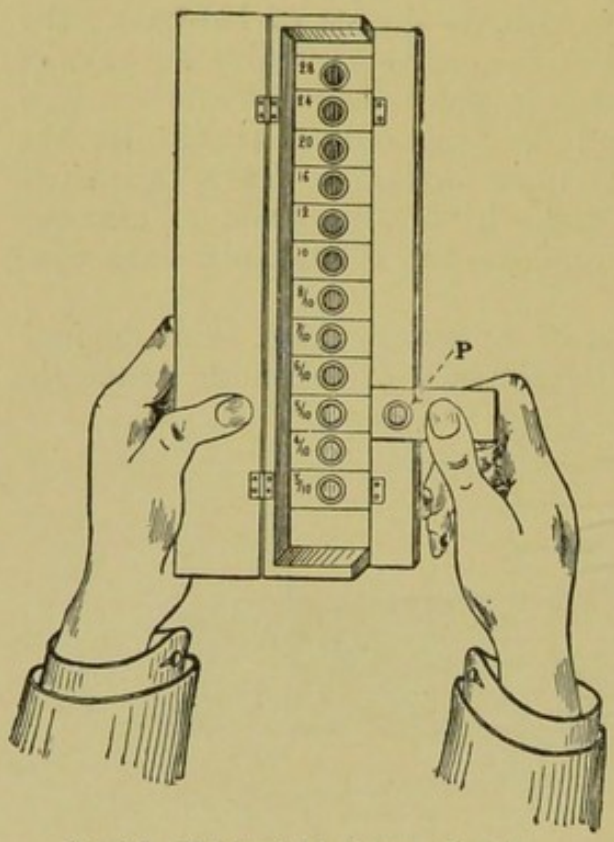


FIG. 118.—Holzknecht's chromoradiometer.

The reagent consists of a small pastille, formed by a thin layer of platino-cyanide fixed on a paper disc. The pastille is placed midway between the anticathode and the part treated, and the change of tint is compared from time to time with that of a standard pastille. This reaction is open to criticism, but it constitutes a practical method of determining the condition of a tube.

Treatment of epithelioma by radiotherapy. — The following technique is used in my clinic for the treatment of epithelioma by the X-rays.

Moderately penetrating rays are used, 7 to 10 of Benoist's radiochromometer. It is sometimes of advantage to prepare the lesion by scraping exuberant vegetations and rendering the lesion as aseptic as possible. The surrounding parts are protected by a localisator. The lesion should be about 15 or 20 centimetres from the anticathode. The duration of each sitting depends on the quantity of rays to be absorbed; at the first sitting, it is usual to commence with a quantity approximately equivalent to 3 or 4 H (3 or 4 Holzknecht's units). When a single sitting is not sufficient, the treatment is repeated under the same conditions, but not before six or eight days.

When the quantity of rays absorbed has been sufficient to cure the epithelioma, the latter undergoes retrogression after an interval of three or four weeks, and gradually cicatrises.

But the most ardent partisans of radiotherapy recognise that the X-rays are not always successful in the treatment of epithelioma. Some epitheliomas are rapidly modified by radiotherapy, while others remain unaffected. In the results published, it is necessary to take into account the age of the tumour, its surface, extent and depth, its situation as regards accessibility to the action of the rays, and also the technique employed, which varies with different operators.

In short, radiotherapy may be used in certain cases on the chance of a successful result, provided it is not employed too late; but we must not have too much confidence in this method.

Radium.—Radium is much more important than radiotherapy in the treatment of epithelioma of the skin and mucous membrane, and this method has given such remarkable results that I have asked Dr Domenici to give a full and detailed account of it.

Radiumtherapy (by Domenici).—Of the various modes of application of radium for the treatment of cancer of the skin, one only need now be described. This consists in submitting the tumours to the action of inexhaustible and permanent apparatus.

I shall explain successively the physical characters to which radium owes its therapeutic action in malignant growths, the technique by which this action can be made use of, and the method of adapting it to the treatment of cancer of the skin.

Radiation.—The salts of radium are the source of a spontaneous and continuous emission of rays, which are designated by the Greek letters α, β and γ. This property of spontaneous and continuous radiation possessed by the salts of radium is the phenomenon of *radio-activity*, discovered by Becquerel, and established first for uranium.

The alpha and beta rays are composed of mobile material particles, the alpha rays being charged with positive electricity, the beta rays with negative electricity. These rays are corpuscular, and are quite different from the gamma rays, which are vibratory. The latter are not produced like the others by the projection of material particles through space, charged with positive or negative electricity, but by a disturbance in the ether; so that the gamma rays resemble the X-rays. But the gamma rays have a greater power of penetration than X-rays, and may be compared to X-rays produced by an extremely hard tube. The gamma rays are also the most penetrating of all the radium rays.

In classifying the rays according to their power of penetration, the gamma rays come first, and the alpha rays last. Between these come the beta rays, which are subdivided into—(1) soft beta rays; (2) hard beta rays, more penetrating than the soft and less penetrating than the gamma rays; (3) medium beta rays.

In order to make these characters more clear, I may mention that an aluminium screen $\frac{3}{100}$ or $\frac{4}{100}$ of a millimetre thick stops all the alpha rays; a sheet of lead at least $\frac{2}{5}$ of a millimetre thick is required to stop all the beta rays; while the gamma rays will pass through sheets of lead more than 10 centimetres thick.

Ionising power of radium. Measure of radio-activity.—However dissimilar may be the different rays, they possess the common property of ionising the air; that is, of decomposing the atoms of its different gases into ions, electrified positively or negatively.

In ionising the air, the rays make it a conductor of electricity; they have, in fact, the property of discharging electrified bodies. But the rapidity of discharge is proportional to the intensity of radiation. Hence, it is possible to measure the intensity of radiation of apparatus containing radium by means of electroscopes adapted for this purpose. The radio-activity of radium is usually compared with that of uranium; if the radio-activity of uranium is taken as a unit, that of pure radium is 2,000,000.

Apparatus.—The first apparatus used for radiumtherapy were glass tubes containing bromide or sulphate of radium. These have now been replaced by apparatus on which the radium salt (bromide, or more usually sulphate) is applied in the form of a varnish. These differ in details, but have the same principle of construction.

The salts of radium are used in the pure state, or mixed in different proportions with a salt of barium (sulphate or bromide). The varnish containing the powder is spread on metal or linen. Some apparatus consists of discs or square sheets of metal covered with the varnish containing radium; others are in the form of a rod, the end of which is bulbous, oval, cylindrical, or spatulate. The different forms of these apparatus can be applied to flat or convex surfaces, or introduced into passages such as the external auditory, or insinuated between the eyelids and globe of the eye.

The second form of apparatus consists of discs or squares of lint, covered on one side by the radium varnish and enclosed at the borders in a metal frame; these can be adapted to the shape of the affected regions.

There is an important difference between these two forms of apparatus. In the metal apparatus, the radium salt is generally completely imbedded in the varnish; while, in the linen apparatus, the grains of radium salt project from the surface of the varnish which fixes them to the linen.

Designation of apparatus.—In the designation of an apparatus we note: (1) the activity (intensity of radiation) of the radio-active powder, which may be a pure radium salt or a mixture with a barium salt; (2) the weight of the powder.

In the case of pure sulphate or bromide of radium the theoretical

activity is 2,000,000. In the case of a mixture of radium and barium salts, the activity of the mixture is said to be inversely proportional to the quantity of the barium salt. Thus, the activity of an apparatus containing equal parts of sulphate of radium and sulphate of barium is taken as equal to 1,000,000; the activity of an apparatus containing one part of radium salt to three of barium is taken as equal to 500,000, and so on.

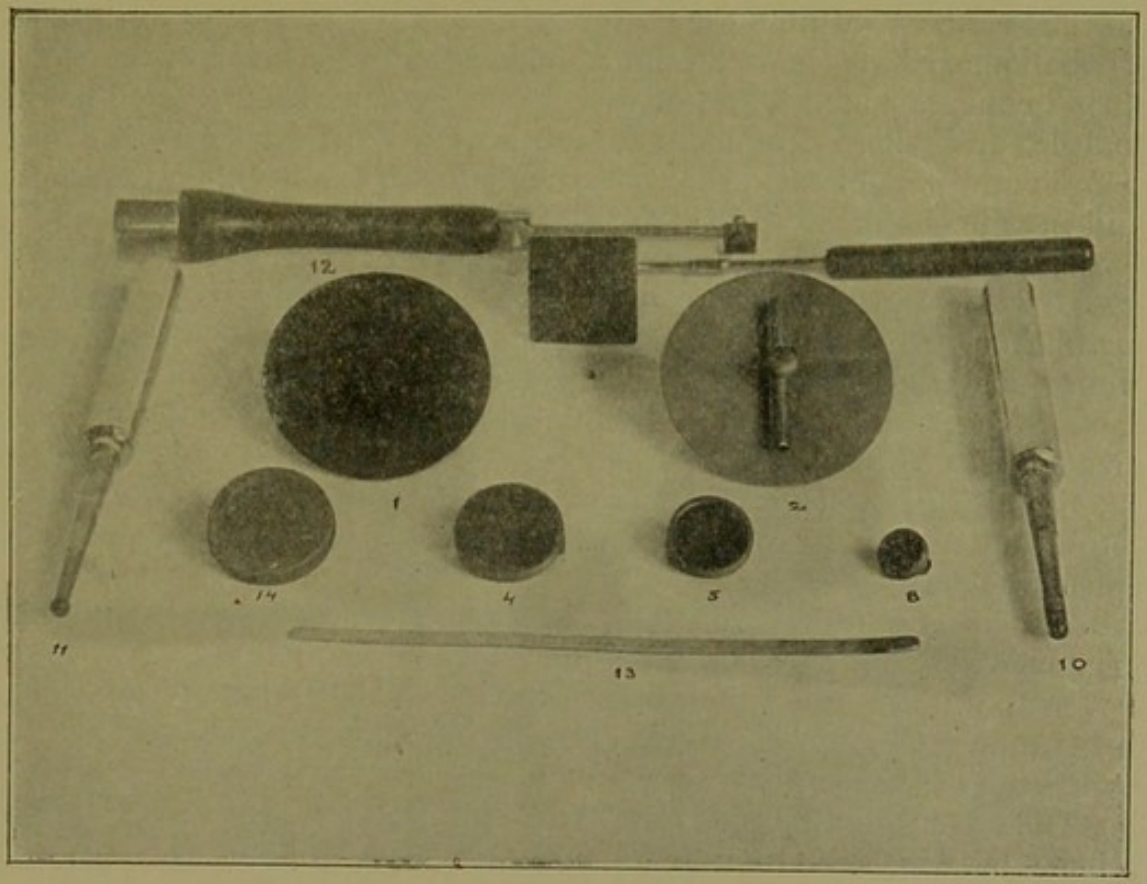


FIG. 119.—Apparatus for radiumtherapy (reduced to a third size), after Wickham and Degrais.

1 and 2. Circular plates. 3. Square plate with rounded angles. 4, 5, and 8. Circular plates. 10. Cylindrical apparatus. 11. Spherical apparatus. 12. Square apparatus. 13. Elongated apparatus.

In the treatment of cancerous tumours, apparatus of 500,000 activity are used.

But this theoretical activity must be carefully distinguished from the effective activity, that is, the utilisable radiation furnished by the apparatus. The latter varies with the quantity of radium salt per unit of surface, and also according to the manufacture of the apparatus.

To simplify this point, I may say that the apparatus most commonly employed contain from 4 to 10 milligrams of powder of 500,000 activity. As this powder contains one-quarter sulphate of radium and three-quarters sulphate of barium, it follows that the

apparatus only carries 1 to 2·5 milligrams of sulphate of radium per square centimetre.

Therapeutic application. — The apparatus above described give excellent results in the treatment of most cancers of the skin. Since the first success obtained by Danlos, we know of numerous observations concerning the resolution of cutaneous epitheliomas, not only cases which are curable by other methods, but tumours which have resisted other forms of treatment.

The cancers suitable for radium treatment include not only small epitheliomas, but those 20 or 30 square centimetres in area; also torpid cancers which take ten years to cause superficial erosion of the temple, and those which, in a few months, invade the forehead, temple and part of the cheek, burrow holes in the walls of the skull, and invade the orbit and side of the nose.

The situation of these tumours is of comparatively little importance, with the exception of regions which border on mucous membranes, especially the buccal mucous membrane. This region would be debarred from radium treatment, if it had not been rendered accessible by one of the technical processes which I shall mention later on.

From the histological point of view the tumours which undergo resolution under the influence of radium are plasmodial embryonic epitheliomas.

Therapeutic technique.—The treatment of cancers of the skin may be carried out by two different methods—

In the first method they are submitted to the action of the alpha, beta and gamma rays, or the beta and gamma, furnished by the apparatus described above.

The second method consists in stopping all the alpha rays, almost all the beta rays, and part of the gamma rays, so as to use only the *ultra-penetrating rays,* viz., the ultra-penetrating gamma rays and the hardest of the beta rays, which are the same, from the therapeutic point of view, as the gamma rays.

Treatment by composite radiation.—This method consists in the application of the apparatus to the tumour, either directly, or with the interposition of a thin sheet of india-rubber, to protect the surface of the apparatus against exudation from the tumour. A fraction of the alpha rays is absorbed by the protecting sheet, but the rest of the rays pass through it.

The apparatus may be used in two different ways:—(1) The so-called dry method of Danlos, which consists in short and frequently repeated applications. This produces resolution of the diseased tissue without external destruction. (2) The destructive method, in which the applications are of long duration, and cause extensive destruction of the morbid tissue, followed by ulceration.

The first method gives excellent results in the case of small tumours. It is also suitable for cancers implicating the palpebral conjunctiva, which are treated by means of flat metallic blades introduced between the tumour and the globe of the eye.

The second method is more generally used, and gives very good results in many cases. It consists in the application of apparatus of 500,000 activity, which is left in place for seven to ten hours. The treatment is usually carried out in two or three sittings at intervals of several days; but one sitting should be enough.

According to Wickham and Degrais, a cancer of the skin exposed for this time to the action of an apparatus containing from 5 milligrams to 1 centigram of powder, of 500,000 activity (1·25 to 2·5 milligrams of sulphate of radium per square centimetre), undergoes an intense reaction, followed by sloughing after about six weeks, and finally by perfect cicatrisation in eight to ten weeks.

Before applying the apparatus, the tumour should be cleansed and all crusts removed. The healthy skin round the tumour is protected by sheets of lead covered with india-rubber and enclosed in linen, cut so as to leave the tumour exposed and protect the adjacent skin. The apparatus is kept in place by means of strips of plaster or gauze bandages. After the applications of radium, the lesion is treated on ordinary aseptic principles.

This method of radiumtherapy, which gives remarkable results in cutaneous epitheliomas, is dangerous for cancers which encroach on the buccal mucous membrane. Gaucher has also pointed out that caustics, which are suitable for the treatment of cutaneous cancers, are contra-indicated for cancers of the mucous membrane and para-mucous cancers, as they stimulate rather than arrest their development.

But these cancers of cutaneo-mucous regions become amenable to radiumtherapy when the method of *filtration* is employed which I introduced in 1907, and tried at the St Louis Hospital in Professor Gaucher's clinic. This method consists in filtering the radium rays so that none but the *ultra-penetrating* rays are used.

Filtration.—Several practitioners have attenuated the intensity of the rays, either by placing the apparatus at a distance from the skin, or by covering it with aluminium; but filtration of the radium rays was purely empirical till I established the rules in a methodical manner. The method which I employ is based on the following principles: (1) the least penetrating rays cause most alteration in the healthy tissues; (2) the most penetrating rays, that is the least alterative, have an extremely powerful therapeutic action; these include the gamma rays and the hardest beta rays.

This method is carried out with one or more apparatus of 500,000 activity. These are enclosed in a lead capsule, from half

to several millimetres in thickness. Outside this is another envelope made of several sheets of paper, forming a sheath from one to several millimetres thick. The whole is then covered with an envelope of india-rubber. The lead lets through the ultra-penetrating rays (the gamma rays and the hard beta rays). The paper serves to stop

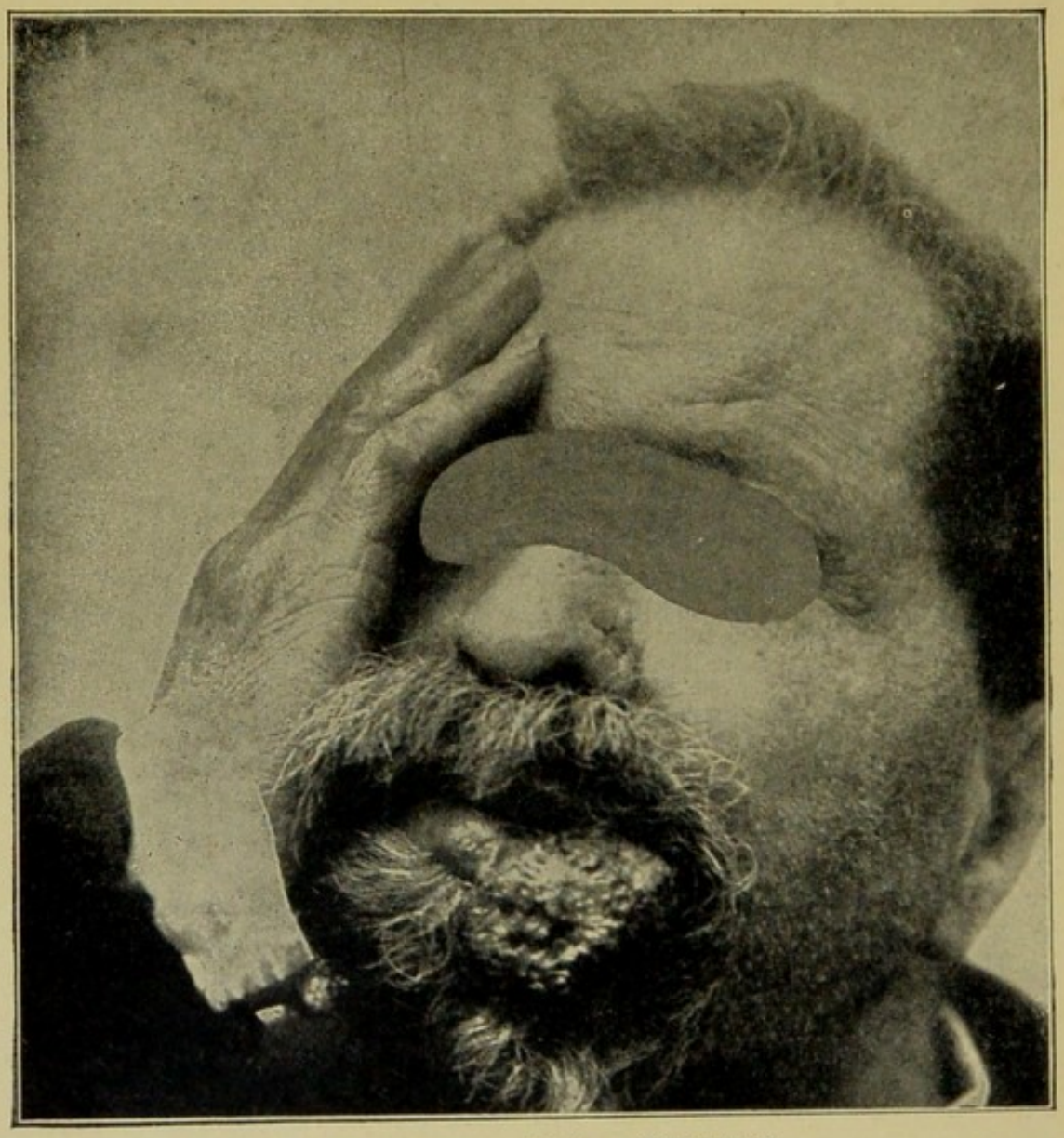


Fig. 120.—Vegetating epithelioma of the lower lip.

secondary radiation emitted by the lead after passage of the gamma rays. This secondary radiation, being only feebly penetrating, is intercepted by the paper and the india-rubber, which are traversed by the gamma rays without appreciable diminution in their intensity. The india-rubber may also be a source of secondary radiation. This is arrested by interposing a sterilised pad of gauze

soaked in a weak antiseptic solution between the tumour and the apparatus.

When the dimensions of the apparatus exceed those of the tumour, it may produce slight dermatitis on the healthy skin

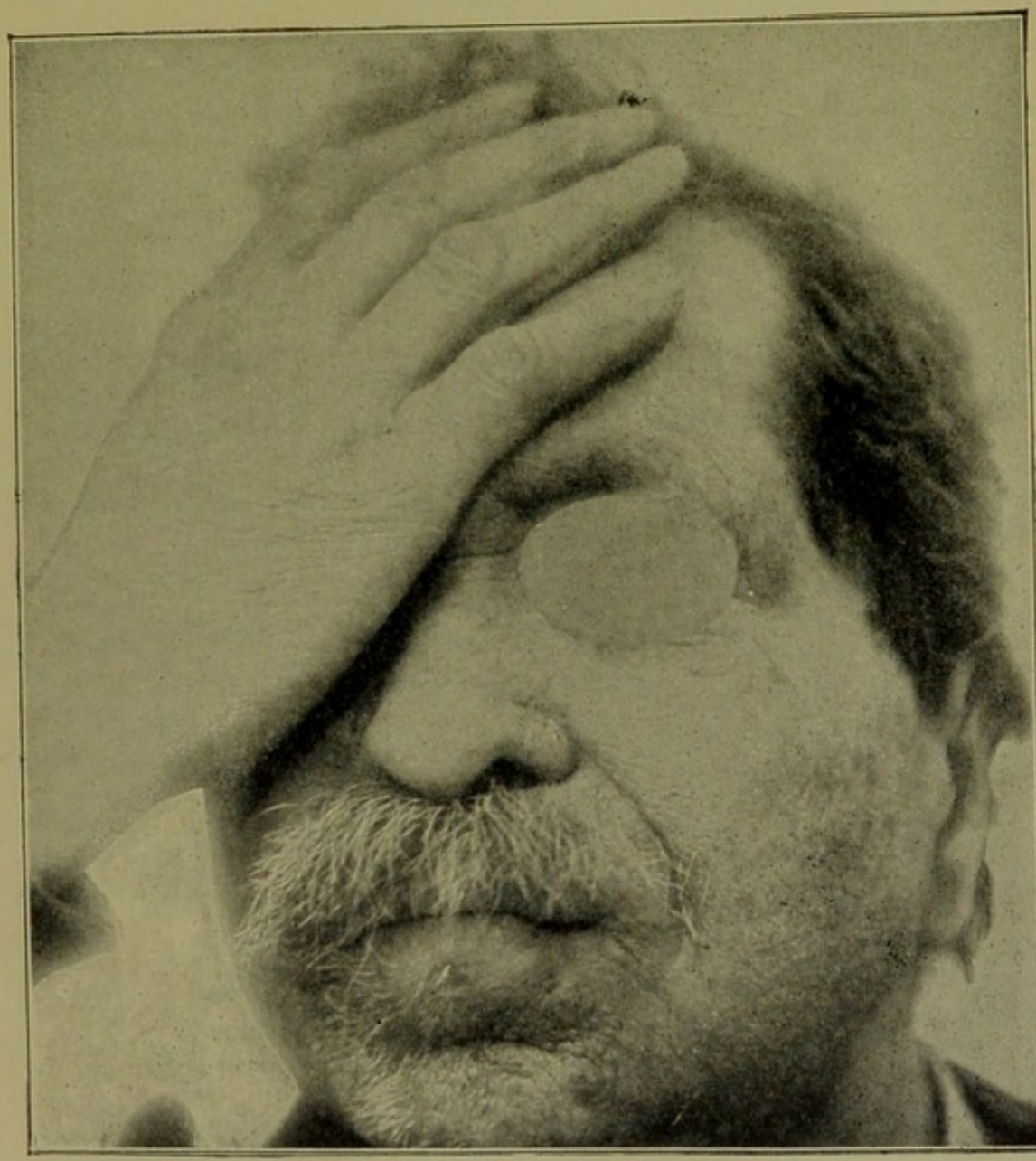


Fig. 121.—The same, after radium treatment.

around the tumour. This may be avoided by protecting the skin with lead and india-rubber in the manner explained above.

Therapeutic applications.—The treatment of cutaneous epithelioma by the penetrating rays varies, both as regards the quality and intensity of the rays, and the duration and order of their application.

Different modes of treatment are required according to the morphological characters and extent of the tumour.

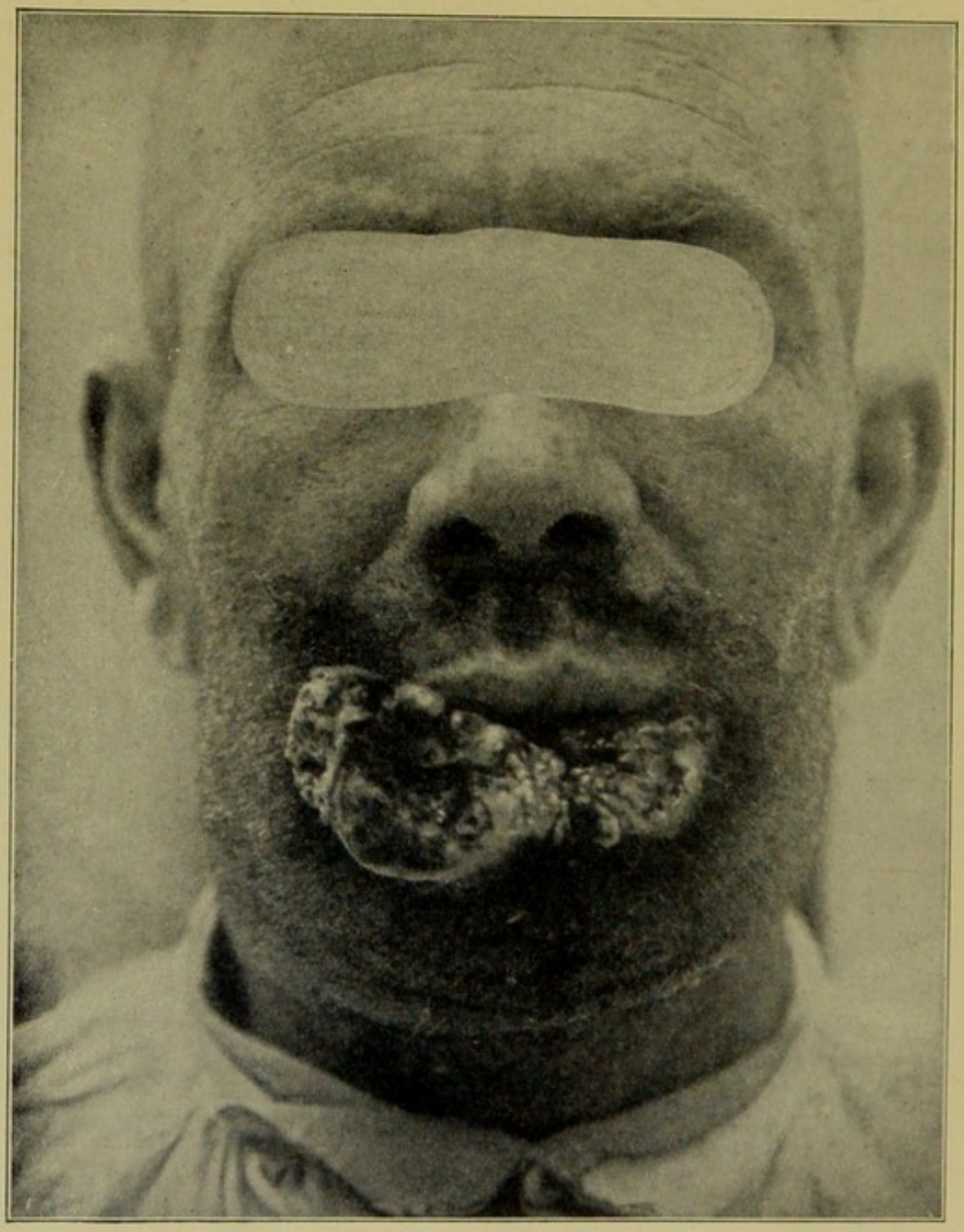


Fig. 122.—Ulcerated and vegetating epithelioma of the lower lip.

The majority of cutaneous cancers can be treated by rays of 3500 to 4500 intensity, filtered through a lead screen $\frac{3}{5}$ millimetre in thickness, and applied for twenty-four to one hundred and twenty hours.

Other tumours require rays of the same quality as the preceding, but of greater intensity (10,000 to 15,000), and applications of

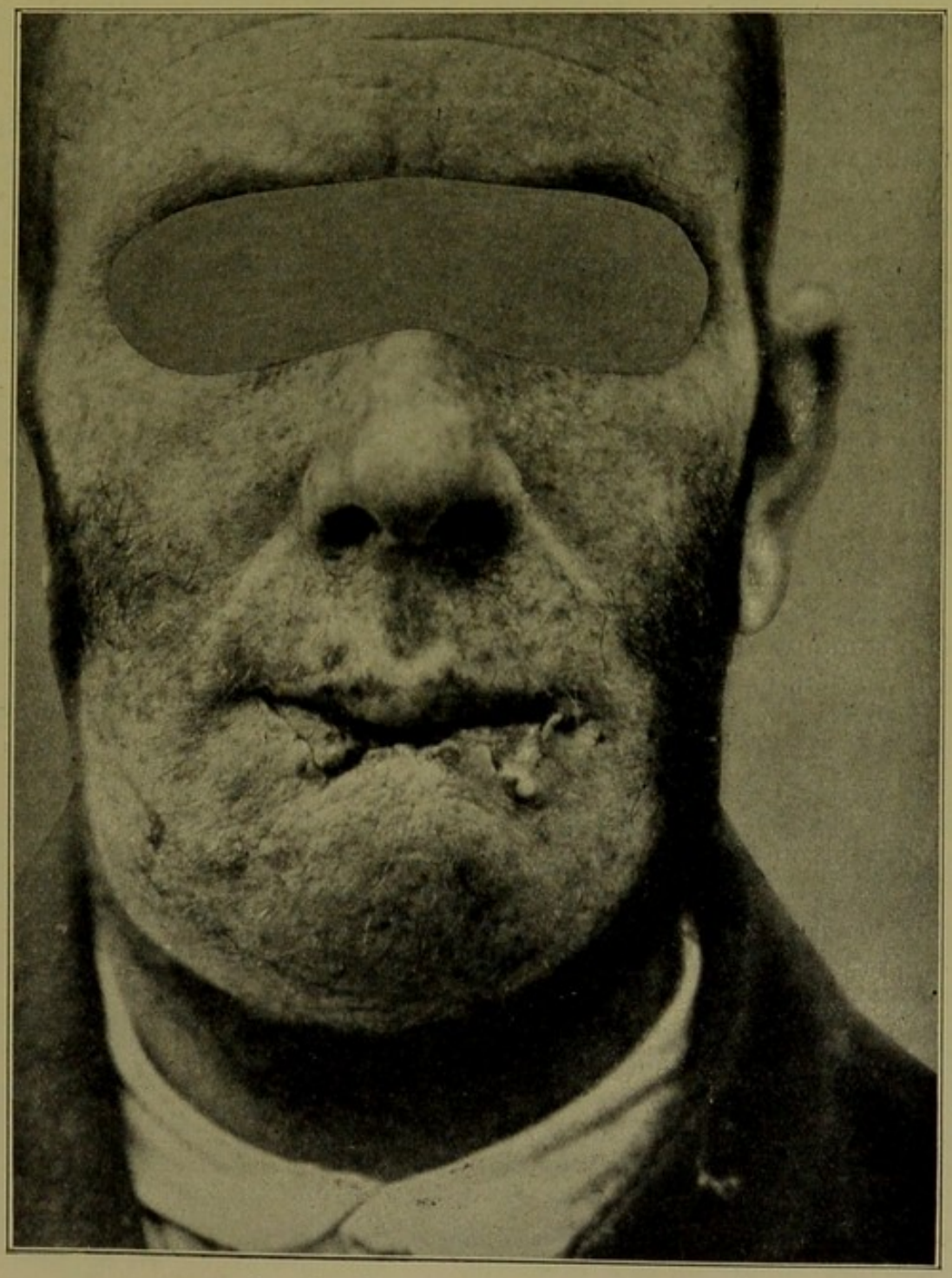


FIG. 123.—The same, after radium treatment.

seventy-two to one hundred and fifty hours' duration. Tumours of this kind are cancers with a thick base, surrounded by a border raised 2 or 3 millimetres above the surface of the skin.

The sittings may be single or multiple. In the former case the apparatus is applied for twenty-four or forty-eight hours or more to the tumour. In the second case the sittings are interrupted, and repeated after an interval of three weeks.

When the dimensions of the cancer exceed those of the apparatus available, it must be treated a part at a time.

The process of resolution of the tumour takes place as follows: In the first stage, lasting from three to eight days, the tumour remains as before; in the second stage, the size begins to diminish, while a sero-purulent and afterwards sanious discharge is produced, lasting a week or ten days; in the third stage, the tumour dries up, and the surface undergoes rapid epidermisation.

The reduction in size of the tumour becomes more evident when it becomes enclosed in a zone of cicatrisation, which extends centripetally. According to the size of the tumour, cicatrisation is completed in two to four weeks, or longer in rebellious cases.

Treatment of cutaneo-mucous epithelioma.—Treatment by the penetrating rays may be applied to cancers of limited area, even when the tumour develops on the mucous portion of the natural orifices, especially on the lips.

Researches made in Professor Gaucher's clinic have shown that these tumours should be treated by the *ultra-penetrating rays.* The following six cases may be given as examples:—

(1) Superficial epithelioma of the mucous membrane of the lower lip, of four years' duration, refractory to cauterisation by nitrate of silver and the thermo-cautery; cured after twenty-four hours' application of an apparatus furnishing a radiation of about 3500, through 1 millimetre of lead.

(2) Epithelioma of the lower lip, recurring three weeks after removal of the tumour, in the form of an indurated erosion occupying the surface of the mucous membrane of the lip; cured after treatment similar to the above.

(3) Epithelioma of the posterior surface of the lower lip, developed on a patch of leucoplasia, in the form of an indurated ulcer with raised borders, essentially localised to the mucous membrane; cured after forty-eight hours' application of an apparatus furnishing a radiation of 4500, through 2 millimetres of lead.

(4) Vegetating epithelioma of rapid growth on the mucous membrane of the left commissure of the lower lip (Fig. 120); cured after one hundred and forty hours' application of an apparatus furnishing a radiation of 4500, through $\frac{1}{2}$ millimetre of lead (Fig. 121).

(5) Epithelioma of the posterior surface and border of the upper lip, in the form of a large deeply excavated ulcer, with a thick border and indurated base; rapid resolution after application of an

apparatus furnishing a radiation of 6500, through 2 millimetres of lead.

(6) Large vegetating epithelioma of rapid growth, destroying the greater part of the lower lip. A comparison of the two figures (Figs. 122, 123) shows the improvement six weeks after the application of an apparatus furnishing a radiation of 11,000 for one hundred and fifty hours, through 2 millimetres of lead. This case is all the more interesting on account of the presence of bilateral glandular enlargement, which was also treated with the same apparatus, applied in two sittings, each of sixty hours. Each application was followed by distinct diminution in the glandular enlargement.

Conclusions.—The study and comparison of the different methods of radiumtherapy, which I have just described in their main outlines, shows the variety of combinations to which radium lends itself from the therapeutic point of view, also the possibility of applying different methods to different cases.

In my opinion, the method which consists in utilising the ultra-penetrating rays, and in prolonging their action on the diseased tissues, is especially suitable for epitheliomas of the mucous membranes. This opinion, with which Professor Gaucher is in accord, is confirmed by the rapidity with which tumours of the labial mucous membrane disappear under the influence of the ultra-penetrating rays, and by the tolerance of the tissues of the lip towards these rays.

These facts have led me to try the same method of treatment in cancer of the tongue. The results so far obtained at the St Louis Hospital seem to show that malignant neoplasms of the tongue are also amenable to radiumtherapy.

Fulguration.—Epitheliomas widely and deeply ulcerated, of too great extent to be destroyed by cauterisation or dispersed by radium, may be treated by the Keating-Hart method of fulguration, which, in some cases, is said to give remarkable results. This method consists in curetting the lesion, and then sparking the surface with currents of high frequency and high tension.

Theoretically, this fulguration of the neoplasm should produce results similar to those obtained by carbonisation with the thermo-cautery, in the case of small epitheliomas. On this subject the inventor of method has sent me the following notes:—

Note by Dr de Keating-Hart.—The fulguration of a cancer is a double operation, consisting of two procedures, which may be alternated several times at the same sitting: a surgical procedure and an electric procedure.

As the operation is very painful, local or general anæsthesia is required, according to the extent of the tumour. In operations on the face, and in all cases during the electric treatment, chloroform is the anæsthetic to be chosen,

because it is not inflammable. Anæsthesia should be deep at the commencement of the sparking, but may be reduced during the operation. A wooden operating table is necessary, to avoid the formation of sloughs at the points of incomplete contact.

The instruments required are surgical and electrical, the former presenting nothing special. The electric apparatus includes: (1) a source of electricity (electric-light supply, dynamo or accumulators); (2) an apparatus for distribution (rheostats, amperemeter, short circuits, etc.); (3) a transformer (coil with 40-centimetre spark, or transformer with closed circuit); (4) a special double condenser; (5) Oudin's resonator; (6) a special electrode (Keating-Hart's), comprising, (*a*) a hollow metallic rod sliding in an ebonite sheath, (*b*) a bellows for carbonic-acid gas or air (the latter for fulguration near the respiratory passages).

Operation.—The patient being anæsthetised and the electrode rendered aseptic, the ulcerated surface and neighbouring parts are first treated by sparking.

The length of spark to be used is measured by the length of metallic rod beyond the sheath; this is 7 or 8 centimetres for most apparatus. Then having passed the current of air or carbonic acid, the electrode is placed over the lesion, and the current turned on. The current of gas and regular and continuous movement of the instrument prevent the production of heat.

This preparatory treatment causes vaso-constriction of the healthy parts round the tumour and softening of the latter, generally in a few minutes. This is followed by surgical treatment, by means of the curette, knife, scissors, etc., according to the nature of the tumour, separating the obviously diseased tissue from the apparently healthy. After stopping hæmorrhage, the surface is carefully examined before commencing the electric treatment. This must be careful and energetic. It is necessary to avoid, as far as possible, carbonisation of the wound, which establishes a barrier between the spark and the living tissues. After this treatment the wound may be sutured and drained; it should be covered with a thick layer of aseptic dressings, on account of the abundant lymphorrhœa which follows.

A single application of this method is usually sufficient, but any focus of recurrence must be treated again. The lymphatic glands may be treated in the same way.

The cases of cutaneous cancers treated in this way are already fairly numerous. They include cancers of the angle of the eye, temporal region, nose, etc., which had invaded the walls of the orbit, the nasal bones, ethmoid, superior maxilla, the eye, and cranial cavity. Many cases have undergone cicatrisation, which, in some cases, has lasted for a year and a half. A melanotic sarcoma of the temporal region remained healed for three years after this treatment.

Cancers of the lips, vulva, and anal region, inoperable by other methods on account of their extent, and glandular complications or post-operative recurrence, have given a fair proportion of successes, remaining free from recurrence for several months up to a year and a half.[1]

PAGET'S DISEASE.

This lesion was first described by Sir James Paget in 1874, and was afterwards studied by Wickham. It must be placed among the

[1] These results have not been confirmed by the investigations of Schulz and others.—*Münch. Med. Woch.*, 1909 Ed.

cutaneous epitheliomas, for even in its initial stage it is nothing else than an expectant epithelioma.

SYMPTOMATOLOGY.—Paget's disease generally occurs on the nipples in women, but it has also been observed in men, on the breast, scrotum and perineum. It appears between the ages of forty and fifty. The lesion is generally unilateral, with a predilection for the right breast; but if the opposite nipple is examined, horny concretions are often found, which seem to indicate the commencement of a similar lesion.

The disease appears either in the form of a small crusted patch covering the tip of the nipple, or in the form of interpapillary

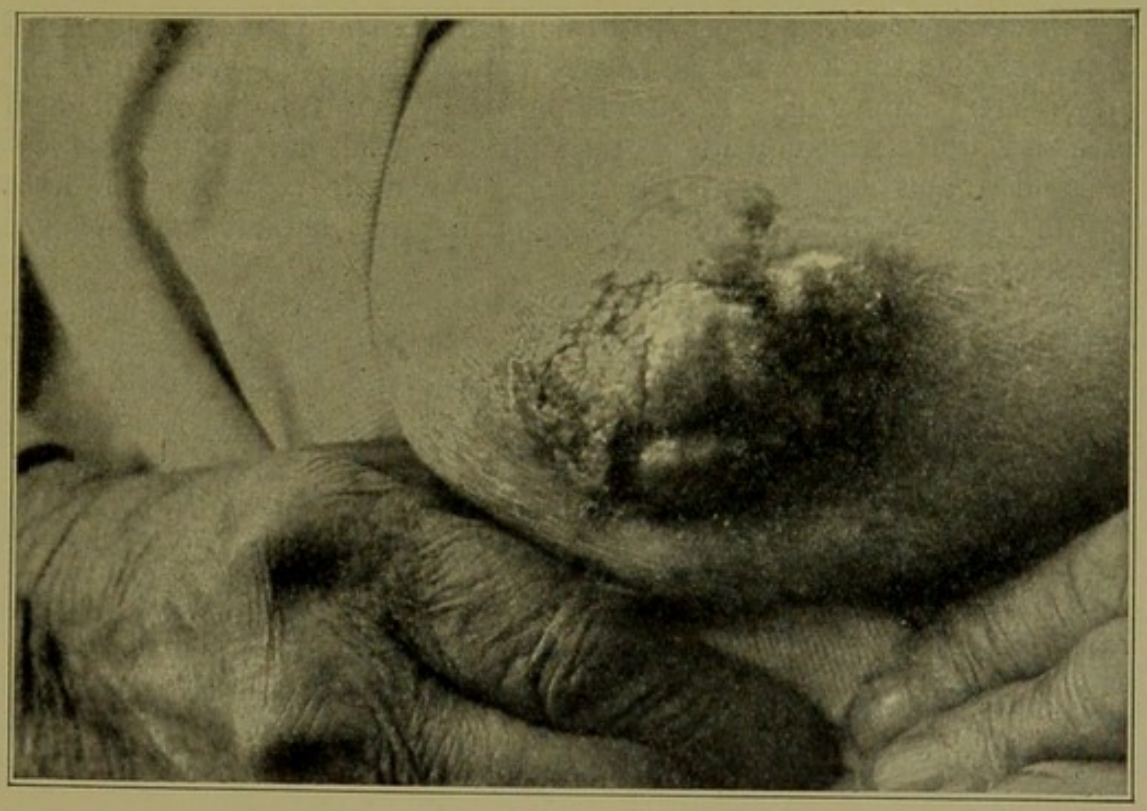


FIG. 124.—Paget's disease.

concretions, which are difficult to remove and tend to recur after removal. Underneath the crust, the dermis is at first normal, but soon becomes red, and presents an excoriation which cicatrises and recurs continually; sometimes it bleeds and becomes covered with a thick crust.

At this period the lesion presents the appearance of a red excoriated patch of variable size, covered with exudation and crusts. There is often also a central fungoid ulceration. This patch is oval in form with sharply defined borders, frequently raised, of a bright red colour accentuated by fine arborescent vessels. The nipple is often retracted. The lesion is sometimes limited to the nipple and

areola, but may extend some distance beyond it. In one case the disease, after twenty years, involved the whole breast and the axilla.

By careful examination smooth dry points can be seen on the red patch; these are undergoing repair. If the patch is seized between the fingers, it is found to be slightly indurated. At this stage the glands are never affected. There is sometimes acute pain on exposure to air, or from contact with the clothing; there is also more or less itching, and sometimes severe intercostal neuralgia.

The characteristic feature of Paget's disease is the development of cancer, after a certain time. However, this termination is not inevitable; in fact, in the case mentioned above, in spite of the twenty years' duration and the age of the patient (seventy), there was no trace of cancer. It is difficult to fix the date of appearance of cancer, but the average is between two and seven years after the onset. The cancer may be superficial or deep. When superficial, it forms an ulcer in the centre of the patch, with irregular, punched-out, and somewhat indurated borders. When deep, it forms a hard nodule situated below the surface, or in the deeper parts of the breast.

PROGNOSIS.—This is grave, as any case may terminate in cancer. The disease, when once established, never undergoes resolution. It sometimes extends for some distance on the skin beyond the primary focus. Its duration is indefinite, and its progress is hastened by the development of cancer.

DIAGNOSIS.—*Eczema of the breast* is the lesion which most resembles Paget's disease; but in eczema the surface is more crusted and fissured, not granular as in Paget's disease; it is also of a paler red colour, and often presents vesicles at the periphery; the borders are more diffuse than in Paget's disease. *Psoriasis, lupus erythematosus* and *rodent ulcer* must also be borne in mind in diagnosing Paget's disease.

PATHOLOGICAL ANATOMY. — Microscopical sections show an irregular arrangement of the cells of the Malpighian layer. In sections stained with carmine, small irregular masses stained red are seen, and a number of clear spots. The red masses were formerly regarded as parasites (*psorosperms*) in various stages of development, and the clear spots were supposed to be spaces round the contracted protoplasm of cystic parasites. It is now known that these bodies are not parasites, but forms of cell degeneration.

The Malpighian layer is thickened and its interpapillary spaces elongated. The dermis is thickened and inflamed. At the stage of ulceration, the stratum corneum and stratum granulosum disappear, but the deeper layers undergo active proliferation; polymorphous cells with large nuclei are seen, which at some points present the appearance of epitheliomatous transformation.

When cancer develops, it may commence in the epidermis or in the ducts of the glands. It presents the microscopic characters of epithelioma.

ETIOLOGY.—The idea that Paget's disease is a parasitic disease produced by psorosperms can no longer be maintained. It is an epithelioma, the cause of which is no more known than that of other cancers.

TREATMENT.—As long as the lesion remains superficial, and there is no cancerous development, it may be treated by the application of a strong solution of chloride of zinc; but it is preferable to destroy it with the thermo-cautery. After-treatment consists in moist dressings and aristol ointment. When cancer develops, it should be treated in the same way as epithelioma, especially by radium.

KRAUROSIS VULVÆ.

This affection (described in 1885 by Breisky) should be classed among the cutaneous epitheliomas.

Kraurosis is characterised by fibrous atrophy of the vulva, with atresia of its orifice. It is preceded and accompanied by leucoplakia, and, like the latter, may end in epithelioma. It has the same origin as leucoplakia, but its evolution is different. Leucoplakia, both of the genital organs and of the mouth, is a horny papilloma, that is, an epidermo-papillary or dermo-epithelial lesion. Kraurosis is also a dermo-epithelial lesion, but the dermic lesions predominate, and lead to sclerosis and retraction, while leucoplakia develops chiefly from the epithelium. In leucoplakia the papilloma is chiefly keratosic, in kraurosis it is chiefly fibrous. This is why the epithelioma which succeeds leucoplakia is vegetating, and that which succeeds kraurosis, atrophic.

ETIOLOGY.—Like leucoplakia, this affection appears to be of syphilitic origin.

COLLOID DEGENERATION OF THE DERMIS.

This is a very rare affection, characterised by colloid degeneration of the superficial layers of the dermis. It was first described by Wagner (1866), under the name of *colloid milium*. In 1879, Besnier and Balzer showed that it was not a colloid milium, but a colloid degeneration of the dermis and its vessels.

The lesions consist of hard, yellow, shiny elevations, the size of a pin's head, isolated or confluent, and easily enucleated by means of a small curette. They are situated chiefly on the cheeks, nose,

forehead and conjunctiva, more rarely on the ears, neck and back of the hands. There are sometimes vascular dilatations near the lesions. Liveing has seen the lesions become inflamed and then disappear.

ETIOLOGY.—This is unknown. The lesions occur only on exposed parts of the body. Balzer has shown that they consist of colloid masses developed in the connective tissue of the upper layers of the dermis, and possibly in the vessels of the dermis.

TREATMENT.—The lesions may be destroyed by the curette or the galvano-cautery.

MICROBIAL DERMATOSES.

IMPETIGO.

THIS affection is characterised by superficial pustules without any peripheral inflammation, followed by yellow crusts which leave no cicatrices after they fall.

ETIOLOGY.—Impetigo is caused by superficial inoculation of the skin with pyogenic micro-organisms. The *streptococcus* is the microbe of impetigo, which is therefore a contagious and inoculable affection. Its contagion was shown clinically by Devergie, and afterwards by Tilbury Fox, who described a special form, *impetigo contagiosa;* but this is incorrect, as it is an ordinary impetigo. All impetigos are inoculable on the same subject, and from one subject to another, according to the researches of T. Fox and Vidal. Children of the same family inoculate each other. Devergie was the first to observe that children affected with impetigo sometimes infect their mothers.

Impetigo, especially in children, often coexists with other superficial suppurations; superficial whitlows, ulcers of the conjunctiva, and ulcers of the buccal mucous membrane (impetiginous stomatitis). All these lesions are produced by the same microbe as impetigo.

The inoculability of this affection explains its development on eczematous surfaces (impetiginous eczema), and on excoriations of the skin produced by scratching, etc. This is why impetigo often complicates scabies and pediculosis. Impetigo occurs chiefly in children, especially in lymphatic subjects, who offer an excellent soil for the culture of pyogenic microbes.

SYMPTOMATOLOGY.—The eruption generally appears without prodromal symptoms, but sometimes there is gastric disturbance. A red, slightly pruriginous, spot first appears, then a small, round, yellow pustule, distended with purulent liquid. Other similar pustules then appear around the first. The pustules may be isolated or grouped in patches of varied extent. They vary from a hemp seed to a lentil in size. Their base is neither indurated nor inflamed.

They are very fragile, and are ruptured in two or three days by scratching, giving rise to a yellow discharge, which dries into soft, yellow, friable crusts, resembling honey. Sometimes the crusts are greenish yellow, or black, from slight hæmorrhage (impetigo nigricans). The crusts fall spontaneously, or as the result of treatment. The subjacent skin is red and eroded. In some places the discharge

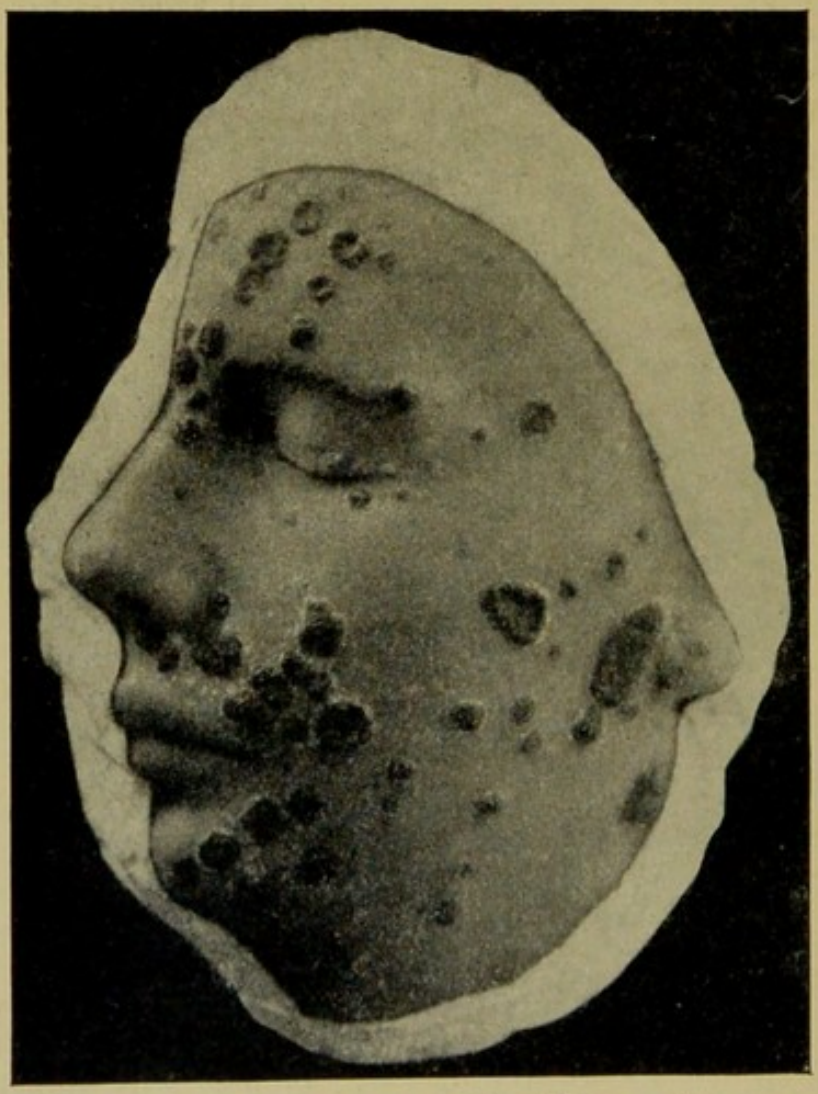


Fig. 125.—Impetigo. (St Louis Hospital Museum.)

continues, forming new crusts, which become progressively thinner. The erosion heals, leaving a red surface, which disappears without leaving any trace.

Impetigo never leaves cicatrices, except in very rare cases, or when the pustules have been scratched through to the dermis.

The disease lasts about a fortnight, sometimes longer when the crusts are thick and repeatedly renewed. It may also be prolonged by the development of new pustules by successive auto-inoculation.

Children scratch the pustules and inoculate themselves, thus showing lesions of different ages.

Varieties.—Confluent pustules constitute *impetigo figurata;* this form occurs on the face, sometimes on the limbs, but never on the trunk. Impetigo sometimes covers the whole face like a mask (*impetigo larvalis*); the eyelids and nose are free, but there are sometimes conjunctival lesions and a nasal discharge, caused by pus being conveyed to the eyes and nose by the fingers. This mask causes pain and intense itching; the submaxillary glands are enlarged and may suppurate. In some cases the pustules are scattered on the face, limbs, and scalp, and sometimes on the trunk (*impetigo sparsa*).

On the scalp, impetigo causes more intense itching than in other regions. Each pustule is traversed by a hair, and when they rupture, the hairs become glued together by the dried pus. This form is often secondary to pediculosis. The itching caused by the lice leads to scratching, and this to excoriations, which become inoculated with the microbes of suppuration. The hairs, adherent to the crusts, sometimes fall out, but they grow again except where the hair follicles have been destroyed. The scalp is sometimes the seat of acute inflammation, which spreads to the subcutaneous tissue and gives rise to small abscesses.

Prognosis. — This is benign. As impetigo is an affection of external origin, it can always be treated without fearing any inconvenience from the sudden disappearance of the eruption, as in eczema.

Diagnosis. — The crusts of *eczema* are gray and thinner than those of impetigo, and the skin is indurated; eczema is of long duration, while impetigo is cured in a short time.

Impetiginous eczema is an eczema with secondary infection with impetigo; in this form the impetiginous element is easily cured by appropriate treatment, but the eczema persists.

The crusts of *herpes* may be mistaken for those of impetigo, especially when they are yellow; but herpes is always more circumscribed and non-exudative; the crusts of herpes are not reproduced when they fall; herpes is generally situated about the mouth or nostrils.

The pustules of *ecthyma* differ from those of impetigo in being always isolated and situated on a hard, red base; the crusts are thick and very adherent, brown, and not yellow like those of impetigo.

The crusts of *favus* differ from those of impetigo of the scalp; they are not soft, but dry; they often form cups, and have a mouse-like odour; the hairs contain spores of the *Achorion.*

The diagnosis between impetigo and the *pustular syphilide* is more difficult; the syphilitic pustules discharge less, and form dry,

adherent, thick, stratified, brown or greenish crusts, which leave brown cicatricial spots after they fall, very different to the red non-cicatricial spots of impetigo.

Impetigo must be distinguished from the *pustulo-ulcerative tuberculide* (formerly named incorrectly *impetigo rodens*). This is characterised by pustules which differ from those of impetigo in their depth and in the ulceration beneath the crusts. In doubtful cases experimental inoculation of liquid from the pustules always causes tuberculosis.

Impetigo must be completely separated from *impetigo herpetiformis*, a special affection consisting of very small pustules arranged in circinate groups, and accompanied by severe general symptoms; it usually occurs in pregnant women.

TREATMENT. — As impetigo often occurs in lymphatic subjects, cod-liver oil and iodine preparations are often indicated.

Local treatment consists first of all in removing the crusts by means of starch poultices, or, better, gauze dressings soaked in boracic lotion and covered with gutta-percha tissue. The discharge may be checked by powdered talc and boric acid.

When the crusts have been removed, ointments should be applied. The simplest is boric acid and vaseline (10 per 100); this may be thickened with oxide of zinc (10 per 100). In cases which resist this ointment, calomel or yellow oxide of mercury ointment may be tried (5 per 100), or glycerole of oil of cade; or even pure oil of cade (Bazin); but the boric acid ointment usually succeeds. Spraying with a one per cent. solution of resorcin is also useful.

Prophylactic measures should be taken in schools and in families to prevent contagion, by keeping the affected children apart from the others.

ECTHYMA AND RUPIA.

These two affections are both constituted by pustules, the only difference being that the pustules of *ecthyma* commence as vesicles, while those of *rupia* are purulent bullæ. The two lesions appear under the same conditions; they only differ in size, and often coexist in the same subject.

ETIOLOGY.—The pustules of ecthyma and rupia result from inoculation of the deep layers of the epidermis with pyogenic microbes; in the pus of the pustules are found monococci, diplococci, staphylococci and streptococci; some of these microbes are very similar to the micro-organisms of impetigo and whitlow.

The pus of the pustules is inoculable and auto-inoculable, but no experimenter has yet succeeded in reproducing the typical ecthymatous pustule by inoculation of micro-organisms obtained

from the pustules. However, Leloir produced a small pustule of ecthymatous appearance, but very ephemeral, by inoculating himself with a pure culture of *staphylococcus pyogenes aureus*. It is possible that the soluble products elaborated by these microbes are necessary for the production of the pustule; Grawitz and Bary have shown that sterilised cultures of the cocci still cause suppuration.

These pyogenic microbes may be internal or external. Ecthyma occurs at the end of certain infective diseases, such as enteric fever, variola, scarlatina, etc. During the decline of enteric fever the ecthymatous eruption may be regarded as a kind of cutaneous bacterial discharge, arising from secondary infection (Bouchard). In other cases, ecthyma results from external infection, by direct penetration of the microbes through excoriations in the skin; this is observed in all parasitic affections as the result of scratching, especially in scabies and pediculosis, and in pruriginous dermatoses, such as eczema, prurigo, etc. Workmen who handle irritating substances which cause excoriation of the skin are liable to ecthyma, especially masons, grocers, ostlers, leather-dressers, etc.

Ecthyma is most common in children and old people, who furnish a favourable soil for its production, on account of the debility natural to the extremes of life. It also occurs in persons broken down in health, and in cases of diabetes and Bright's disease.

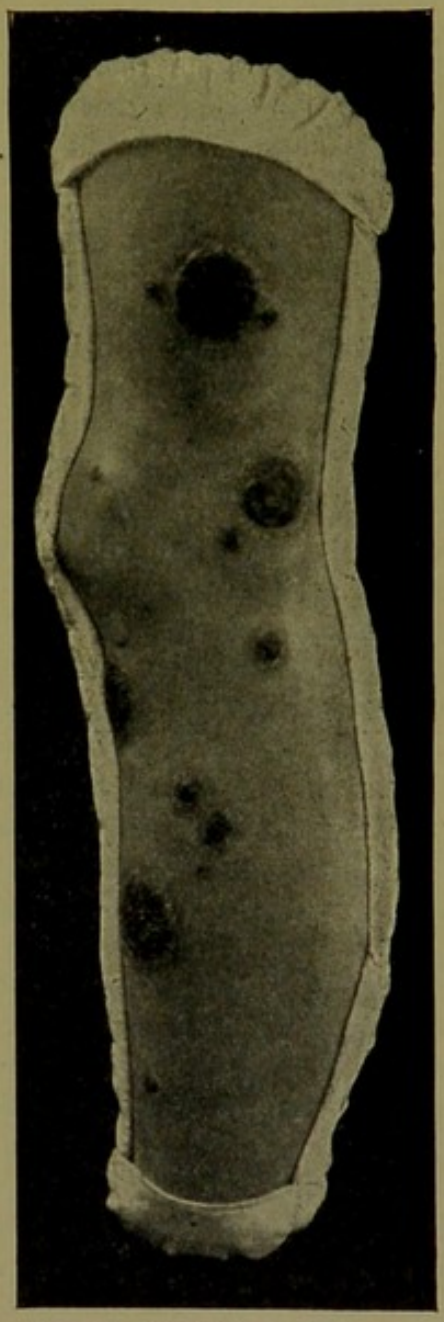


Fig. 126.—Rupial form of ecthyma. (St Louis Hospital Museum.)

Symptomatology.—*Ecthyma* begins as a red pruriginous spot, which develops into a red acuminate papule with a hard base. On the second day, or earlier, a yellow vesicle appears at the tip of the papule, and on the third or fourth day the pustule is completely formed. The pustule is surrounded by a red areola, and situated on an indurated base; it varies from a lentil to a sixpence in size. About the seventh day the pustule ruptures

and the pus dries in the form of an irregular, thick brown crust. After a time the crust falls off and leaves a red spot, or, when the inflammation has extended more deeply, a purple cicatrix. In old people and cachectic subjects an ulcer forms under the crust, which leaves a permanent cicatrix.

In *rupia* the lesion commences as a bulla, the contents of which are often sanious rather than purulent. The crusts are larger, and raised in the form of a cone; they have a stratified appearance like limpet shells, which is due to their tendency to extend at the periphery. Around the original lesion other bullæ develop, which form crusts in the same way. The crusts of rupia are adherent, and the subjacent ulceration wider and deeper than in ecthyma. Rupia, which has the same origin as ecthyma, is rarer, and occurs exclusively in cachetic individuals.

Ecthyma and rupia affect chiefly the lower limbs, back and shoulders; they are rare on the face. The pustules are always isolated and generally few in number. The eruption is sometimes generalised, but it always remains discrete. It may occur in successive crops. The lymphatic glands are enlarged and sometimes suppurate. General symptoms depending on the health of the patient are usually present; fever is rare. Augagneur has described an infective nephritis secondary to ecthyma; in certain cases where the eruption was very extensive and the general condition of the patient bad, there was albuminuria. The mechanism of this albuminuria is the same as that of all infective albuminurias.

Varieties.—Ecthyma secondary to infective diseases appears chiefly on the back, buttocks and thighs. It is constituted by numerous isolated pustules, which soon rupture and give rise to painful ulcers, which often heal without leaving cicatrices.

In old people and debilitated subjects, ecthyma is characterised by large pustules, followed by thick adherent crusts covering deep and rebellious ulcers (cachectic ecthyma). These ulcers sometimes become gangrenous and leave cicatrices surrounded by a pigmented ring.

In children, ecthyma is also a sign of cachexia occurring in debilitated and badly nourished subjects. It appears in the form of disseminated pustules, on the limbs, especially the lower limbs and buttocks, and on the trunk, rarely on the face. The pustules are followed by deep ulcerations (infantile gangrenous ecthyma). Children affected with ecthyma waste rapidly and suffer from digestive disorders, diarrhœa and vomiting, symptoms due to the cause which produces the ecthyma.

In some cases the pustules are very large and form bullæ, surrounded by a purple areola. The contents of these bullæ is serosanious and does not form crusts; the bullæ rupture and leave

deep gangrenous ulcers (*rupia escharotica*). This eruption is accompanied by fever and a bad general condition, which often ends in death.

DIAGNOSIS.—The diagnosis from *impetigo* has already been given (p. 303). Ecthyma cannot be mistaken for *furuncle;* this forms a hard, deep swelling, more inflammatory than ecthyma, and ending in the elimination of a core, resulting from mortification of the cellular tissue. The diagnosis from the *pustular syphilide* (incorrectly called syphilitic ecthyma) is more difficult. This often has a copper-coloured areola, and is frequently situated on the face; it gives rise to thicker crusts, which are gray or greenish and sometimes stratified (*syphilitic rupia*); the cicatrices are more regular, more superficial, and smoother than those of ecthyma, white in the centre and pigmented at the periphery. Infantile gangrenous ecthyma is often mistaken for congenital syphilis, but differs from this in the intensity of the inflammation and more rapid ulceration.

TREATMENT.—General treatment consists in nourishing diet and tonics, such as quinine and iron. Bottle-fed infants should be given to a wet-nurse.

In the pustular stage, before the pustules have ruptured, moist applications and poultices should be avoided, as they hasten rupture of the pustules and hinder the formation of crusts; the lesions should be dusted with a mixture of talc, iodoform, and boric acid, and covered with a dry dressing. If the crusts are slow in becoming detached, they may be hastened by means of cold poultices and moist boracic dressings; but they should not be removed too soon, as they constitute an excellent protective covering, underneath which the ulceration may cicatrise spontaneously. Ulcers remaining after the crusts have fallen, and those which occur without any crusts, may be washed with boracic lotion or diluted oxygenated water, and then dressed with iodoform vaseline (10 per 100), or dusted with iodoform, traumatol or dermatol. Rebellious ulcers may be dressed with styrax ointment.

ORIENTAL BOIL.

This affection is also known by other names according to the region where it occurs (*Aleppo boil, Biskra button, Delhi boil*, etc.). It may develop on the healthy skin, or may be inoculated on some lesion of the skin, such as an acne pustule, etc. It is met with at all ages, both in men and women, in Algeria, Gabon, the Congo, Tunis, Egypt, Syria, Persia (where it is known as salek), and in India, between the months of September and February. The period of incubation seems to be at least eighteen days (Vidal).

The lesion occurs chiefly on the face and limbs, on the exposed parts of the body; it is rare on the trunk. Aleppo boil occurs on the face chiefly, Biskra button on the limbs. It commences as a small, red, conical, slightly pruriginous papule, which increases in size till it becomes a tubercle. After two or three weeks the apex of the tubercle becomes pustular, and dries to form a brown crust.

In some cases the crust is renewed after it falls, and the disease remains crustate during its whole evolution, without deep ulceration. Sometimes the crust becomes thick and rupial. This is the *dry form.* In other cases the crust is not renewed, and an ulcer is left with callous borders, punched-out or undermined, and a sanious base secreting a sero-purulent liquid. This is the *ulcerative form.* The ulcer extends in depth, and also superficially by the development of new papules at its borders, which undergo the same evolution as the primary

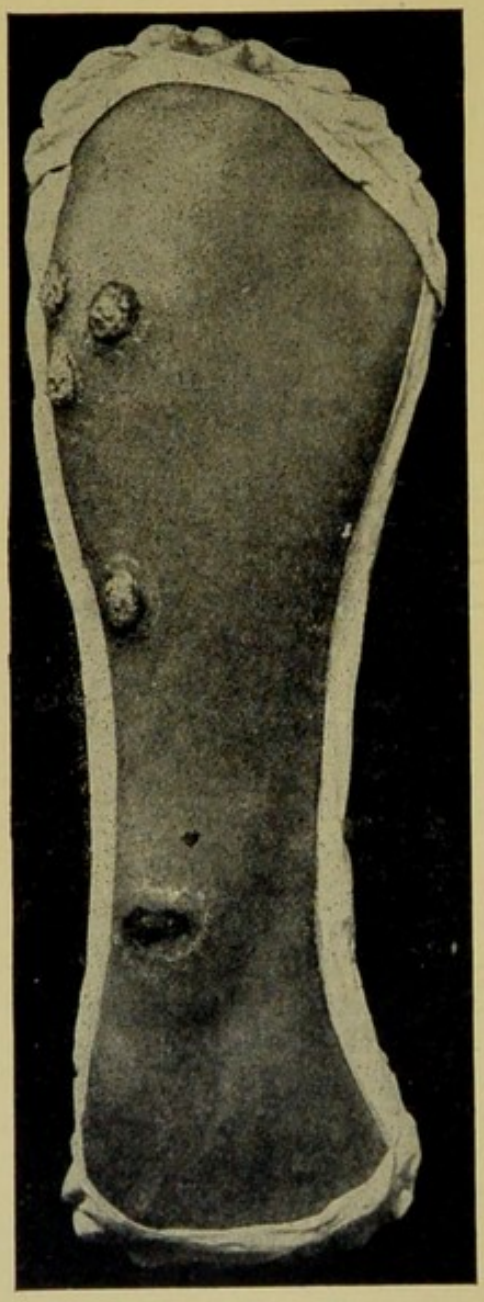


Fig. 127.—Biskra button (dry form). (St Louis Hospital Museum.)

Fig. 128.—Biskra button (ulcerative form). (St Louis Hospital Museum.)

papule. The resulting ulcer attains the size of a shilling or larger; in a case of Bertherand's it occupied the whole buttock. Sometimes the ulcers remain distinct from each other.

After four or five months the ulceration ceases to extend; its base becomes covered with fleshy granulations which give the lesion a papillomatous appearance. Cicatrisation takes place gradually; the cicatrix is first purple, then yellow, or white in the centre and brown at the periphery, finally white and indelible. In the dry form, the fistulous openings close and the crust falls, leaving a red, squamous, depressed cicatrical surface. The redness fades, leaving a white, indelible cicatrix.

Apart from inflammatory complications, the affection is quite

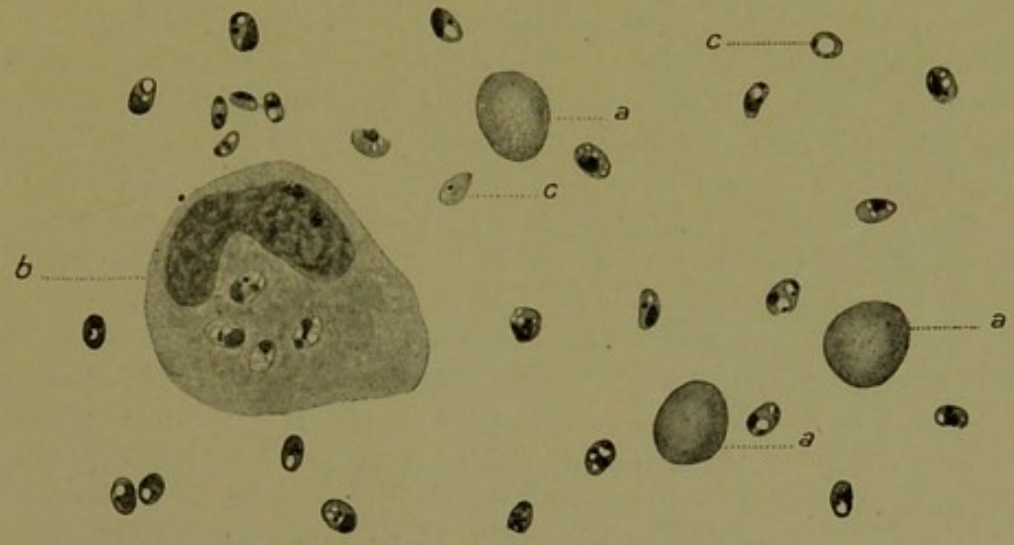


FIG. 129.—Piroplasma in Biskra button. (Nattan-Larrier.)

a, Red blood corpuscles; *b*, a macrophage containing six piroplasmas, two of which are situated on the nucleus and four beyond it; *c*, two degenerated forms of piroplasma. Besides these, there are twenty-five other piroplasmas, in which the nucleus, chromatosome, and vacuoles can easily be distinguished. Eleven of these are in process of reproduction (division of nucleus and chromatosome, and multiplication of vacuoles).

painless. Its average duration is six or seven months. Aleppo boil has a longer duration, about a year. The lesion may be single, but is more often multiple; usually from two to six, sometimes (Algeria) twenty, forty or more.

DIAGNOSIS.—Oriental boil has a certain resemblance to *lupus*, but the latter presents characteristic nodules around the principal lesion. It may be mistaken for *tertiary syphilis* or *soft chancre;* but syphilides have not the red colour of Oriental boil, and their evolution is different; soft chancre is a simple, punched-out ulcer, without subjacent induration. *Furuncle* and *carbuncle* are more deeply situated, more inflammatory, and of more limited evolution, which ends in the expulsion of a core.

ETIOLOGY.—Oriental boil is a parasitic affection, inoculable and

auto-inoculable. A first attack does not confer immunity, at any rate in Algeria. Boinet and Dépéret have observed cases of contagion in subjects who had never left France, and who had contracted the disease from infected soldiers who came from Tunis.

PATHOGENY.—Recent research has shown that the parasite of all forms of Oriental boil is a special form of protozoon. This protozoon, known by the name of *piroplasma*, is allied to the *trypanosomes*. It was discovered by Wright in 1903, and has since been studied by Nattan-Larrier.

The piroplasmas are intracellular parasites, occurring in the form of ovoid bodies within the macrophage cells, to the number of fifty or more; they may also be free. They occur not only in the sore itself, but also in the neighbouring tissues, and in the local blood-vessels and lymphatics. They have not been found in the general circulation.

The piroplasma has been found by different observers in Delhi boil, Biskra button, Egyptian boil, and in Aleppo boil. According to Nattan-Larrier, it is only pathogenic for man, and is probably transmitted by the agency of mosquitoes.

TREATMENT.—According to Moty, the evolution of the lesion can be arrested by a dressing of perchloride of mercury (1 in 1000). Gemayel uses the same drug in the form of a 1 per cent. ointment. If the lesion is an old one, the crust should be detached, the ulcer dusted with calomel, and then dressed with gauze soaked in Labarraque's solution; this is renewed on the following day, and the treatment then continued with solution of perchloride of mercury. In reality, antiseptic dressings seldom arrest the progress of this lesion. The natives generally leave it to cure itself. However, I think the initial papule might be aborted by deep cauterisation with the thermo-cautery.

In countries where the disease is endemic, it is well to protect excoriations of the skin by a dressing, as a prophylactic measure.

TROPICAL ULCER.

Under this generic name are included: *Annamite ulcer, Cochin-china ulcer, Gabon ulcer, Congo ulcer; Mozambique ulcer,* which occurs chiefly among Kaffirs; *Madagascar ulcer,* also seen in the Comores, at Reunion and Sierra Leone, and, according to Monestier, at Hayti; *Guadeloupe, Guiana,* and *New Caledonia ulcer; Yemen ulcer,* seen especially among the negroes of Sennaar, Kordofan and Darfour, and among the Arabs of Zanzibar and Massouah.

By comparing the descriptions of these various ulcers, we must conclude that they represent the same disease, which has also been named *tropical phagedena.*

I have personally observed two cases of Annamite ulcer and two cases of Gabon ulcer, and have obtained descriptions of these affections from army and naval surgeons. By the aid of this evidence, I shall attempt to give a succinct description of tropical ulcer. This must be distinguished from simple varicose ulcer and syphilitic ulcer, with which it has often been confounded, and which, on the other hand, have sometimes been regarded incorrectly as tropical ulcers.

Symptomatology.—The lesion begins by a papule or a papulo-pustule. It is sometimes apparently spontaneous, but more often occurs on some slight lesion of the skin, such as the excoriations produced by thorns, fragments of coral, etc., or insect punctures, such as those of the chigoe (*Pulex penetrans*), etc. In fact, any simple wound or abrasion may become the seat of tropical ulcer.

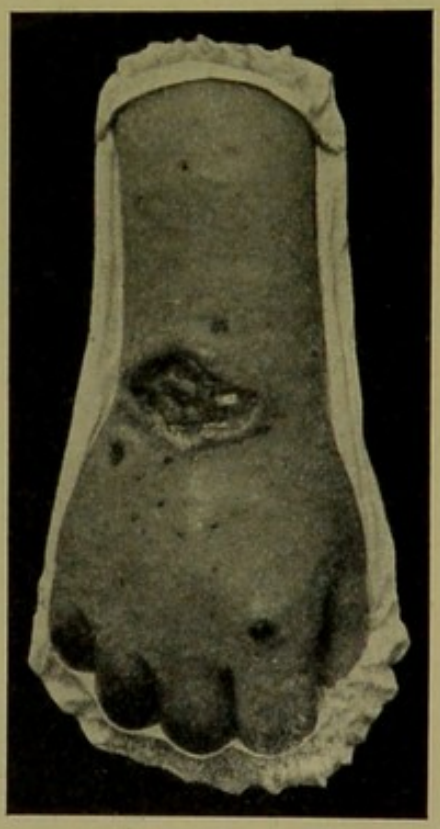


Fig. 130.—Annamite ulcer. (St Louis Hospital Museum.)

The primary papule, which is very pruriginous, becomes excoriated, and gives rise to an erosion, which gradually extends and is surrounded by a circular detachment of the skin. A round ulcer results, with undermined borders and a sero-purulent discharge, which dries into a soft crust; the skin round the ulcer is red and œdematous. The peripheral undermined skin is gradually destroyed, and the ulcer assumes its characteristic appearance.

At its period of maturity the ulcer is circular, unless its development is modified; in this case it may form a segment of a circle, or may be oval or linear when situated between the toes. Sometimes it is serpiginous; or two ulcers may coalesce, in the form of two incomplete circles. The size of the ulcer varies, but it is usually about 2 inches in diameter. The borders are callous, everted, and sometimes surrounded by œdema. The base is cup-shaped or irregular, with a grayish colour and fœtid odour; it may be covered with granulations.

This ulcer presents a strong resemblance to a *syphilitic gummatous ulcer*, which appears to have caused many errors in diagnosis. Unless very extensive, the ulcer is painless, except on contact. It is usually situated on the lower limbs, which are the regions most frequently exposed to traumatism and abrasion. It also occurs on

the sacrum and back of the thighs by auto-inoculation, owing to the native habit of sitting on the heels. It may also occur on the face, especially on the lower lip, on the back of the hands; in fact, on any exposed part of the body.

There is generally a single ulcer on one leg, sometimes one on each leg, sometimes three or four on the legs and forearms, as I have seen in a patient from Gabon.

Evolution.—This is generally slow, and the lesion may remain for a long time stationary. In some cases, however, it extends rapidly both superficially and deeply, invading the muscles, tendon-sheaths, and bones. In this case it merits the name *phagedenic*, and may necessitate amputation. Gangrene has been observed in some cases; but apart from these grave complications, the affection is seldom fatal. As a rule, it remains localised, either spontaneously or under the influence of treatment, and gradually cicatrises from the periphery to the centre; but cicatrisation is very slow, and difficult to obtain. The ulcer sometimes persists indefinitely, when the affected subjects remain in infected countries; it may even relapse after healing. It always leaves a pigmented cicatrix, which is identical in appearance with that of *syphilitic gumma*. This pigmentation was very clear in the cases which I observed.

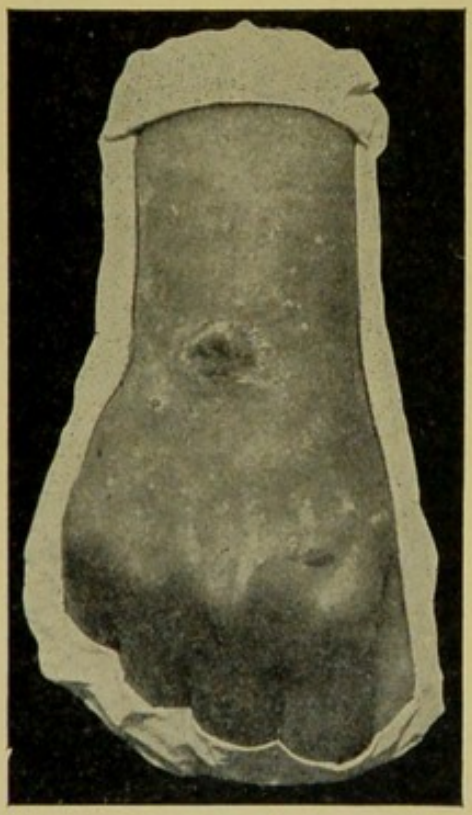


Fig. 131.—Annamite ulcer in process of healing (same as Fig. 130). (St Louis Hospital Museum.)

Diagnosis.—Tropical ulcer must be distinguished from *varicose ulcer*, and from *Oriental boil*, which has a different evolution, and is often covered with a brown crust. The diagnosis from *syphilitic ulcer* is much more difficult, for the two lesions are sometimes identical in appearance; the history is not sufficient to establish the diagnosis, for an old syphilitic may be affected with tropical ulcer; in many cases the therapeutic test is necessary. *Yaws* is a febrile affection, characterised by multiple lesions situated on the face, trunk and limbs; the rapid appearance of multiple lesions distinguishes the ulcers of yaws from tropical ulcer.

Etiology.—This affection occurs chiefly among the black race, who expose their skin and take no hygienic precautions; but it

may also occur in white people who inhabit the same countries. It is observed especially in debilitated subjects.

It is a contagious disease, inoculable and auto-inoculable, the parasitic origin of which is certain; but it appears to be peculiar to man. Vinson, who studied Mozambique ulcer in the island of Reunion, found that animals were immune, even when bitten by flies.

The parasite of tropical ulcer has been sought for without success. The results of different observers do not agree and are inconclusive. Microbiological examination is difficult, on account of secondary infection of the sore. The lesion should be examined in its early stage, while the published researches have always dealt with mature cases secondarily infected. I have myself examined a case of Gabon ulcer, and one of Annamite ulcer, and found various cocci and bacilli, but no specific organism. Le Dantec, Boinet, Tournier, and Vincent have described bacilli and micrococci, but none of these have been proved to be the pathogenic agent of tropical ulcer. Possibly it is a protozoon.

TREATMENT.—From my own observations, and from the information I have obtained from medical men practising in countries where this affection is endemic, it appears that the whole treatment of tropical ulcer consists in asepsis. Caustics and all antiseptics, even boric acid, do more harm than good, because they are irritating. Plasters and ointments are equally harmful.

The only treatment which I have found successful is the application of permanent aseptic dressings soaked in boiled water, and spraying with the same. Change of country is also an important element in cure.

When the lesion is seen in its earliest or papular stage, it should be destroyed with the thermo-cautery.

BOTRIOMYCOSIS.

This affection, first described by Poncet and Dor, is common to man and the horse; in the latter it constitutes "castration fungus." It consists of a small pedunculated tumour, the size of a pea or nut, and occurs most often on the palmar surface of the hands and fingers, and around the lips.

The lesion generally develops after a septic puncture, especially when a small foreign body remains in the wound. It has the appearance of a large fleshy bud, which bleeds easily, and may be covered with a yellow purulent crust. It often appears to be sessile, but the pedicle can be seen by raising it. The colour is dark red, the surface granular, and divided by fissures which bleed freely, so that the lesion may be compared to a raspberry.

At first sight, it might be mistaken for a *vegetating epithelioma.*

PATHOGENY.—According to Poncet and Dor, the lesion originates in a sweat gland, which becomes dilated, and transformed into a fleshy bud. But this view appears to be incorrect, for the tumour may appear in parts where sweat glands are completely absent; for instance, the free border of the lips.

Histologically, the lesion consists of granulation tissue, and numerous newly formed capillaries, with yellow granules in the centre, which constitute the characteristic element. These granules are not, as was formerly believed, the debris of a pathogenic fungus, but are due to hyaline degeneration of cells enclosed in a focus of chronic suppuration.

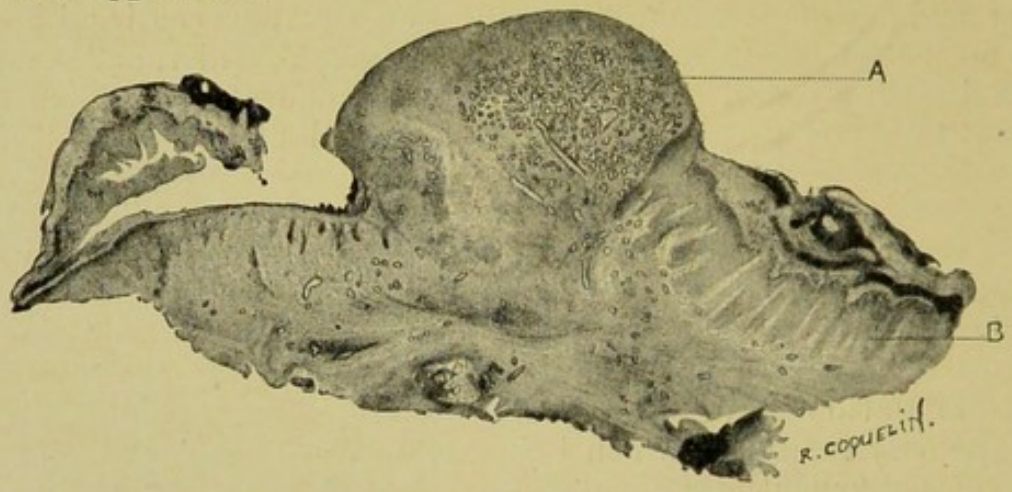


FIG. 132.—Section of botriomycosis. (Achard and Loeper.)
A, tumour formed by vascular tissue; B, epidermis.

Botriomycosis was at first regarded as a specific parasitic affection; but more recently it has been attributed to the ordinary agents of suppuration, especially the staphylococcus. Botriomycosis would, therefore, appear to be nothing more than a large fleshy granulation implanted in a focus of ordinary suppuration. According to Letulle, the inflammation may be caused by amœbæ, whose degeneration constitutes the yellow granules; the suppurative form may be due to superadded infection with *staphylococcus pyogenes.* In short, although the clinical characters of the affection are well known, its etiology still remains obscure.

TREATMENT.—This consists in ligaturing the pedicle and destroying the tumour by the thermo-cautery.

PERLÈCHE.

This is a special affection of the commissures of the lips. It nearly always affects both commissures, the epidermis of which

becomes white and wrinkled. The lesion may extend for a short distance above and below the commissure. The loosened epidermis is easily detached, leaving a fissure in the dermis at the angle of the commissure; sometimes there are two or three small secondary fissures above or below the principal fissure. The lesion does not extend much beyond the commissure, but there are sometimes small white epithelial detachments on the adjoining mucous membrane. In some cases the fissure becomes deeper and bleeds slightly; it then becomes covered with a crust, around which there is an inflammatory areola. The duration is usually two or three weeks, but may be four to six weeks. The lesion tends to heal spontaneously, but easily recurs; it leaves a characteristic reddish white surface for a long time after healing.

DIAGNOSIS. — Perlèche differs from *herpes* in the absence of vesicles. It somewhat resembles commissural *mucous patches*, but the latter consist of opaline erosions, and are accompanied by other signs of syphilis.

ETIOLOGY.—Perlèche is very contagious, and may be conveyed by kissing, or by using the same glass or serviette. It occurs at all ages, but is most common among school-children, who infect each other. According to Lemaistre, it is caused by a streptococcus in long chains (*streptococcus plicatilis*), which he cultivated and also found in water. Raymond has found the *staphylococcus albus* and *aureus*. But none of these microbes have been experimentally inoculated, either in man or animals.

TREATMENT.—The lesion may be touched with sulphate of copper or alum.

CUTANEOUS TUBERCULOSIS.

Pustulo-ulcerative Tuberculosis.

This affection, which I described for the first time in 1889, is the most attenuated form of cutaneous tuberculosis. It is as frequent as tuberculous gumma, and includes most of the cases formerly described under the name of *impetigo rodens*. It is constituted at first by pustules resembling those of common impetigo, formed by small epidermo-papillary collections of pus, deeper than impetiginous pustules, soon followed by yellow crusts, under which are round ulcers, deeper and more persistent than those of ulcerated impetigo.

These pustules occur on the face and neck, less often on the arms, thighs and buttocks. They are always isolated like those of *impetigo sparsa*, never agglomerated. The affection often coexists with other tuberculous lesions of the skin, especially with gummas, sometimes

with lupus tubercles; but there are no signs of visceral tuberculosis, and the general health generally remains good.

ETIOLOGY.—The tuberculous nature of these pustules has been proved by inoculation in animals. The pus, when inoculated in the peritoneum of the guinea-pig, always gives rise to a tuberculosis of slow evolution. On the other hand, I have never found the bacillus, either in the pus or on the surface of the ulcers.

DIAGNOSIS.—Pustulo-ulcerative tuberculosis may be mistaken for *impetigo*, and for certain *ulcerative syphilides*, either of acquired or hereditary syphilis. The latter are much deeper and more persistent. The diagnosis can be settled by experimental inoculation and by the therapeutic test.

TREATMENT.—The ulcers heal in three or four weeks after dusting with equal parts of powdered talc and boric acid, followed by a dressing of boric vaseline.

Tuberculous Gumma of the Skin.[1]

Gummas are sometimes isolated manifestations of tuberculosis, sometimes associated with glandular and visceral tuberculosis; but in both instances they result from general infection of the organism, and are not produced by direct inoculation of the skin, like lupus and tuberculous ulcers. They are observed at all ages, but especially in childhood and adolescence.

There are two varieties: *cutaneous gumma*, which represents the *dermic abscess* of the older authors, and *subcutaneous gumma*, which corresponds to the old *scrofulous gumma*.

SYMPTOMATOLOGY.—Tuberculous gummas begin in the form of small cutaneous or subcutaneous nodules, more perceptible to the touch than visible, over which the skin becomes red or livid. They are torpid, and hardly painful on pressure; they extend superficially, and may unite to form placards of varied extent; they then undergo softening, and, through one or more openings, discharge grumous and sometimes sanious pus, which dries into brown crusts, only slightly adherent and easily detached by the subjacent suppuration. Sometimes the opening is narrower than the base, which may communicate with the cavities of neighbouring gummas; sometimes the whole extent of skin over the gumma becomes ulcerated, forming a wide, deep, irregular ulcer with a sanious vegetating base. The gummas occur chiefly on the face, in front of the ears, and on the neck below the lower jaw. They are usually localised to a restricted area, but sometimes invade a considerable extent of the skin. Their evolution is slow, and when ulcerated, cicatrisation takes

[1] Prof. Gaucher uses the term *gumma* in connection with tuberculosis and sporotrichosis, as well as syphilis.—ED.

a long time to complete. The cicatrix is purple, irregular, and bridled.

PATHOLOGICAL ANATOMY.—Gummas present three stages in their evolution: *induration*, *softening*, and *elimination*. In the two first stages, microscopic examination shows characteristic tubercles with giant cells, epitheloid cells, round cells, and obliterated vessels. The gumma increases gradually in size, and undergoes rapid caseation and necrobiosis of the neoplastic tissue. These characters distinguish it from the *syphilitic gumma*, which is more circumscribed, has a greater tendency to become encysted, and undergoes fibrous transformation in places.

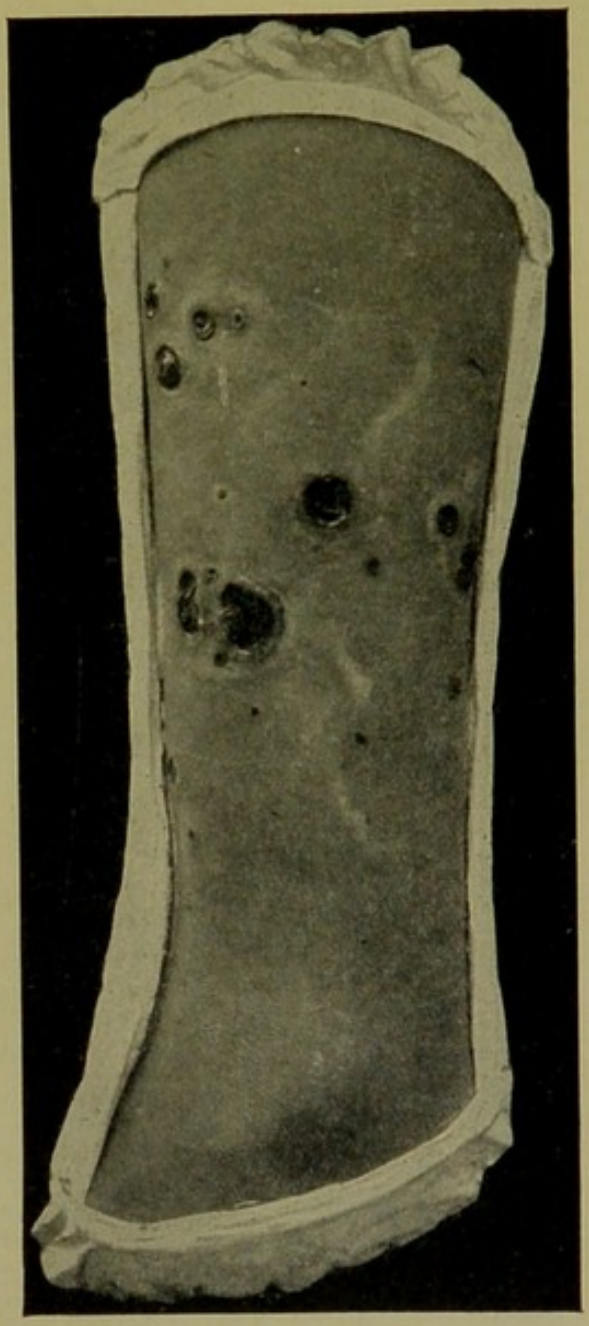


FIG. 133.—Open tuberculous gummas. (St Louis Hospital Museum.)

Sometimes a few bacilli are found in the walls of the gummatous cavity. The pus, when inoculated in the guinea-pig, generally produces tuberculosis. As the tuberculous gumma is not very virulent, inoculation is sometimes slow in producing its effects. In one of my inoculations the guinea-pig did not die till after four months.

PROGNOSIS.—This is always serious, because, even when the gumma is the only tuberculous manifestation present, visceral tuberculosis may develop later on.

DIAGNOSIS. — Tuberculous gumma must be diagnosed from *syphilitic gumma*. At the stage of induration this is only possible by the presence of other syphilitic or tuberculous lesions; it is sometimes necessary to resort to the therapeutic test. At the stage of softening, the diagnosis is easier; the tuberculous gumma soon opens externally, the syphilitic gumma evolves more slowly, and may undergo absorption under the influence of specific treatment. At the period of ulceration the diagnosis is also easy: the syphilitic ulceration is

more circular, with hard, punched-out borders, and its base is covered with a gray pseudo-membranous coating, or with thick, greenish, stratified crusts; lastly, it is surrounded by a brown or copper-coloured areola. The cicatrices of tuberculous gummas are irregular

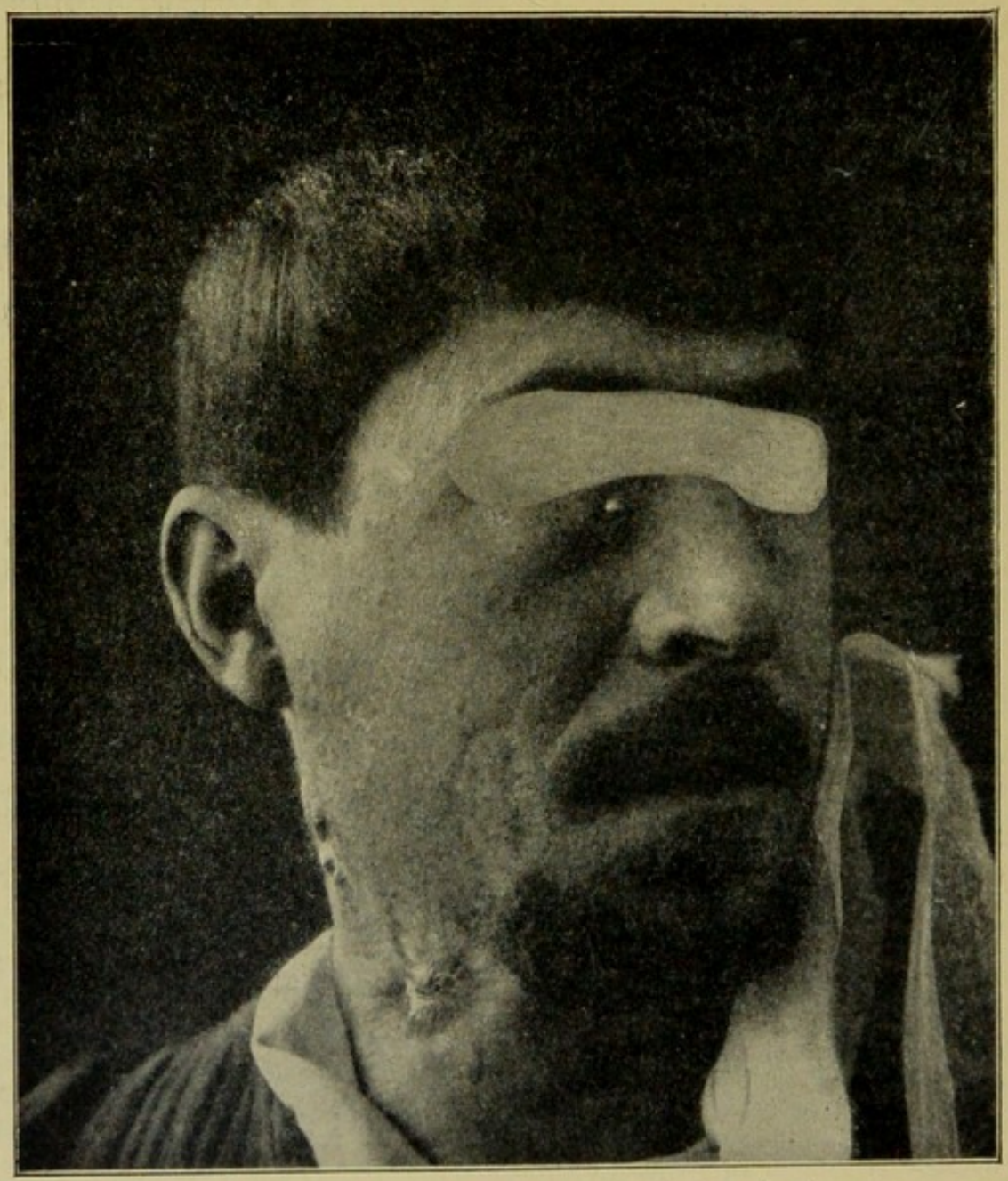


Fig. 134.—Tuberculous gummas and adenitis of the face and neck.

and purple; those of syphilis are pigmented, round and regular. The diagnosis between tuberculous gummas and those of congenital syphilis is still more difficult, in the absence of other signs of congenital syphilis, such as interstitial keratitis and Hutchinson's teeth.

The diagnosis from *sporotrichosis* will be mentioned with the latter affection. Tuberculous gummas may be confounded with *erythema nodosum*, but the latter presents more numerous lesions, which never undergo softening, and are only found on the limbs.

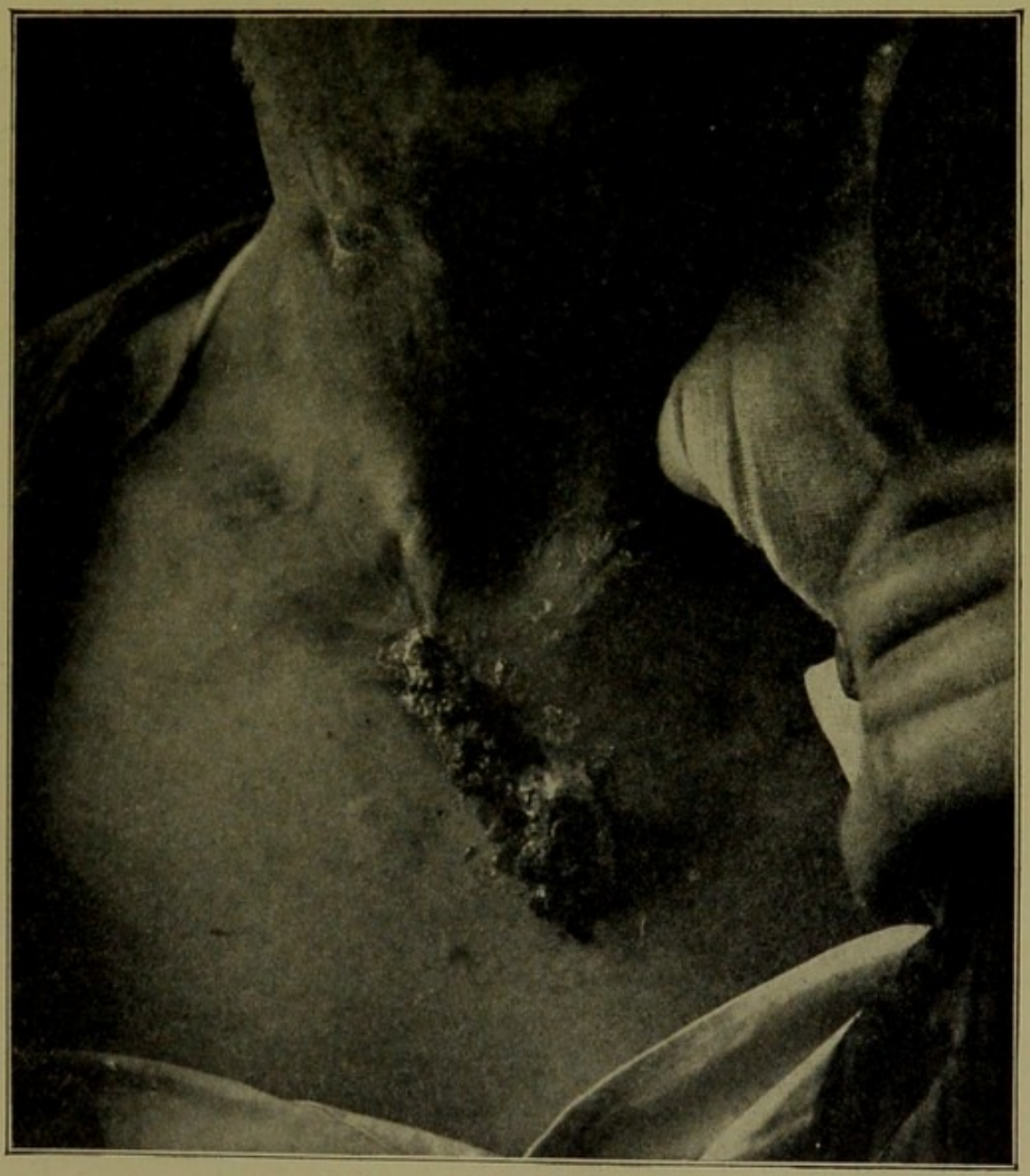


FIG. 135.—Tuberculous gummas of the neck and chest.

Fibroma is always hard. *Sarcoma* is accompanied by shooting pains, which are absent in gumma; moreover, it ulcerates by progressive invasion of the skin, and not by softening.

TREATMENT.—General treatment is the same as that of tuberculosis in general; cod-liver oil, iodo-tannic syrup, arsenic, etc.;

mineral waters, such as those of Biarritz, etc., are also useful. In the early stages, this treatment may prevent softening of the gummas and lead to their resolution.

Local treatment.—Tincture of iodine should first be applied. When the skin threatens to give way, the abscess should be opened. When the gummas have opened, artificially or spontaneously, a concentrated solution of nitrate of silver or chloride of zinc should be applied to the cavity, after scraping with a sharp spoon. But the best treatment consists in applying successively crayons of metallic zinc and silver nitrate to the whole wall of the cavity. Large ulcerated surfaces may be scraped and cauterised with silver nitrate or chloride of zinc, or treated with silver and zinc crayons, and dressed afterwards with iodoform, iodol, or aristol.

Tuberculous Ulcer of the Skin.

Tuberculous ulcer may develop in two ways: (1) it may result from accidental inoculation in healthy subjects, without previous tuberculosis; for example, inoculation of excoriations on the hands and forearms by contact with contaminated linen, etc., in nurses and others; (2) it may occur in patients affected with pulmonary or intestinal tuberculosis; in this case it is an auto-inoculation of small erosions by bacilli contained in the sputum or stools. The latter is the more frequent mode of origin. This inoculation explains the frequency of tuberculous ulcers on the lips, tongue and bucco-pharyngeal mucous membrane, and on the nostrils and around the anus. In the same way, tuberculous ulcer of the vulva is secondary to tuberculosis of the uterine appendages, and tuberculous ulcer of the penis to tuberculosis of the genito-urinary tract. In these cases, inoculation is due to contact with the genital secretions or urine containing tubercle bacilli. Tuberculous ulcer has also been observed on the face and upper limbs, more rarely on the lower limbs.

Symptomatology.—At the point of inoculation a small nodule develops, which ulcerates and discharges a little caseous pus. The ulcer extends by the production of fresh nodules, which soften and ulcerate in their turn. But, as a rule, the ulcer is already constituted when the patient is first seen. It is usually small, about half an inch in diameter; when situated on the lower lip or anus, it may be still smaller, but on the limbs it may attain considerable dimensions. The lesion does not extend deeply, and its base is neither infiltrated nor indurated; its shape is irregular, its borders red or livid and sharply defined, not undermined like those of ulcerated gumma. The base is granular and covered with pale flabby granulations, among which are yellow miliary tubercles the

size of a pin's head, some hard, others undergoing caseation. Occasionally the ulcer is covered with a soft crust, slightly adherent. The ulcer is painful on pressure and on movement; when situated on the lips, it causes trouble in mastication; when situated at the anus, it renders defæcation painful and causes tenesmus.

As a rule, the ulcer is single on the same region, but it presents different characters according to its situation. On the lower lip there may be several ulcers, median and lateral; on the upper lip the ulcer occupies the free border and remains small, or it may extend to the ala of the nose; at the anus it is sometimes fairly large and extends into the rectum.

The cervical glands are affected in the case of ulcer of the lips, and the inguinal glands in the case of anal ulcer. The glands do not usually suppurate, if the cutaneous lesion is secondary to visceral tuberculosis; but when inoculation has taken place on a healthy subject, the glands swell rapidly, undergo caseation, and open externally. The cutaneous lesion may become the point of origin of general tuberculosis.

Prognosis.—This is grave. In subjects who have had anterior tuberculosis, tuberculous ulcers seldom heal.

Pathological Anatomy.—Renaut and Vallas have described two forms of tuberculous ulcer of the skin. In the first, the granulations contain chiefly embryonic nuclei and very few giant cells; the interstitial tissue undergoes caseous degeneration, the vessels are obliterated, and the ulcer extends gradually at its borders. In the second form, there are complete tuberculous follicles in the dermis, but the diffuse interstitial inflammation is absent; the perifollicular tissue of the dermis undergoes slow necrobiosis, ending in ulceration.

A few bacilli are sometimes found in the exudation and in the granulations. Inoculation of the exudation and of pieces of granulation tissue, in the guinea-pig or rabbit, gives rise to tuberculosis.

Diagnosis.—Tuberculous ulcer is easily distinguished from ulcerated *lupus*, which usually affects the face, and presents small tuberculous nodules, the colour of barley sugar, around the principal lesion; also more or less tuberculous infiltration. It should not be mistaken for *hard chancre*, which has an indurated base, no tendency to extend, and is accompanied by glandular induration. *Mucous patches* are multiple and superficial. *Tertiary syphilitic ulcers* are sometimes very extensive, and cicatrise in places while they extend at other parts; their cicatrices are smooth and pigmented; the base of the syphilitic ulcer is not covered with yellow granulations like the tuberculous ulcer, but with a hard, greenish, stratified crust; the surrounding skin is infiltrated and copper coloured. In doubtful cases, bacilli may be found in the tuberculous ulcer. *Simple chancre* is distinguished by its undermined borders and suppurating surface,

by the absence of yellow granulations, and by the presence of suppurating bubo. *Epithelioma* is characterised by a red, painful, fungating ulcer, which bleeds easily, and has hard raised borders.

TREATMENT.—In healthy subjects who have been accidentally inoculated with tuberculosis, the lesion should be cauterised with the thermo-cautery or galvano-cautery. Instead of this, camphorated naphthol or pure lactic acid may be applied daily, the former being the better of the two. Total ablation of the ulcer is not advisable, as it nearly always recurs. In advanced cases of phthisis, iodoform or aristol ointment may be applied, mixed with 1 or 2 per cent. cocaine.

Tuberculous Lupus.

Tuberculous lupus, or *lupus vulgaris*, is characterised by small superficial nodules or tubercles imbedded in the dermis. These nodules, or lupus tubercles, are covered by the epidermis, but are visible through it; they are more or less raised above the surface, according to their depth. They are round in form, of a reddish yellow or barley-sugar colour, translucent, soft and friable, and vary in size from a pin's head to a millet seed. They are very vascular, and bleed easily when torn. Sometimes the vessels are almost as numerous as in a nævus (angiomatous or telangiectasic lupus). The tubercles are usually painless, but sometimes sensitive on pressure. They may be isolated from one another on a small area or on an extensive surface, or confluent, forming irregular patches of varied extent. Each of these patches extends slowly and centrifugally. While the periphery extends, the centre of the patch undergoes modifications; it may ulcerate superficially or deeply, or may undergo cicatricial atrophy without ulceration. This cicatricial condition is characteristic, and is always present after a certain time in lupus of any extent, considerably assisting in the diagnosis.

Lupus tubercles are often obscured by secondary lesions; general infiltration of the skin, crusts, vegetations, and inflammatory lesions. They are sometimes surrounded by yellow spots which are easily enucleated; these are grains of milium (Besnier). The combination of the primary lesion with these secondary lesions gives rise to different appearances, which may be divided into two main groups, according as the lupus tends to cicatricial atrophy (non-ulcerative lupus or *lupus non-exedens*), or to ulceration (ulcerative lupus or *lupus exedens*). However, these two forms are not always distinct, for lupus may be at first non-ulcerative and afterwards ulcerative, or non-ulcerative in one part and ulcerative in another.

Non-ulcerative Lupus.—This presents three varieties: flat lupus, raised lupus, and elephantiasic lupus.

Flat Lupus.—In this form the tubercles sometimes project so slightly that the affection may be confused with lupus erythematosus. Flat lupus generally occurs on the cheek. It begins as a very small patch, which may increase by extension or by confluence of several

Fig. 136.—Squamous (psoriasiform) lupus.

patches; it may extend over a great part of the face. The patch is reddish brown and sharply defined, with a smooth, glistening, varnished surface, sometimes covered with small or large squames (pityriasiform and psoriasiform lupus). In some cases the patches undergo a kind of colloid infiltration and become very transparent (colloid lupus). This form of lupus is generally painless, but is

sometimes very pruriginous. After a time, it shows a tendency to cicatricial atrophy, a white cicatrix being formed in the centre of each patch. But, at the periphery, the lesion often extends by the formation of yellow tubercles. Its progress is very slow; sometimes it becomes cured by cicatricial atrophy of the whole patch, but this may be interrupted by a relapse. As a rule, the affection lasts for life, unless it is treated.

Lupus Vulgaris.—This form, which is accompanied by infiltration of the skin, constitutes the ordinary form of lupus. The

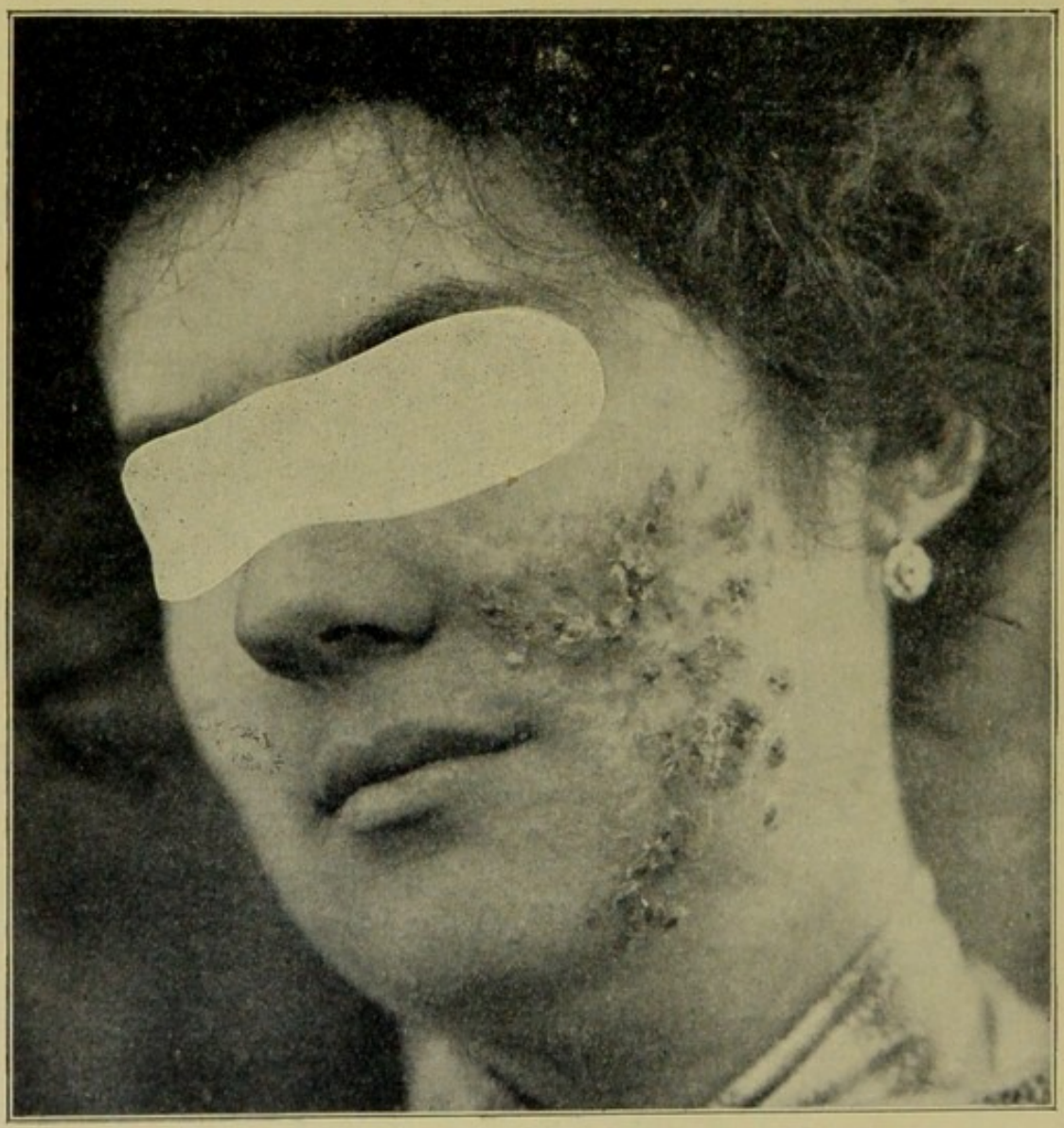


FIG. 137.—Lupus vulgaris.

primary nodules form soft elastic elevations, isolated or united to form round patches, of a pale red or dark red colour, and the size of a pea or nut. Besides these projecting nodules, there are deeper

ones, which infiltrate the dermis and even the subcutaneous tissue; these are only appreciable by palpation.

The nodules are of different ages, some old and some recent, sclerosed in some places, vegetating in others. At their periphery

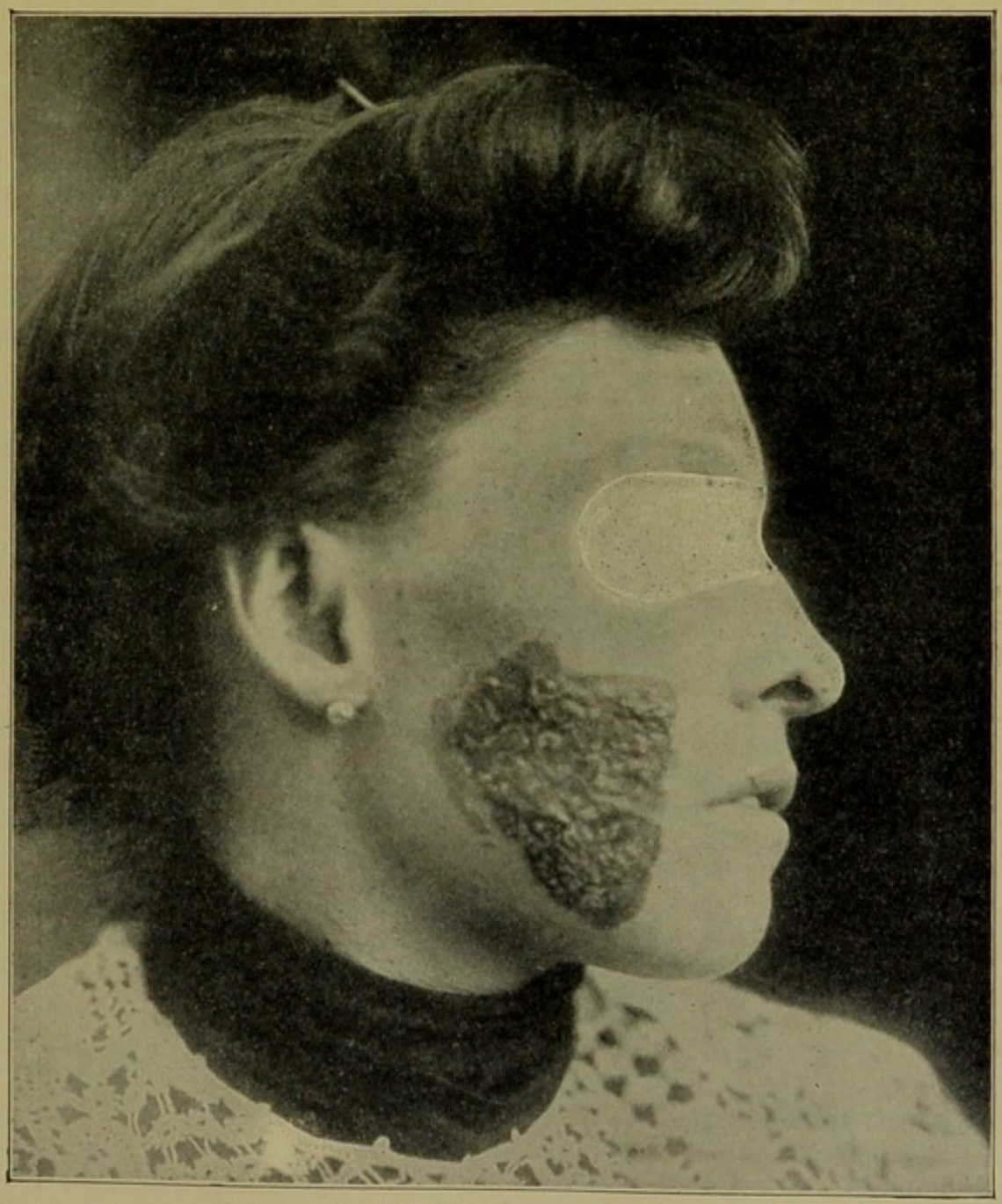


Fig. 138.—Circumscribed lupus.

there is an inflammatory reaction, with excessive vascularisation of the dermis and sometimes lymphatic œdema of the dermis and subcutaneous tissue. The surrounding skin is thickened and purple.

Lupus vulgaris may become *vegetating;* if it affects the nose, the latter becomes transformed into a nodular swelling covered with a greenish crust, under which are found red fungating granulations. Vegetating lupus also occurs on the lips and may extend from there to the face, which becomes red and covered with vegeta-

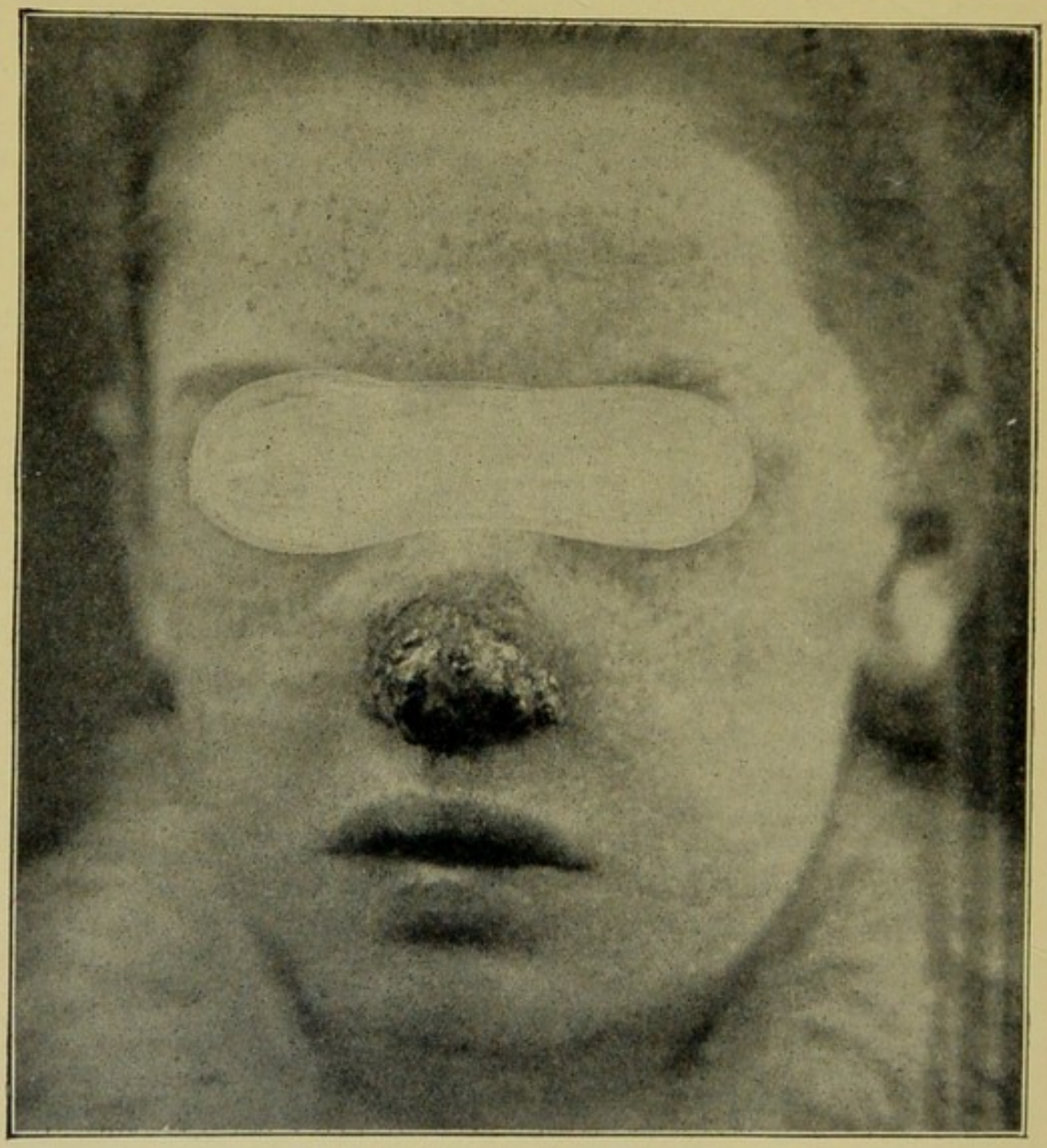


FIG. 139.—Vegetating lupus.

tions and nodosities, simulating hypertrophic acne rosacea, or even tubercular leprosy.

Elephantiasic Lupus.—The tuberculous infiltration of the skin and œdema of the subcutaneous tissue attain their maximum in this hypertrophic form of lupus, which occurs usually on the lower limbs, sometimes on the upper limbs and face. Elephantiasic lupus

of the limbs is often accompanied by lupus vulgaris of the face, the presence of which aids the diagnosis. The elephantiasic condition is due to defective circulation, especially in the lower limbs, and to the acute attacks of inflammation, recurrent erysipelas, and chronic lymphangitis which often complicate lupus.

After each attack of lymphangitis, the œdema and induration of the skin increase; the leg becomes twice its normal size, and its natural prominences disappear; the foot is swollen and separated from the leg by a deep furrow; the toes are deformed and buried in the elephantiasic mass, from which their extremities project. The surface of the lupus is sometimes glistening, sometimes thick and horny. Papillary hypertrophies may be present; sometimes red and bleeding with suppurating fissures, sometimes dry and covered with stratified horny tissue, like lupus verrucosus. On this lupoid mass are found ulcerations, cicatrices, and tubercles of different sizes and ages.

On the upper limbs, elephantiasic lupus gives rise to similar lesions, but less pronounced; the fingers are swollen and deviated, and present deep ulcers, which may extend to the bones and cause caries. On the face, it causes much deformity; the cheeks are swollen and pendulous, the lips thickened, the mouth contracted, the eyelids swollen, the ears enlarged and deformed.

This form of lupus may also affect the genital organs, especially the penis, which is swollen and infiltrated. On the vulva, it causes considerable deformity, and constitutes the condition formerly known as *esthiomenus*.

Ulcerative Lupus.—This includes two varieties: tuberculo-ulcerative and pustulo-ulcerative.

Tuberculo-ulcerative Lupus.—When lupus ends in ulceration, the epidermis becomes thin and broken, the tubercles undergo caseous degeneration and give rise to round ulcers. The borders of the ulcer merge insensibly into the adjacent skin; sometimes they are undermined, or covered with fungating, bleeding granulations. The skin round the ulcer is purple, and infiltrated with tubercles. The ulcers may be single or multiple; they are generally shallow, but sometimes more deeply excavated. The base is yellowish gray, friable and infiltrated with neoplastic tissue, covered with granulations and a sanious or purulent liquid, which dries into crusts, sometimes thin and gray, sometimes thick and yellow, sometimes as thick as in the pustulo-ulcerative form. The evolution of the lupus ulcer varies; it may remain superficial and circumscribed, or it may extend both superficially and deeply, constituting two more varieties—serpiginous lupus and phagedenic lupus.

In *serpiginous lupus*, the ulceration pursues an irregular course, extending gradually by softening of the tubercles at its borders,

cicatrising at one part and extending at another, and leaving cicatrices, still infiltrated in places with tubercles, which may again soften and ulcerate.

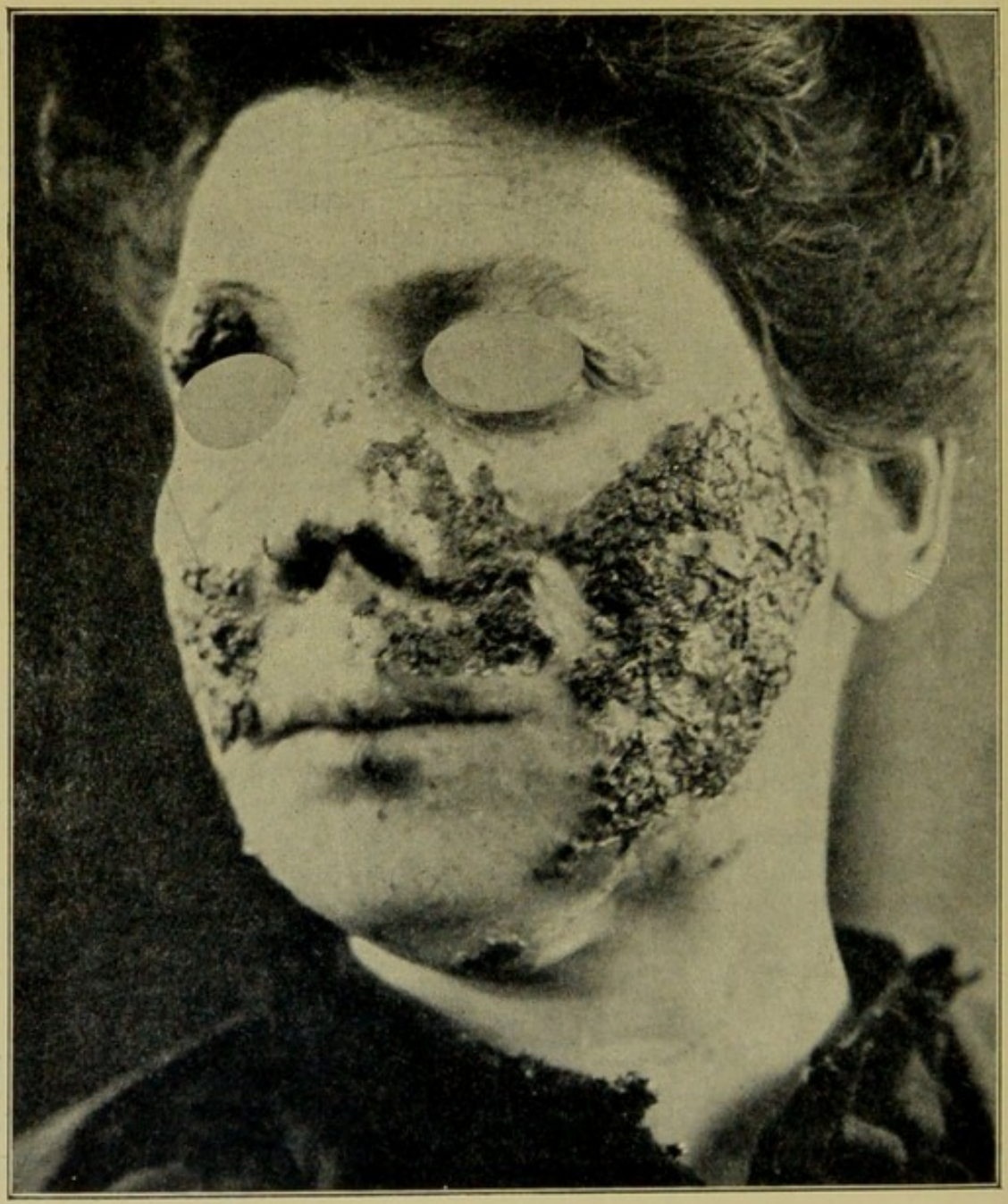


FIG. 140.—Ulcero-crustate lupus.

Phagedenic lupus nearly always occurs on the face. It begins on the end or side of the nose, sometimes inside the nares. The tubercles soften rapidly and give rise to red, fungoid ulceration, which gradually destroys the nose, causing great disfigurement. Lupus of the nose may invade the hard palate and perforate the

bone, so that the buccal cavity communicates with the nasal fossæ. The ulceration may destroy the lips and invade the gums and the floor of the mouth.

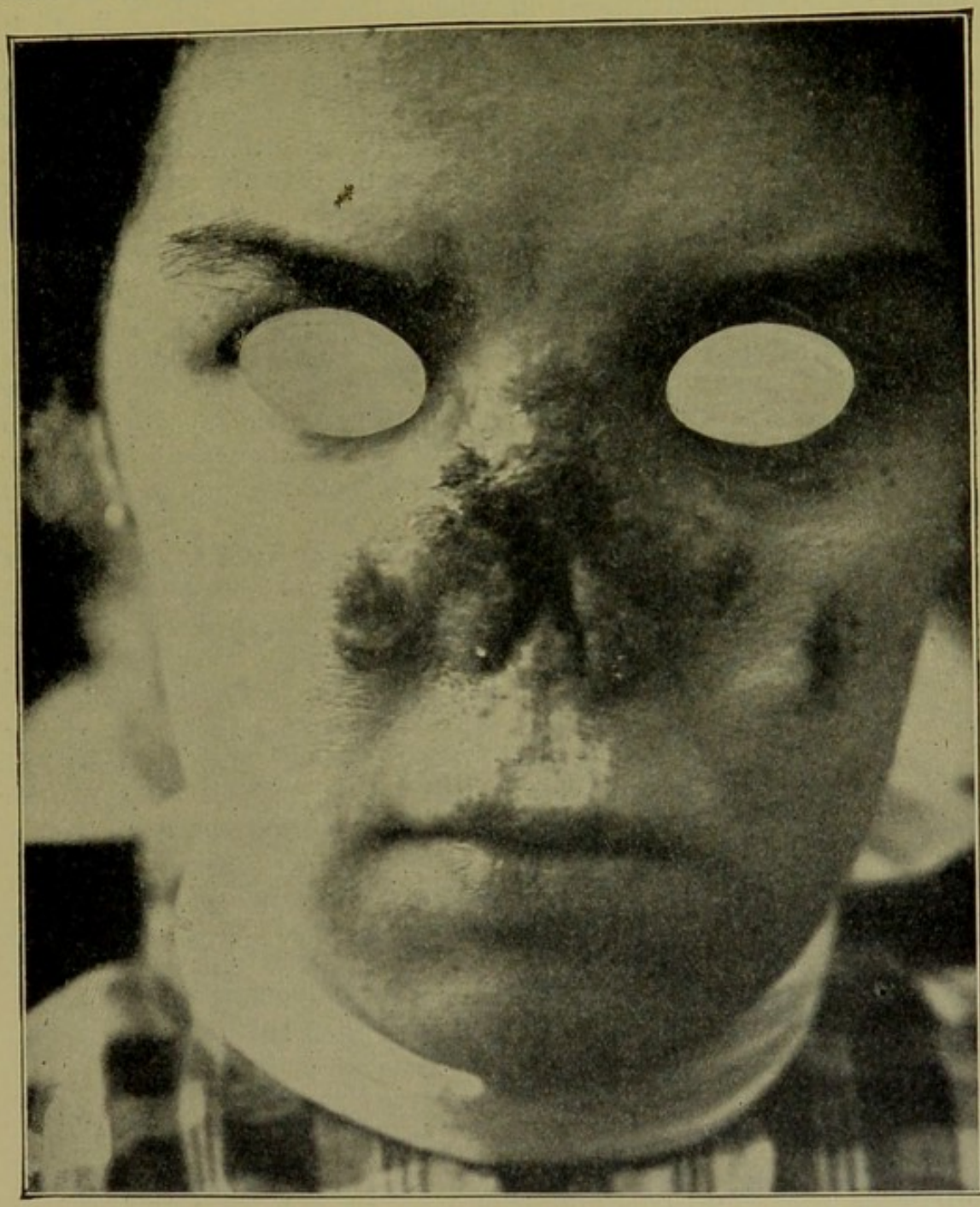


Fig. 141.—Phagedenic lupus.

Ulcerative lupus, both serpiginous and phagedenic, may extend to the eyelids, which are destroyed and everted, especially the lower eyelid. It may invade the conjunctiva and cause adhesion of the latter to the eye, or it may destroy the eye itself. The ear may also be destroyed, or contract adhesions to the scalp.

This form is occasionally observed on the extremities, and may cause destruction of one or more fingers (*lupus mutilans*).

Pustulo-ulcerative Lupus.—This form is characterised by a red patch of lupoid infiltration, on which develop secondary pustules, sometimes small and confluent, sometimes large and isolated. The latter soon rupture and discharge pus, sometimes mixed with blood, forming crusts of varied thickness, sometimes stratified (impetiginous or rupial lupus). This form has a rapid evolution, but does not produce such deep and extensive lesions as tuberculo-ulcerative lupus. It occurs chiefly on the face.

Lupus of the Mucous Membranes.—Lupus may attack the mucous membranes, either by extension of cutaneous lupus or independently, or it may coexist with lupus of the skin. The mucous membrane is red, and covered with small fungosities and ulcerations, according to the stage of the disease. It occurs chiefly on the lips and gums, rarely on the tongue, thus differing from syphilis, which often affects the tongue. It is also met with on the soft palate, pharynx and larynx, and has been observed in the nasal fossæ.

Evolution.—Lupus generally begins in youth; it is usually slow in evolution, and lasts many years. The general health remains unaffected for a long time, but numerous complications may arise.

Lupus can only be cured by the production of cicatricial tissue. Non-ulcerative lupus undergoes a kind of fibrous transformation. In ulcerative lupus the cicatrix, instead of being atrophic as in the preceding form, is hypertrophic and irregular, and may become transformed into cheloid. New tubercles often develop at the border of the cicatricial tissue, and give rise to fresh ulceration.

Complications.—The glands and lymphatic vessels corresponding to the lupus area may be affected with tuberculous adenitis and lymphangitis. Visceral tuberculosis may also occur, especially chronic pulmonary tuberculosis, sometimes acute tuberculosis, the latter being sometimes due to treatment by scarification or scraping, methods which may facilitate the entrance of tubercle bacilli into the general circulation.

Other complications are lymphangitis and erysipelas, which sometimes have a beneficial influence on the lesion; the same result is observed after variola, or any other cutaneous suppuration which produces a substitutive inflammation. In old people, ulcerated lupus may be complicated by epithelioma, in the form of a vegetating tumour which rapidly invades the glands, and is accompanied by severe pain.

Prognosis.—Lupus is a severe disease, which is difficult to cure, and very liable to recur. Moreover, there is always the possibility of visceral or pulmonary tuberculosis.

DIAGNOSIS.—The diagnosis between lupus and *tuberculous ulcer* has already been discussed. The diagnosis from the *tubercular syphilide* is difficult; but the syphilitic nodules are hard and brown or copper coloured, not soft and reddish yellow like those of lupus; they are less friable, do not bleed, and are more circumscribed. In the *pustulo-ulcerative syphilide* the crusts are greenish, the ulcers are more regular, and not surrounded by the purple areola seen in lupus ulcers. The cicatrix of syphilitic ulcer is more regular, and surrounded by a brown areola; moreover, syphilitic lesions have a more rapid evolution than lupus. In some cases the therapeutic test is necessary. *Ulcerated epithelioma* is sometimes difficult to distinguish from ulcerated lupus; but the ulceration is irregular, with an indurated base and ragged, everted and raised borders; it bleeds easily, gives rise to lancinating pains, and is accompanied by glandular swelling. The tubercles of *leprosy* may be confounded with lupus, especially in negroes; but in leprosy anæsthesia is present. In leprosy localised to the ear, the lobule is often enlarged, but always remains free and pendulous; while, in lupus, the hypertrophied lobule is always adherent to the adjacent skin. It is impossible to confuse lupus for *impetigo*, in which there is no infiltration of the skin nor ulceration. *Sycosis* may be sometimes mistaken for lupus, and inversely when the latter occurs in hairy regions; but in sycosis the lesions are inflammatory and painful, the hairs are altered and broken, and in trichophytic sycosis spores can be found by microscopic examination. Elephantiasic lupus of the limbs must be distinguished from *elephantiasis Arabum;* lupus begins in childhood, and in the midst of the elephantiasic lesions lupus tubercles can be found, ulcerated or not, but always characteristic.

PATHOLOGICAL ANATOMY.—The lupus neoplasia is essentially tuberculous in nature. In the nodules disseminated in the dermis are found typical tuberculous follicles with giant cells and epithelioid cells in the centre and embryonic cells at the periphery. Embryonic cells are also present around the vessels, glands and hair follicles. Tubercle bacilli can be found in the follicles, especially in the giant cells, but they are few in number, and it is sometimes necessary to make many preparations to find them.

Secondary lesions include: elongation of the interpapillary processes; hypertrophy of the papillæ, which are swollen by embryonic tissue; dilatation of the vessels, with thickening of their walls and sometimes obliteration of their lumen; vacuolar degeneration of the cells of the Malpighian layer. When ulceration is impending the nodules undergo caseation and softening, like all tuberculous tissue.

The tuberculous nature of lupus is proved by inoculation, which is always positive when performed under suitable conditions.

Inoculation of lupus tissue on the peritoneum of the guinea-pig is nearly always positive, but subcutaneous inoculation generally fails. Leloir's method of *dermo-epiploic grafting* is a good one; this consists in making an incision in the abdominal wall, and introducing pieces of lupus tissue, which are thus situated both on the peritoneum and in the wound. The rabbit is more refractory to inoculation, and the only means of producing tuberculosis is by inoculating the anterior chamber of the eye.

ETIOLOGY.—Lupus may develop in subjects who are already tuberculous; affected with tuberculous adenitis or arthritis, or pulmonary tuberculosis; it is then the cutaneous manifestation of a more or less general tuberculosis. It may also be the first sign of hereditary or acquired tuberculosis. But, in the majority of cases, lupus is due to cutaneous inoculation, either from another patient, or by auto-inoculation of the patient himself, already affected with pulmonary tuberculosis, with the products of expectoration. Inoculation may take place on a simple excoriation, or on excoriations secondary to eczema or impetigo.

However, although most cases of lupus are due to cutaneous inoculation, it may also arise from internal infection. I have observed the following case, which proves the possibility of the internal origin of lupus: A patient was operated upon for lupus of the cheek, by the knife, and the wound was repaired by a skin graft from the arm; four years afterwards lupus tubercles appeared on the grafted skin. Again, lupus is much more common in tuberculous families, even when the subjects affected show no other sign of tuberculosis.

Lupus is observed at all ages, but most often begins in youth. It is usually situated on the exposed parts, especially the face, but may occur on any part of the body, on the trunk or limbs.

TREATMENT.—Lupus requires both internal and external treatment.

Internal Treatment.—This is the same as for all forms of tuberculosis. Cod-liver oil in large doses gives the best results, if the patient can support it. Iodo-tannic syrup may also be prescribed. Hardy recommended chloride of sodium in doses of 45 to 75 grains daily. Arsenical preparations or creosote are also useful. I often give the following preparation:—

Phosphate of lime	3 parts
Pearson's solution	2 „
Iodo-tannic syrup	100 „

Sulphur waters and residence at the seaside are also beneficial.

External Treatment. *Curetting.*—This consists in removing the diseased tissues by means of a sharp spoon; hæmorrhage is arrested by the thermo-cautery, and the wound dressed with

iodoform. This method is only suitable for small patches of lupus, for it leaves rather extensive cicatrices; moreover, it does not always remove all the diseased tissue, and is also liable to cause secondary tuberculous infection.

Excision.—This gives good results in small patches of lupus. The wound may be covered with skin grafts, which have the disadvantage of leaving unsightly cicatrices, or better, by sliding and suturing the skin, which gives the most æsthetic result.

Scarification.—This method was introduced by Balmanno Squire, who used a scarifier with several blades. Vidal recommends a single blade. As the operation is painful, local anæsthesia may be induced by the chloride of ethyl or ether spray. The scarifications consist in parallel linear incisions, intersected by other series of incisions at an acute angle to the first. The incisions should be deep enough to penetrate all the diseased tissues. Small isolated patches may be treated at a single sitting, but for larger patches the operation should be repeated once a week. Hæmorrhage is stopped by wool pressure, and the wound treated with sprays of boracic lotion.

This method is tedious, but gives very good cicatrices, and is suitable for lupus of the face; on the other hand, it may expose the patient to the risk of secondary tuberculous inoculation; but this is rare.

Thermo-cautery.—This may be used in the form of cauterisation *en masse*, or in the form of punctiform cauterisation. In the latter method, a fine point is used and the punctures made perpendicularly and close together. Cauterisation *en masse* is more painful, but gives better results.

Galvano-cautery.—This causes less burning than the thermo-cautery. The punctures should be deep and close together; the operation should be repeated when the punctures of the previous sitting have healed. After cauterisation, the wound is sprayed with boracic lotion. The cicatrices after cauterisation are less æsthetic than those after scarification, but cauterisation has the advantage of being free from the risk of secondary tuberculous infection.

Chemical caustics.—Caustics are now seldom used except for lupus erythematosus. Arsenical paste has been abandoned, on account of the danger of arsenical intoxication. Lailler used an ointment of biniodide of mercury and iodide of potassium:—

Biniodide of mercury Iodide of potassium	aa 1 part
Lard	200 parts

A solution of monochlorphenol in absolute alcohol (20 per 100) has given good results, in a case of widespread lupus of the face. Perchloride of mercury has been recommended, in the form of com-

presses soaked in a 1 in 1000 solution, or in the form of interstitial injections of a few drops of a 1 per 100 solution. Lactic acid often gives good results in ulcerated lupus and in lupus of the mucous membranes. Butte recommends painting with a solution of permanganate of potassium (2 to 5 per 100). Hallopeau applies this solution before proceeding to linear scarification.

But of all caustics, the best is *absolute phenol.* A solution of 25 parts of pure anhydrous phenol in 5 parts of pure ethyl alcohol (95 per cent.) is painted on the surface of the lupus, which must be perfectly dry (because a mixture of phenol and water produces scars). These applications are repeated every three or four days; during the intervals carbolised oil (12 per 100) is applied. This method is perhaps superior to all the above.

Treatment of Lupus by New Methods.—This comprises phototherapy, radiotherapy and radiumtherapy.

Phototherapy.—Sunlight and the light from an electric arc contain heat rays, light rays, and chemical or actinic rays. The properties of the chemical rays of the spectrum suggested to Finsen the treatment of lupus by phototherapy.

This treatment is based on the following facts:—(1) The chemical rays of the spectrum, that is the ultra-violet and the violet and blue rays, possess bactericidal properties; they also exert an inflammatory action on the tissues, as shown by solar and electric erythema and the erythema of pellagra. (2) These rays act not only on the surface of the skin, but also penetrate it. (3) The rays penetrate tissues more easily when they are deprived of blood.

Solar Phototherapy.—This may be carried out in countries where there is sufficient sunlight. The rays are condensed and concentrated by means of a large hollow lens, formed of two plates of glass 10 or 12 inches in diameter, one of which is flat and the other convex, and which are united by a metallic ring about 3 inches wide. The cavity of the lens is filled with sulphate of copper, the blue colour of which absorbs the heat rays. The sun's rays are focussed by the lens on to the diseased tissue.

Electric Phototherapy.—The sun's rays may be replaced by a voltaic arc with constant current of 60 to 80 amperes intensity. This necessitates the use of a complicated apparatus. Finsen's apparatus consists of a stand formed by a metal ring, above which is the arc lamp. This ring carries four tubes 2 feet long, placed at equal distances apart, so that four patients can be treated at the same time. These tubes consist of two distinct parts: (1) the upper part is provided with a system of quartz lenses at the end next the lamp, by which the light is concentrated; (2) in this slides another tube, the terminal part of which is slightly conical and forms a cavity limited above and below by lenses of rock crystal 12 inches

apart, which form a condenser. This cavity is filled with the blue solution of sulphate of copper, and its walls are surrounded by a metal sheath in which circulates a current of cold water, to prevent heating of the solution by the heat rays. Thus, the blue, violet and

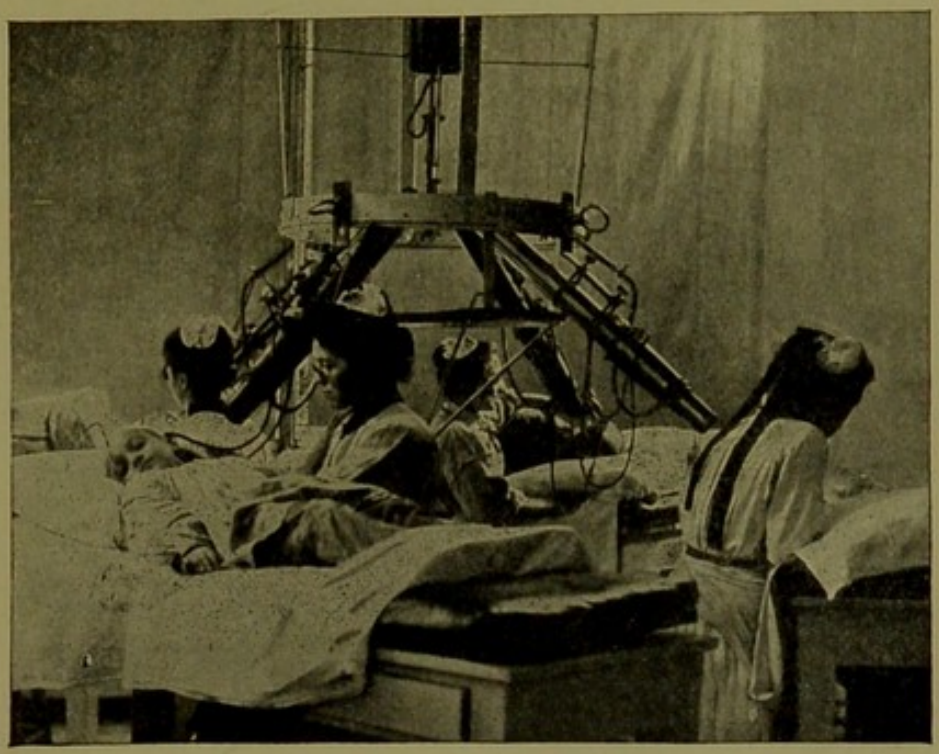


Fig. 142.—Finsen's apparatus.

ultra-violet rays are concentrated by the lenses and cooled by the current of water, while the yellow and red rays are absorbed by the blue solution.

To allow the chemical rays to act more effectively on the tissues, the latter are rendered anæmic by compression. This is effected by hollow lenses of quartz, through which circulates a current of cold water, which cools the skin as well as the light rays. The lens is applied firmly to the part to be treated.

Fig. 143.—Finsen's compressor.

The technique consists in focussing the rays of light on the part to be treated, and applying the compressor for about an hour. In this way an area of about half an inch is treated at each sitting, and this area should not be treated again for one or two weeks.

The treatment is not painful; the immediate effect produced consists in redness and swelling of the skin, and after twenty-four to forty-eight hours a bulla similar to that produced by a blister.

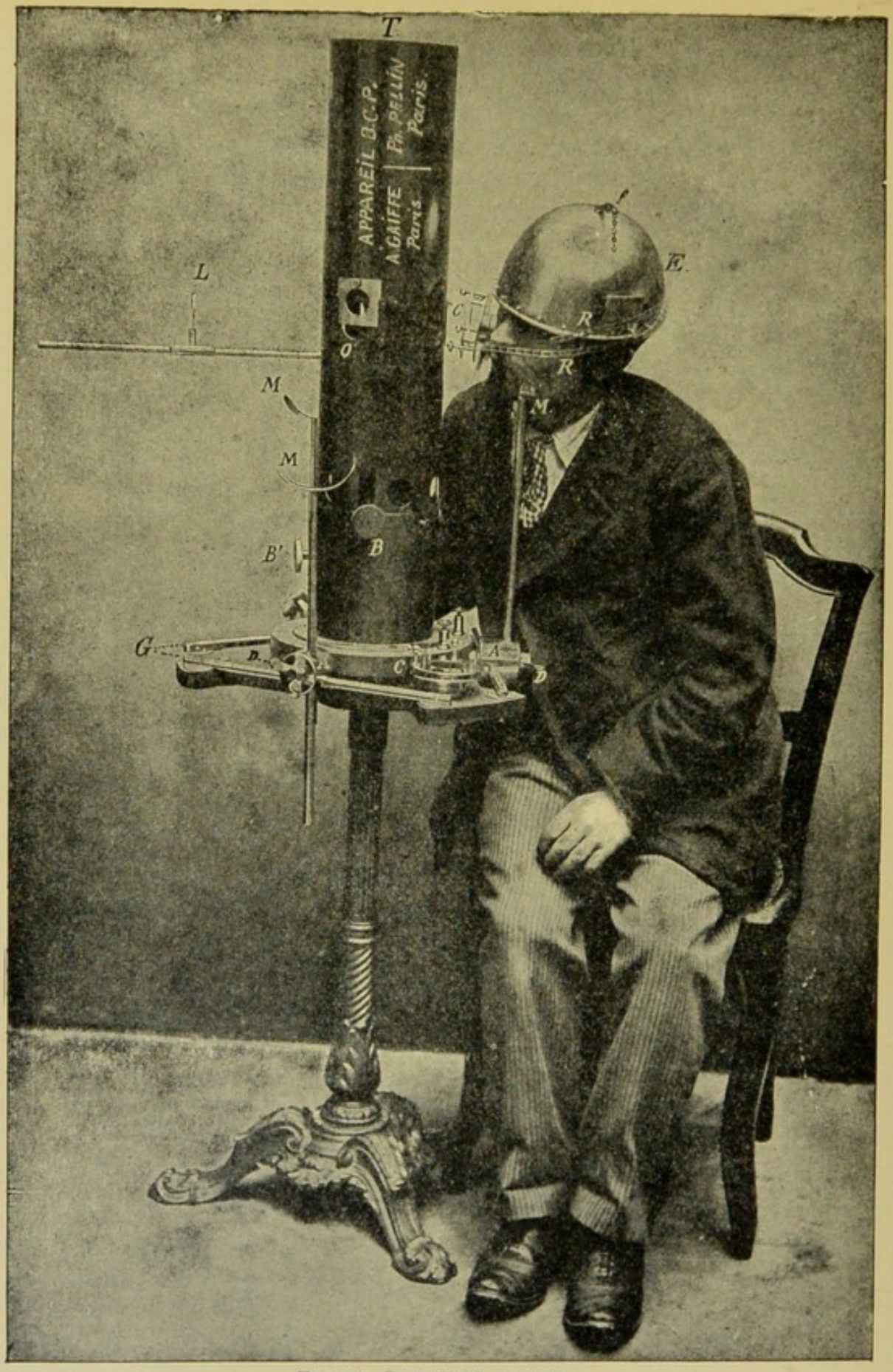


FIG. 144.—Broca-Chatin apparatus.

A, clamp holding chin supports M, M; B, B', screws by which the electrodes can be moved vertically and horizontally; C, compressor with quartz lens; E, helmet; G, horizontal rods bearing clamp A; L, crystal lens projecting an inverted image of the electrodes on the wall or on a screen; O, one of the three openings through which pass the ultra-violet rays, and opposite which is placed the compressor C; T, chimney covering the whole.

Before each sitting the lesion should be cleansed with boiled water. If the bulla is followed by a crust, the latter should be removed by means of moist dressings.

The curative effect is good, but it generally takes several months to produce, if the lupus is at all extensive.

Simpler apparatus than that of Finsen have been devised, such as the Lortet-Genoud. The Broca-Chatin apparatus is simpler still.

Bang invented an apparatus in which the arc is not produced by ordinary carbons, but by iron electrodes, which, according to Bang, give an arc very rich in chemical rays. André Broca, improving on Bang's apparatus, uses a negative electrode of carbon, but a positive electrode formed by a sheath of carbon enclosing a rod consisting of an alloy of iron, copper and carbon. The electric arc thus produced emits chiefly violet and ultra-violet rays and very few heat rays, the latter being absorbed by the metal. This does away with the necessity for the current of cold water of Finsen's apparatus. The arc works with an intensity of 15 amperes, corresponding to 70 volts. The distance of the arc from the lesion to be treated is from 4 to 8 inches.

The apparatus is arranged as follows: the electrodes are vertical, the positive being below. A cylindrical iron chimney, pierced by four holes, isolates the electrodes from the region to be treated, which is placed opposite one of the holes. Under the fourth hole is a rod carrying a movable lens, which shows whether the apparatus is working properly by projecting an image of the arc on a white screen. Each orifice which serves for treatment is furnished with a lens terminated by a plate of quartz which serves as a compressor, and against which the region to be treated is applied. The duration of each sitting is from three-quarters of an hour to an hour. The only disadvantage of this apparatus is the necessity for a chimney, on account of the oxide of iron produced.

The curative results of this apparatus are excellent, but the treatment is usually of long duration.

Radiotherapy.—The technique of radiotherapy has been already described (p. 277). For the treatment of lupus, rays corresponding to Nos. 4 and 5 of the radiochromometer are employed. Below No. 4, the tubes emit rays which are not penetrating enough; above No. 5, the rays are too penetrating.

The region to be treated should be from 6 to 8 inches from the tube. The surrounding parts may be protected by sheets of lead, but better, by enclosing the tube in a lead box with a hole opposite the anticathode, to avoid radiodermatitis. The quantity of units H to be absorbed and the frequency of the sittings vary according to the extent, depth and age of the lesions, and their degree of reaction to the rays. It is well to commence with doses of 3 or 4 H at

intervals of a week or a fortnight; after having tested the reaction of the lesion, the duration and frequency of the sittings can be gradually increased. Great care must be taken during the sittings to regulate the emission of the tube, that is, to make it produce rays of the same quality, by using the osmo-regulator if necessary.

Radiumtherapy.—In 1896 Becquerel showed that *uranium* and its salts, without any exciting cause, constantly emit radiations, presenting great analogies with the X-rays, and like the latter, possessing the property of penetrating opaque bodies and affecting photographic plates. Madame Curie discovered that thorium possessed the same properties, and called these bodies radio-active substances. Later on, Madame Curie found that pitchblende, a mineral containing oxide of uranium, had a radio-activity four times as great as uranium, and therefore concluded that pitchblende must contain other bodies more active than uranium and thorium. From pitchblende she isolated *polonium*, a metal belonging to the bismuth group, and *radium*, belonging to the barium group, and found that radium has a radio-activity two million times as great as uranium. More recently, Debierne has separated a similar body, called *actinium*.

Radium produces spontaneously, continually, and without wearing away, similar effects to those of a Crooke's tube traversed by an electric current of high tension. Radium is luminous in the dark, and its rays render a number of bodies fluorescent. The rays have a similar effect on the tissues as that produced by the X-rays; they may give rise to the same cutaneous lesions, and have a similar action on the healthy and diseased skin.

This analogy of radium rays with the X-rays led Danlos to try radium in the treatment of lupus; but radium does not appear to act as rapidly as the X-rays, and hence requires a longer application. Danlos employed sachets of india-rubber or celluloid, containing a mixture of chloride of radium and chloride of barium in various proportions, the radio-active power being proportional to the amount of radium in the mixture. Taking the radio-active power of uranium as the unit, Danlos used mixtures with radio-active powers of 5200 and 19,000. The sachets were placed directly on the lesion, for one sitting of twenty-four to forty-eight hours. Speaking generally, the effects produced by the radium rays are proportional to the intensity of the sachet and the duration of the sitting.

According to Danlos, after an application of sachets of 19,000 or 5200 intensity, for several hours, redness of the skin is first produced; then, after six to twenty hours, maceration of the epidermis, sometimes preceded by a blister; then superficial ulceration, which heals in six weeks. The cicatrix is white and æsthetic.

Besides this prolonged method, which produces ulceration, there

is another method by short and repeated applications, which does not produce ulceration. This result is obtained with apparatus of high intensity, applied for short periods and frequently repeated. For this purpose, an apparatus is used consisting of a square plate fixed on a handle, the lower surface of the plate being coated with radium. The radiation from this apparatus is four or five times as great as that of the sachets. The radium is spread on the plate by means of a thin layer of a varnish which resists the prolonged action of boiling water and antiseptics; this is essential, in order that the apparatus may be disinfected after each application.

Wickham and Degrais have also tried radium in the treatment of lupus. They use energetic and destructive doses in all cases of lupus, whether vegetating or not. The apparatus they employ has a radiation of 580,000 (10 per 100 alpha, 87 per 100 beta, and 3 per 100 gamma rays) and is applied for three or four hours, without the intervention of screens. In ten or fifteen days a sphacelus is formed, which in the course of one or two months leaves a thin white scar, neither depressed nor bridled.

Recurrent lupus nodules can be detected by wiping the cicatrix with a little alcohol.

Isolated lupus nodules can be treated by the same method when they are close enough together to form a patch. When the nodules are far apart, Wickham and Degrais treat them by excision or by cauterisation with the galvano-cautery, followed by radium.

Of these three methods of treatment for lupus, *phototherapy* gives excellent results, but takes a long time; *radiotherapy* does not give better results than other methods of treatment; *radiumtherapy* has not been employed long enough for its value to be estimated in the case of lupus.

Lupus Erythematosus.

The name *lupus erythematosus* was given to this dermatosis by Cazenave, who connected it with tuberculous lupus. It manifests itself in the form of an erythema of centrifugal evolution, slightly squamous, with a tendency to central cicatricial atrophy. Many dermatologists regard it as an affection distinct from cutaneous tuberculosis. Malcolm Morris has proposed the name of *erythema atrophicans;* others, including Besnier, maintain the tuberculous nature of the affection, and I am also of this opinion with regard to the majority of cases, but not for all, as I shall shortly explain.

According to Besnier, lupus erythematosus is chiefly observed in tuberculous families, and patients with this dermatosis often become affected, sooner or later, with pulmonary, glandular or articular tuberculosis. Again, it is not uncommon for lupus erythematosus to become transformed into tuberculous lupus; there are inter-

mediate cases which have been called *erythemato-tuberculous lupus.* The adversaries of this view raise the objection that tuberculous follicles, giant cells, and tubercle bacilli are absent in lupus erythematosus, and that experimental inoculation is always negative. But these arguments were for a long time used against the tuberculous nature of lupus vulgaris, till new experiments gave positive results from inoculation. Moreover, it is possible for lupus erythematosus to be tuberculous in nature without being bacillary; the toxin formed by the bacillus may play the chief part in the production of this lesion. The influence of the tuberculous toxin is especially evident in the cases of generalised lupus erythematosus described by Kaposi, Besnier and Hallopeau, which may be allied to certain erythemas observed in the course of acute or chronic pulmonary tuberculosis.

But the etiology of lupus erythematosus is not always the same. Brocq draws attention to the existence of two clinical types of lupus erythematosus which seem to me to correspond to two different etiologies, according to the researches of Paris and Dobrovici in my clinic. These two types are: *fixed lupus erythematosus* and *migratory lupus erythematosus.*

In *fixed lupus erythematosus,* Paris and Dobrovici found that the agglutination test for tubercle bacilli in homogeneous culture was positive, but negative in the *migratory type.* It would, therefore, appear that the fixed forms of lupus erythematosus are of tuberculous origin, and the migratory forms of some other origin, as yet undetermined.

Like lupus vulgaris, lupus erythematosus is often observed in lymphatic subjects. It is an affection of adult age, and is more common in women; it is rare in children. Besnier states that it is more common in country people, who live in the open air.

Symptomatology.—Lupus erythematosus may be *local* or *general;* the local form is subdivided into *simple* and *acneic.*

Simple Lupus Erythematosus.—This begins in the form of small red spots, generally on the face, which extend gradually and become confluent, forming several isolated round patches, or a single patch. The colour of the patches is red, sometimes bluish red. The redness diminishes on pressure, and is increased by congestion of the face, after meals or alcohol for instance. It is sometimes associated with vascular dilatations, which give the affection a certain resemblance to varicose acne rosacea (telangiectasic lupus). The patches vary in their elevation above the skin; sometimes they consist in a superficial thickening of the skin; sometimes in a deep induration, but never so pronounced as in lupus vulgaris. The squames on the surface are fine, dry and very adherent; this adhesion is due to prolongations into the glandular orifices. In some cases the squames are abundant (squamous lupus erythematosus). The borders of the

patch are well defined, red and slightly raised. The patch may extend by its whole periphery, or only at one part. The central part of the patch is depressed, and undergoes cicatricial fibrous atrophy. The cicatrix is smooth and white, and is produced spontaneously by a kind of interstitial absorption, without ulceration; in some cases there are isolated cicatricial islets. In rare cases the central cicatrix may be absent during the whole course of the disease.

Lupus erythematosus is generally more painful than lupus vulgaris; it is sensitive to pressure, and may also give rise to burning, pricking and shooting sensations.

FIG. 145.—Lupus erythematosus. (St Louis Hospital Museum.)

Its most common situation is the face. Sometimes it takes the form of symmetrical centrifugal erythema, occupying the centre of the face, the bridge of the nose, and the adjacent parts of each cheek, resembling a bat with extended wings; hence the name *vespertilio*, which was formerly given to it. Sometimes it is asymmetrical, and constituted by one or more patches situated on the nose, cheeks and ears, etc.

Lupus erythematosus may occur on the scalp, in the form of raised, red patches with a smooth surface or covered with squames. After a time, the patches undergo central cicatricial atrophy, which causes destruction of the hair bulbs and loss of hair.

Lupus erythematosus may also occur on the buccal mucous membrane, either independently or by extension from the lips. It is more rare on the limbs than on the face, but may affect the extremities symmetrically.

A special type, *lupus pernio*, occurs on the face and fingers, especially in young lymphatic subjects. This form resembles chil-

blain, and affects the same regions—the fingers, back of the hands, ears, nose and cheeks. The skin is swollen, painful, and of a purple colour. On the ear, lupus pernio often causes small sloughs, leaving a granulating surface; on the fingers, it leads to ulceration, some-

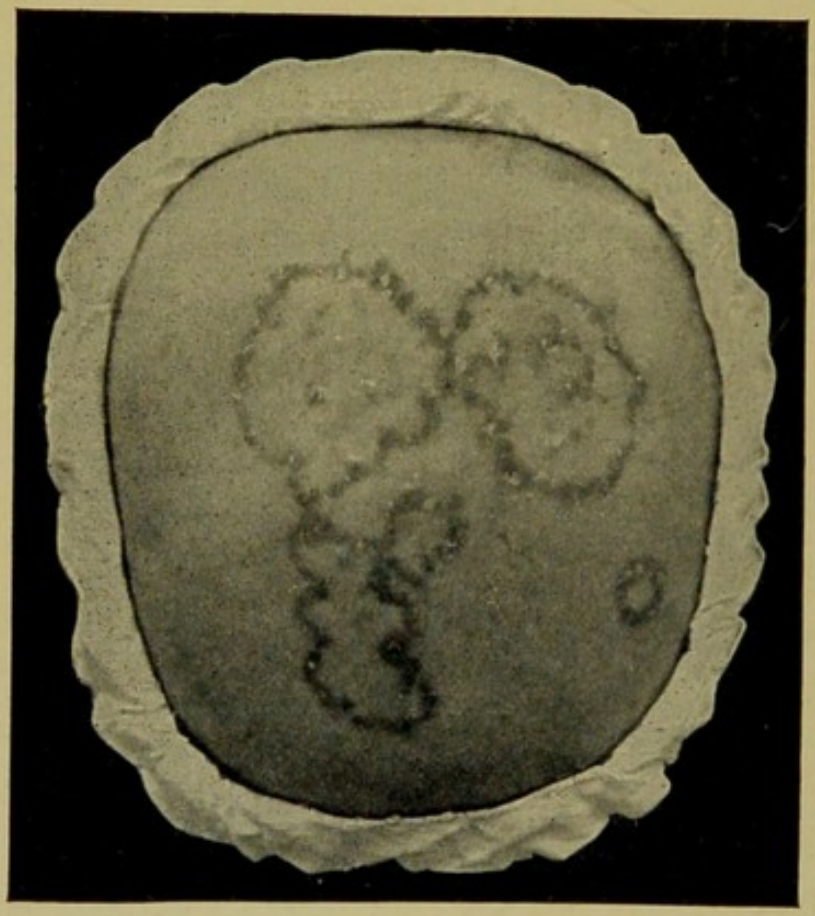


Fig. 146.—Lupus erythematosus of the scalp.
(St Louis Hospital Museum.)

times accompanied by tuberculous synovitis; or it may be covered with chalky squames, as in acneic lupus.

Acneic Lupus. — This form owes its special appearance to the predominant part played by the glands in the pathological process. It is constituted by round, gray, slightly raised patches, with a granular surface, and surrounded by a bluish areola. Each patch is covered by a rough, dry squame, of chalky appearance, very adherent, and prolonged into the orifices of the sebaceous glands. If the squame is detached, the prolongations from its under-surface and the dilated glandular orifices can be seen. The squames are formed by a mixture of desquamated epidermis and sebaceous secretion; they cover nearly the whole extent of the patch, so that its red or purple colour is only visible at the borders. After a time, the squame is spontaneously detached, and is not renewed; the patch becomes

gradually depressed from the centre towards the circumference and is replaced by an atrophic cicatrix, without ulceration.

Acneic lupus occurs on the face, fingers and dorsal surface of the hands, more rarely on the trunk.

The squames are not always dry; sometimes, on the nose and ear, they are soft and fatty like the crusts of seborrhœa (seborrhœic lupus). This is due to the predominance of sebaceous matter over epidermic scales in their formation. Moreover, chalky patches, seborrhœic patches, and even ordinary squamous patches, may coexist.

The *evolution* of local lupus erythematosus is very variable, but presents two principal types: *migratory* and *fixed*. In the *migratory*

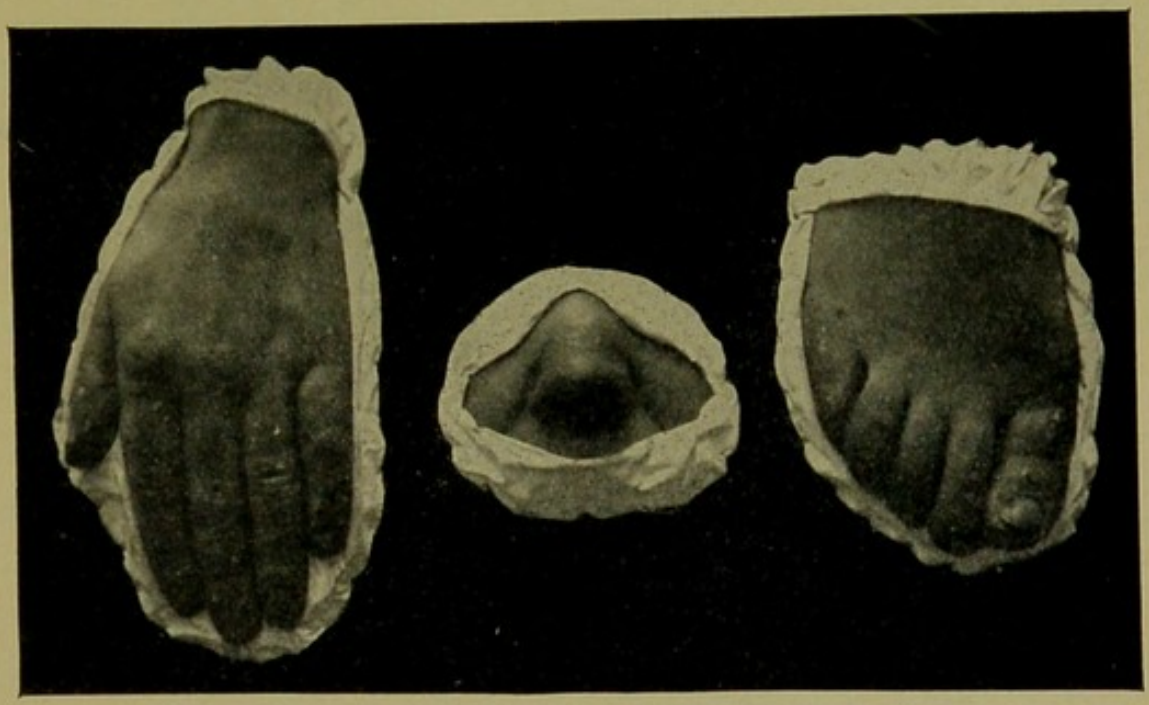


Fig. 147.—Lupus pernio. (St Louis Hospital Museum.)

type, the eruption extends rapidly; the patch, commencing on the nose, soon spreads to the cheeks (centrifugal erythema). In other cases the patches remain isolated and disseminated on the face; they appear rapidly, retrogress and then recur. The lesion may undergo spontaneous cure after a few years, after alternations of retrogression and recurrence, especially occurring in the spring and autumn. This form of lupus generally leaves indelible atrophic cicatrices, but it sometimes disappears without leaving any trace.

In the second or *fixed type*, the patches are more localised and less numerous, but extend more deeply. They persist longer, and end in the formation of a cicatrix. This fixed type includes acneic lupus.

Generalised Form.—According to cases reported by Hebra,

Kaposi, Besnier and Hallopeau, generalised or disseminated lupus erythematosus may be acute or chronic.

The *acute form* is of rapid evolution and grave prognosis, presenting all the signs of an infective exanthematous fever. The eruption occurs not only on the face and scalp, but also on the trunk and limbs. The patches are slightly raised, of a dark red colour,

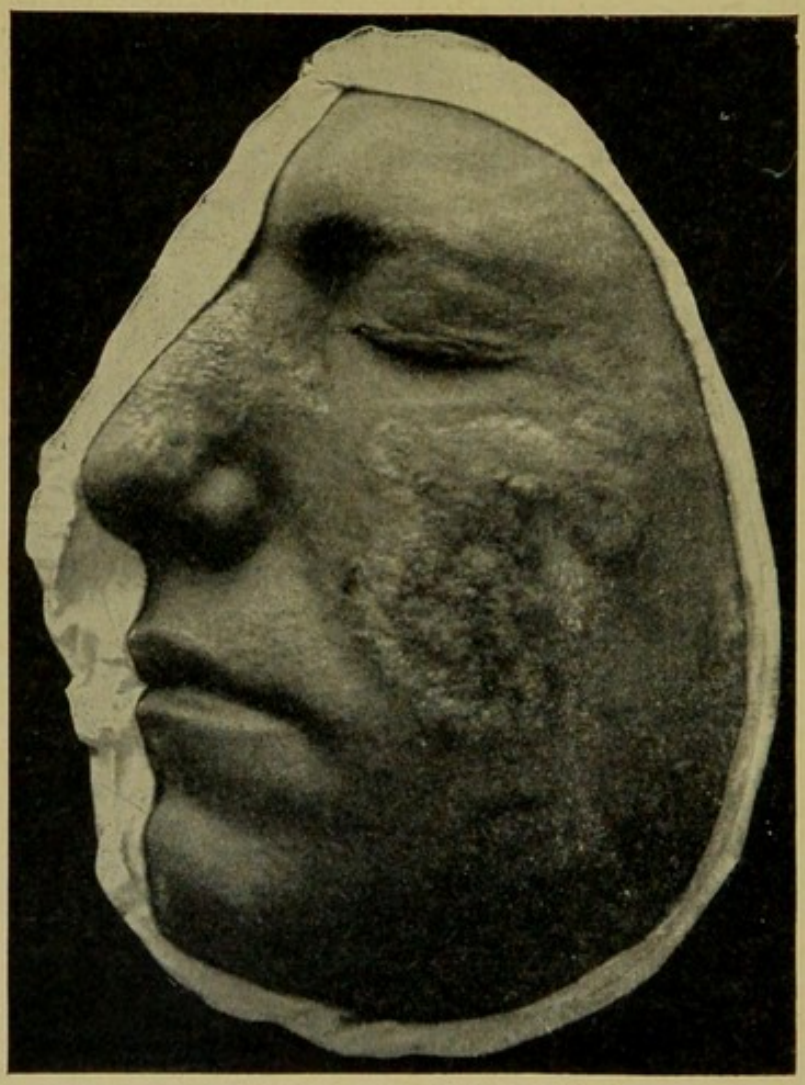


FIG. 148.—Acneic lupus. (St Louis Hospital Museum.)

sometimes livid. They develop eccentrically. The eruption is accompanied by high fever (40° C). It is complicated by arthralgia, hydrarthrosis, ostealgia, pulmonary congestion, endocarditis, albuminuria, and even acute tuberculosis; one of the latter complications may cause death. But this acute form is not always fatal; sometimes the eruption retrogresses in places, but appears in other places and passes into the chronic stage.

Acute or subacute forms may present a special aspect, resembling bullous polymorphous erythema or hydroa. This variety occurs on

the face and extremities. The patches are annular, with a pale centre and two concentric peripheral rings, an internal pink ring, and an outer bright red, by which the lesion extends peripherally.

The *chronic form* may succeed the acute form, or may occur independently, without fever or disturbance of the general health. The patches develop gradually, and end by invading nearly the whole surface. The lesions may undergo resolution after a time without leaving any trace; sometimes they undergo cicatricial atrophy.

DIAGNOSIS.—Lupus erythematosus, especially the telangiectasic form, may be mistaken for *acne rosacea*, when acneic pustules are absent; but the lupoid patch has well-defined raised borders, is covered with squames, and presents cicatrices, which do not occur in acne rosacea. *Trichophytic circinate herpes* has an eccentric evolution, but is distinguished by the following characters: the lesion is very superficial, the centre heals without undergoing cicatricial atrophy, vesicles are present at the periphery of the patch; lastly, microscopical examination reveals the fungus. *Tertiary syphilides* of the face have a special copper colour and larger squames; some cases are decided by the effect of treatment. Fournier has published a case cured by antisyphilitic treatment, which had been diagnosed as lupus erythematosus. Inversely, I have seen a case of lupus erythematosus affecting the face and scalp, which had been diagnosed by several leading dermatologists as a syphilitic affection, and treated as such for several years without any effect. *Psoriasis* of the face is sometimes difficult to distinguish from acneic lupus, in spite of its squames being larger, less adherent, and often situated on papules; but there are no central cicatrices in the patches of psoriasis, and characteristic lesions are often present on the knees and elbows. *Chilblains*, which affect the same regions as lupus pernio, differ from the latter in the absence of sphacelated spots and cicatrices; although chilblains may ulcerate, they never present spontaneous cicatrices, which are not preceded by ulceration; moreover, the lupoid patches persist during the summer. In *seborrhœic eczema*, there is less cutaneous infiltration underneath the patches than in lupus erythematosus, and the patches are covered with soft, fatty, non-adherent squames; lastly, there is no cicatricial atrophy. Lupus erythematosus of the scalp is easily distinguished from *alopecia areata*, which is white and smooth and not infiltrated; the borders of the lupoid patch are red and raised, with a central cicatrix.

The distinction between lupus erythematosus and *lupus vulgaris* is sometimes difficult; the former is an erythema, while the latter is formed by the aggregation of tuberculous nodules; there are nearly always some isolated tubercles around the principal patch.

Disseminated lupus erythematosus must not be confused with

scarlatiniform erythema, an affection of rapid evolution, consisting in a diffuse red eruption with abundant squames and no infiltration of the dermis; nor with *polymorphous erythema*, which never presents cicatricial atrophy, and is of shorter duration. Lastly, the premycosic erythematous patches of *mycosis fungoides* are distinguished from disseminated lupus erythematosus by the intensity of the pruritus and the presence of glandular enlargement (Besnier, Hallopeau.)

PATHOLOGICAL ANATOMY.—The essential lesion of lupus erythematosus is a diffuse small-celled infiltration of the dermis, varying in depth, but especially marked along the vessels and around the glands (Schutz, Vidal and Leloir). The vessels are dilated and in some places ruptured, giving rise to local or diffuse hæmorrhages. Tuberculous nodules, giant cells and tubercle bacilli are absent, Some of the embryonic cells undergo colloid or fatty degeneration, but there are never caseous masses.

In *acneic lupus* the sebaceous glands are infiltrated with embryonic cells and their ducts filled with epidermic cells, which form the prolongations of the squames mentioned above.

The nutrition of the connective tissue is gradually interfered with, leading to degeneration, atrophy and partial absorption of fibrous and elastic tissue; the vessels become invaded by the embryonic neoplasm, and partly obliterated. In the epidermis, the Malpighian layer becomes atrophied, the stratum granulosum and stratum lucidum disappear, so that the stratum corneum represents nearly all that is left.

TREATMENT.—General treatment is the same as for lupus vulgaris, and includes cod-liver oil, iodine preparations, phosphates, arsenic, etc.

Local treatment.—When there is acute inflammation, applications of boiled water, starch poultices, and zinc ointment are indicated. When there is no acute inflammation, the following method may be used: soft soap dissolved in alcohol and spread on flannel is first applied to the part for several hours; after this is washed off, boric acid or zinc ointment is rubbed in. This treatment is repeated for several days, but if it causes much irritation, it must be suspended for a time. Strong iodine applications, made several times, with intervals of a few days, are useful:—

Iodine	1 part
Iodide of potassium	1 „
Distilled water	2 parts

The following ointment often gives good results:—

Iodide of potassium Biniodide of mercury	aa 1 part (or more)
Lard	200 parts

Equal parts of glacial acetic acid and tincture of iodine are also good; in this mixture, the acetic acid acts as a kind of mordant and favours penetration of the iodine, which is undoubtedly the best application for lupus erythematosus. Lactic acid, pure or diluted, and carbolic acid have also been used, but cause rather acute inflammation.

Scarification is very effective and harmless in lupus erythematosus, in which there are no inoculable bacilli; it is especially useful in the fixed and infiltrated forms.

The galvano-cautery should only be employed in localised lupus erythematosus with deep infiltration, in the same way as in lupus vulgaris.

Phototherapy and radiotherapy have also been tried in lupus erythematosus, but without much result.

High frequency currents appear to be very efficacious in lupus erythematosus of the scalp, but inferior to caustics in the case of the face, when employed alone.

But when high frequency treatment is preceded by scarification, the *combination of these two methods* is certainly superior to every other therapeutic method in localised, fixed lupus erythematosus, whatever its situation, especially in infiltrated and squamous forms.

The principle of this method consists in making the electric discharge from a condensing electrode penetrate as far as the healthy tissues by means of deep scarifications. The high frequency discharge from a condensing electrode, previously rendered aseptic and excited by a resonator, is applied immediately after scarification. The latter should penetrate the healthy tissue. Coagulation of the blood, which forms an obstacle to the penetration of the electric discharge, is prevented by applying a compress soaked in a solution of citrate of soda (6 per 100). A discharge of medium intensity is passed over the whole extent of the lesions and some distance beyond, the glass sheath being continually applied to the tissues. The duration of the application is two or three minutes for a patch of 8 square inches. This method seems to me the best and most rapid form of treatment for lupus erythematosus.

Sclerous Lupus, or Papillomatous Tuberculosis.

Sclerous lupus, better called papillomatous tuberculosis, is constituted by papillomatous excrescences, grouped in the form of more or less raised patches of variable extent, covered by horny tissue, and separated from each other by fissures.

This affection was called *verrucose scrofulide* by Hardy, and has been studied by Riehl and Paltauf under the name of *verrucose*

tuberculosis. It includes the *anatomical tubercle,* the tuberculous nature of which was demonstrated by Verneuil, Vidal and Besnier.

Etiology.—This lesion occurs on the hands and fingers in medical men and hospital attendants, who have wounded themselves with instruments used for autopsies on tuberculous patients. It may occur after abrasion by an object soiled by tuberculous matter. It may also affect persons who have to deal with tuberculous animals, or who handle tuberculous meat, such as veterinary surgeons, butchers, knackers, coachmen, cowherds, etc. It may also be produced by auto-inoculation in a wound in a tuberculous subject. Lastly, it may develop by auto-inoculation around a fistula connected with tuberculous bone, around a glandular abscess, or an anal fistula. However, in spite of the capital part played by inoculation in its development, individual receptivity has a great influence on the gravity and persistence of the lesion; tuberculous heredity, alcoholism, and organic affections of any kind, are the principal predisposing causes.

Symptomatology.—The lesion begins either as an ulcer, resulting from the unhealed wound of inoculation, which is sometimes covered with a crust and soon becomes papillomatous, or as a small, hard, red nodule, the centre of which becomes crustate, and which is surrounded by similar nodules. The aggregation of these nodules forms a round or irregular patch, varying in size from a sixpence to a five-shilling piece. Its appearance is that of a papilloma with an irregular, raised surface, covered with gray, horny vegetations, separated by fissures and linear ulcerations, from which pus can sometimes be exuded by pressing the base of the papillomatous patch.

Three zones have been described in the patch: a central zone, more projecting than the periphery, representing the oldest part of the lesion; a middle zone, formed by smaller elevations mixed with pustules; an external erythematous zone. The warty elevations are covered with thick, adherent scales of horny epidermis. If these are softened by means of poultices and then removed, the subjacent papillomatous surface is seen to be covered with red elevations separated by fissures exuding pus. The horny covering is sometimes replaced by hard, thick, adherent crusts.

Evolution.—This is always chronic. The patch extends slowly and intermittently, either at its whole periphery or at one part. But the affection, in spite of its continual growth, has a certain tendency to spontaneous cicatrisation. Sometimes the centre of the patch heals, leaving a thin, glistening, white or pink cicatrix, while the periphery continues to extend. Sometimes the whole patch cicatrises, but recurrence may take place at some part.

PROGNOSIS.—Verrucose tubercle is generally a local lesion and does not become generalised; but in predisposed or tuberculous subjects the local tuberculosis may spread to the lymphatic vessels and glands, and even invade the viscera.

DIAGNOSIS.—A patch of papillomatous tuberculosis must not be mistaken for a simple *wart;* the latter has no indurated base nor erythematous areola, and is not covered with crusts. *Simple papilloma* has a strong resemblance to verrucose tubercle, but has no indurated base, no peripheral erythema, and no central cicatrix; however, the diagnosis is sometimes so difficult that bacteriological

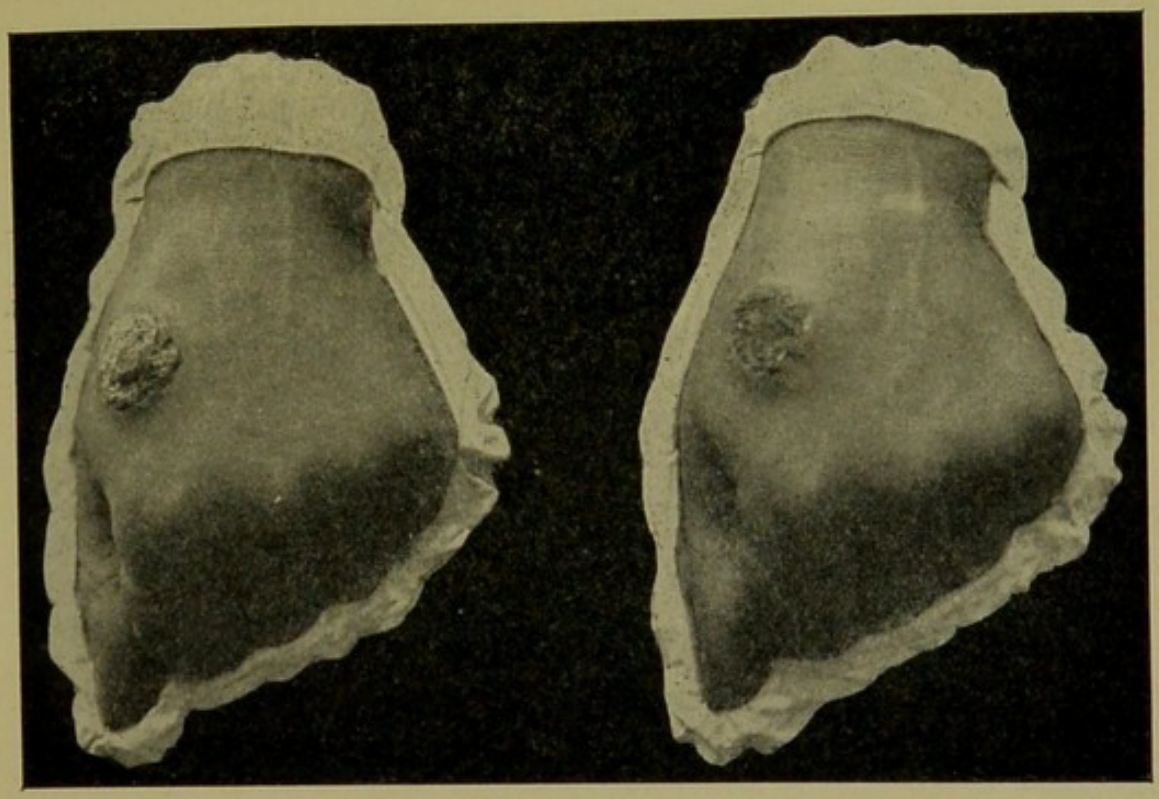


FIG. 149.—Verrucose tubercle. (St Louis Hospital Museum.)

examination and experimental inoculation are necessary. Verrucose tubercle is easily distinguished from *vegetations,* which are soft, moist, digitated excrescences, without crusts.

Trichophytic agminated folliculitis and perifolliculitis occurs on the back of the hand or forearm; it is not fissured, but studded with holes, from which white plugs of thickened pus can be expressed. *Lichen planus cornea* is formed by hard, dry papules, which can always be recognised even when they are grouped in a patch. *Papillary epithelioma* is fairly easy to distinguish by its bleeding vegetations, covered with slightly adherent crusts. *Verrucose* or *papillomatous nævus* is a congenital lesion, or appears soon after birth; suppuration and crusts are absent, and there is only a seborrhœic exudation.

Pathological Anatomy.—The lesions are situated chiefly in the superficial layer of the dermis and in the papillæ, and consist in diffuse embryonic cell infiltration, and, in places, tuberculous follicles. The papillæ are elongated and thickened, the papillary vessels dilated or sclerosed. As the lesion extends, the tuberculous follicles become transformed into fibrous tissue, and the embryonic tissue also tends to fibrous organisation. This sclerous and atrophic evolution leads after a time to spontaneous cicatrisation of the lesions. The superficial follicles suppurate and form small abscesses which open into the interpapillary sulci. The horny and granular layers of the epidermis are hypertrophied, and its interpapillary processes are elongated and thickened.

This lesion is not very virulent and contains few bacilli; it is necessary to make many preparations in order to find them, and the examination is sometimes negative; but experimental inoculation in the guinea-pig is usually positive.

Treatment.—The lesion should be destroyed either by cauterisation or by scraping; liquid caustics are not sufficient. Cauterisation may be performed by the thermo-cautery or galvano-cautery; it should be deep, and repeated at several sittings. It has the advantage of avoiding tuberculous infection of the blood.

Scraping should be performed under local anæsthesia, after applying an Esmarch's tourniquet. It is well to cauterise the wound with the thermo-cautery after scraping. The wound should first be dressed with simple moist dressings, and afterwards with iodoform or boracic ointment.

Radiotherapy sometimes gives better results than other forms of treatment. Broca cured an anatomical tubercle in my clinic, after a few sittings, which had resisted chemical caustics and the thermo-cautery for several months.

Tuberculous Lymphangiectasis with Perilymphangitis.

This variety of lymphangiectasis often originates from a tuberculous lesion of bone or from an anatomical tubercle.

The lesion begins with diffuse swelling around the primary osseous or cutaneous lesion. Bluish red elevations then appear, some isolated, others aggregated, forming swellings like those of large varices. Similar swellings develop, one above the other, along the course of the lymphatics. The surrounding skin is dark red or purple, swollen and indurated; the lymphatic vessels are indurated, and the glands enlarged. These swellings, formed by dilatation and chronic inflammation of the lymphatics and induration of the surrounding tissues, eventually rupture, and discharge a liquid resembling pure lymph, or lymph mixed with pus, and become

fistulous. The pus contains tubercle bacilli and pyogenic microbes; inoculation of guinea-pig with the liquid is positive (Hallopeau, Jeanselme).

TREATMENT.—Hallopeau obtained good results by injecting iodoform and oil of vaseline into the fistulous openings.

Papular and Nodular Cutaneous Tuberculosis.

We have now to study a form of cutaneous tuberculosis in which the lesions are due, not to the tubercle bacillus itself, but probably to its *toxins*. This form includes many morphological varieties, the nature and origin of which have long been misunderstood and are still disputed, although present opinion tends to regard them as tuberculous.

These toxinic forms of cutaneous tuberculosis belong to the group arising from internal causes; but, contrary to tuberculous gummas, in the walls of which a few tubercle bacilli are sometimes found, tubercle bacilli are usually absent and animal inoculation usually negative.[1] Sometimes histological examination has shown the existence of tuberculous follicles, but, as a rule, there is only a formation of embryonic tissue around the vessels, with a tendency to necrobiosis; a histological feature which is not conclusive.

So far, the tuberculous origin of these lesions rests on clinical evidence. In the first place, they only occur in tuberculous subjects; secondly, the history and present state of these subjects reveals no other cause than tuberculosis for their production.

Toxinic cutaneous tuberculosis manifests itself in two forms: one papular, the other nodular, so that we can distinguish a *papular cutaneous tuberculosis* and a *nodular cutaneous tuberculosis*.

Papular cutaneous tuberculosis includes lesions which are sometimes follicular and sometimes independent of the sebaceous follicles. They are: (1) acnitis; (2) folliclis; (3) lichenoid or follicular papular cutaneous tuberculosis, which represents the *lichen circumscriptus* of Rayer and the *lichen scrofulosorum* of Hebra; (4) acneiform papular cutaneous tuberculosis (cachectic acne).

Nodular cutaneous tuberculosis includes nodular lesions of variable size, which sometimes end in suppuration or ulceration, and sometimes remain stationary indefinitely. There are three forms: (1) ecthymatous tuberculosis; (2) erythema induratum (Bazin); sarcoid (Boeck).

These forms of cutaneous tuberculosis, whatever their form and variety, have the following characters in common: they are composed of elements which are sometimes isolated, sometimes aggregated in patches of greater or less extent; the eruptive element

[1] See pp. 354, 355.

is a papule or a nodule situated in the dermis or subcutaneous tissue, of a dark red or copper colour, except on the lower limbs, where it is purple, on account of venous obstruction.

As a rule, the lesions become pustular or suppurate, except some cases of lichenoid tuberculosis and erythema induratum, which rarely suppurate, and sarcoid, which never suppurates.

When pustules develop, which is most frequently the case, they either dry up and form crusts, or rupture and discharge pus, which forms crusts. In both cases the scab or crust is adherent, and covers a deep conical ulcer. The latter is replaced by a bluish cicatrix, which becomes pigmented like a syphilitic lesion. The pigmentation slowly disappears, and finally a depressed cicatrix is left, like that of variola.

These dermatoses may occur on any part of the body except the genital organs and mucous membranes. They are painless, and are never accompanied by fever, unless complicated.

Papular Cutaneous Tuberculosis.

1. Acnitis.—This is a rare dermatosis, constituted by small papules, situated in the deeper part of the dermis or in the subcutaneous tissue. It has no connection with the pilo-sebaceous follicles, so that the name acnitis, given to it by Barthélemy, is incorrect.

The papules are rounded, and vary from a millet seed to a hemp seed in size. They first appear on the face, and for a long time remain localised to this region, sometimes exclusively; they may afterwards appear on the trunk, but rarely on the limbs. They are disseminated, and never form patches. The papule is hard at first, but later on softens and forms a pustule, similar to an acne pustule; this discharges pus and becomes covered with a crust; this eventually falls off, leaving a cicatrix with a pigmented areola.

Acnitis develops in crops and lasts several months; it is only met with in subjects presenting tuberculous lesions.

Diagnosis.—It may be mistaken for *acne vulgaris* and *cicatricial acne pilaris;* but acnitis is at first subcutaneous and only affects the skin secondarily; moreover, it is never painful.

2. Folliclis.—The name *folliclis,* given by Barthélemy to this other form of cutaneous tuberculosis, is equally incorrect, because the lesion is not situated in the sebaceous follicles. The term suppurative *hydrosadenitis,* formerly employed by Dubreuilh, is no better, as the lesion is not connected with the sweat glands.

Folliclis resembles acnitis, but the elements are more superficial and situated in the dermis. It occurs on the limbs, especially at the extremities and on the extensor surfaces, never on the face. It is often symmetrical, and may occur on the palms or soles.

The papules may be isolated, but, contrary to those of acnitis, are generally grouped in patches. They are the size of a lentil. The papule becomes a pustule, which is followed by a crust, under which is a cup-shaped ulcer, which terminates in a pigmented cicatrix.

In short, acnitis and folliclis only differ in their situation. They are both non-follicular papular forms of cutaneous tuberculosis. Both develop in crops, so that lesions in all stages of evolution are seen in the same subject.

DIAGNOSIS.—Folliclis is easily distinguished from *acne* by its situation in the dermis, its occurrence on the limbs, its long duration and appearance in successive crops, and by its persistent cicatrices. The latter also distinguish it from the *acneiform syphilide*, which always disappears without leaving any traces.

3. Lichenoid Cutaneous Tuberculosis.—This dermatosis is a *tuberculous folliculitis* of the pilo-sebaceous follicles.

There are two varieties: (1) a dry papular form, which represents the *lichen circumscriptus* of Rayer; (2) a pustular form, which corresponds to the *lichen scrofulosorum* of Hebra.

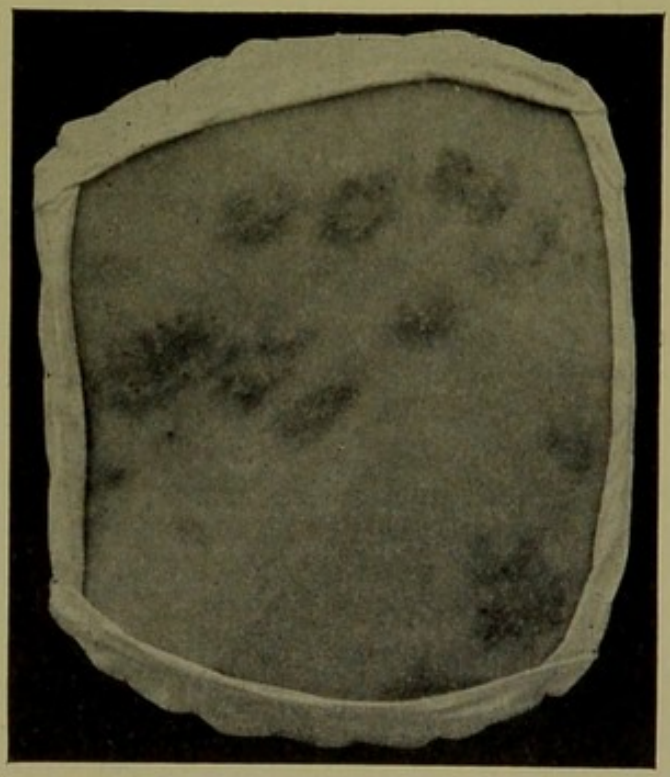


FIG. 150.—Lichenoid cutaneous tuberculosis. (St Louis Hospital Museum.)

The *dry papular form* resembles lichen. The papule is miliary, yellow or red in colour, and acuminate instead of flat, as in the non-follicular forms. It is situated at the orifice of a hair follicle, and is covered with a thin squame.

The papules are sometimes isolated, but more usually grouped in irregular round patches; but each papule preserves its independence. The eruption occurs on the back, abdomen, limbs and nape of the neck.

DIAGNOSIS.—This dermatosis resembles in many points *lichenoid eczema*, but differs from it in the absence of itching, exudation and vesicles; the patches are not so well defined as in lichenoid eczema, and there are isolated papules around them.

The *evolution* is very slow; the patches extend eccentrically, while the centre is replaced by a pigmented spot.

In the *papulo-pustular form* (*lichen scrofulosorum*), the papules may be isolated or confluent, and are larger than in the preceding form. They suppurate and form large pustules with an indurated red base. The pustules give place to ulcers, which may be extensive when the elements are confluent; these ulcers have raised borders, and leave a pigmented cicatrix. The eruption occurs chiefly on the lower limbs, where the papulo-pustules are surrounded by a purple areola.

In severe cases such as those described by Hebra, an eczematiform eruption with fœtid exudation is present on the scrotum and pubic region.

This dermatosis occurs in subjects affected with local tuberculous affections of the bones and glands, or tuberculous gummas. Its evolution is slow and intermittent, and the lesions are liable to recur.

Jacobi found the tubercle bacillus in the lesions, and showed that the papules present the same structure as miliary tubercles. Hallopeau also found that the papules had the histological structure of tubercle, but could find no bacilli. More recently, Wolf has found bacilli. Subcutaneous injections of tuberculin have confirmed the tuberculous nature of this dermatosis. Neumann observed disappearance of the papules under this treatment, while Schweniger and Buzzi observed a similar eruption develop in a tuberculous subject after injections of tuberculin. Lastly, inoculations in animals have given positive results (Jacobi, Wolf, Pellizari, Haushalter).

DIAGNOSIS.—In some cases lichen scrofulosorum may be mistaken for *acute lichen*, but the latter is a benign, transitory affection, occurring in persons in good health. It may also be mistaken for a small *papular syphilide*, but in the latter the papules are hard, shiny, ham-coloured, arranged in circles, and covered with fine squames at their periphery. In doubtful cases the diagnosis can be settled by the history or by the effect of antisyphilitic treatment.

4. Acneiform Papular Tuberculosis.—This form is what Hebra called *cachectic acne*. It is a folliculitis, which resembles acne vulgaris, and occurs in cachectic subjects, especially in phthisical patients.

It is formed by small papulo-pustules of a dark red colour, sometimes purple, with a hæmorrhagic tendency, which occur on the trunk and limbs, especially the lower limbs. It is a torpid lesion, with no general or local reaction, and leaves cicatrices.

According to Hallopeau, this form of folliculitis is of tuberculous origin; but this has not yet been proved, either by its histological structure, or by the discovery of the tubercle bacillus in the lesions, or by animal inoculation.

Nodular Cutaneous Tuberculosis.

1. Ecthymatous Cutaneous Tuberculosis.—In this form, the lesions are situated in the deeper parts of the dermis; some resemble small gummas, others ecthymatous pustules with an indurated base. At first there is a dark red nodule, which is succeeded by a conical ulcer with red elevated borders. The lesion is torpid, cicatrisation is slow, and the cicatrix pigmented.

Of all forms of cutaneous tuberculosis, this is the least known, and is still subject to dispute. Its diagnosis depends on the presence of other tuberculous lesions, and on the exclusion of syphilis. The lesions are very similar to those of syphilis, especially congenital syphilis.

2. Erythema Induratum of Bazin.—This is a chronic indurated erythema, the tuberculous nature of which has only recently been

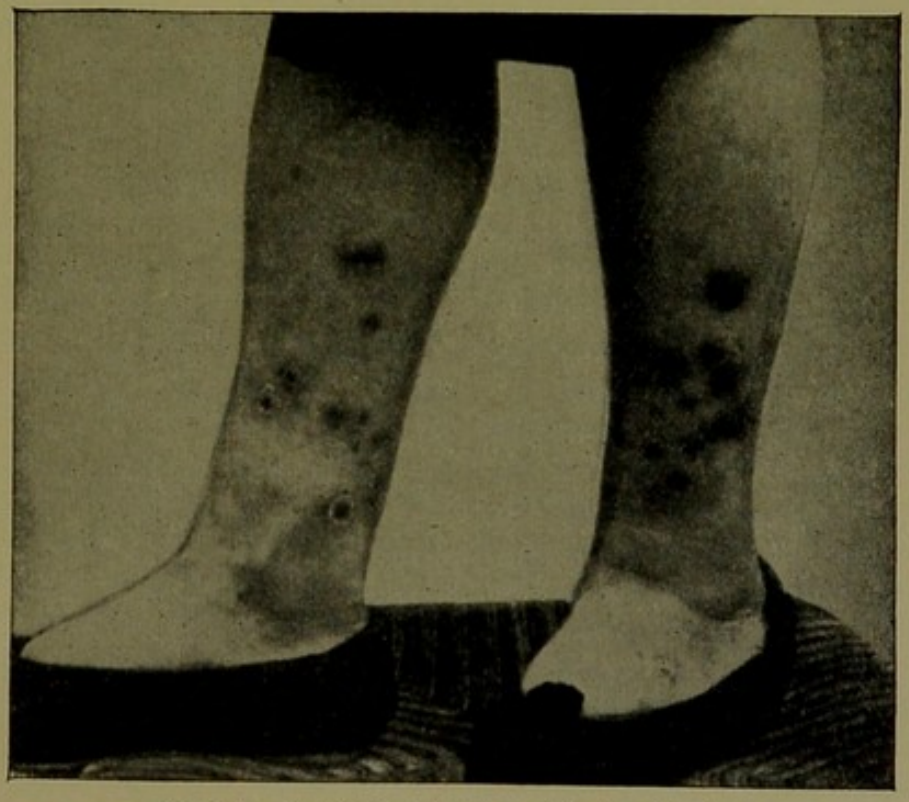


Fig. 151.—Erythema induratum of Bazin. (Audry.)

shown by positive animal inoculations made by Thibierge and Ravaut (1899). It was described clinically by Bazin.

This affection occurs chiefly in young, overworked lymphatic girls who come to town from the country, and consequently change their mode of life; for instance, washerwomen, domestic servants and others who work in a close atmosphere. Fatigue, want of air, and insufficient food are the predisposing causes. It is a chronic

affection, occurring usually on the lower and external parts of the lower limbs.

It presents itself in the form of nodosities or patches of deep infiltration in the dermis, of a bluish red colour, resembling erythema nodosum. There is often hard œdema round the patches, which lasts for months, and slowly disappears under the influence of rest and tonic treatment. Sometimes the lesions ulcerate, but the ulceration remains superficial, and terminates in a more or less pigmented cicatrix. The eruption develops in crops, especially in the winter. It may coexist with acnitis or lupus erythematosus.

DIAGNOSIS.—Erythema induratum is often mistaken for *erythema nodosum*, especially as it has the same situation; but the nodosities of erythema nodosum are very painful to pressure and present the changes of colour of ecchymoses, while the nodosities of erythema induratum are of a persistent bluish red colour, and are hardly at all painful. *Tuberculous gummas* are soft, not firm like erythema induratum, and undergo suppuration. The diagnosis from *syphilitic gummas* is sometimes difficult, especially if there are no other signs or history of syphilis; but ulcerated syphilitic gummas have a characteristic sloughy base. Lastly, erythema induratum may be mistaken for the tuberous forms of *bromide* and *iodide* eruptions.

3. Sarcoid of Boeck. — The cutaneous sarcoid, described by Boeck, is the only affection that I include in this group; for the name sarcoid has been given to different affections, and even to tuberculous lesions distinct from cutaneous sarcoid; for instance, subcutaneous sarcoids are small gummas, and the lympho-sarcoid of Gougerot is a form of disseminated miliary lupus.

Cutaneous sarcoid consists of small round tumours, pale red in colour, the size of a pea or nut, and somewhat resembling mycosis fungoides. The tumours are multiple, and may occur on the face, back, and extensor surface of the limbs. They remain solid and do not ulcerate. They may disappear and leave a slightly squamous, pigmented spot, which is sometimes telangiectasic.

Histological examination shows that the tumours are formed of tuberculous tissue.

TREATMENT. — The internal treatment of all forms of toxinic cutaneous tuberculosis is the same as for tuberculosis in general; cod-liver oil, iodo-tannic syrup, iodide of iron, phosphates and arsenic; nourishing diet, sea-air, sulphur baths, etc.

Local treatment varies a little, according to the variety. In acnitis and folliclis, and in the acneiform and ecthymatous forms, if the lesions are ulcerated, they should be dressed with boric lotion, or a solution of perchloride of mercury (1 in 5000) if they are not too extensive, and afterwards with iodoform.

In lichen scrofulosorum, more active preparations are required

such as salicylic acid, tartaric acid or resorcin ointments, or tincture of iodine. Hebra recommended the local application of cod-liver oil, but this is difficult to carry out in practice.

In erythema induratum, besides the general treatment indicated above, a change of occupation is advisable. The leg should be elevated and uniform pressure applied by means of a bandage: the lesions may be treated with tincture of iodine.

SPOROTRICHOSIS.

Sporotrichosis is an affection which has not long been known, but which, on account of the comparatively large number of cases already observed, has been the subject of many important articles. It is a mycosis, generally but not always cutaneous, due to a fungus of the order Hyphomycetes. The first cases were observed in America by Schenk (1898), Brayton (1899), Hektoen and Perkins (1900). The first case seen in France was described by Beurmann and Ramond (1903). In 1906 Beurmann and Gougerot published fresh cases. Since then, the same authors and others have reported other cases, so that the total number of cases published, with exact determination of the parasite, is already more than fifty. Recently, Lutz and Splendore have observed five cases of human sporotrichosis, and have shown that the disease may occur spontaneously in the rat.

This disease is, therefore, not at all uncommon, and it is at first sight surprising that an affection met with so frequently should not have been identified earlier. This is probably explained by the great similarity in the clinical and histological characters of the lesions to those of tuberculosis and syphilis, also to the special conditions necessary for culture of the *sporotrichum*.

Etiology.—The parasite (*sporotrichum*) develops on most of the ordinary media, but best of all on Sabouraud's media with maltose or glucose. The colonies appear on the sixth or ninth day. Each colony is at first white, punctiform, and very adherent to the agar; it soon extends peripherally, while the centre, which is folded and convoluted, becomes brown and then black. In liquid media, the parasite forms flakes which develop on the surface and fall to the bottom of the tube on shaking. The media never become turbid.

Examination of the culture in a hanging-drop shows a long granular septate mycelium, with irregular lateral branches. Attached to the mycelial filaments are lateral spores on short stalks, and at the ends of the filaments are bunches of three to nine spores.

The general conditions of culture are the same as in most fungi. The *sporotrichum* is pathogenic, not only for man, but also for many

animals, including the monkey, cat, rabbit, mouse and guinea-pig. Intraperitoneal inoculation in the rabbit produces lesions of a pseudo-tuberculous appearance. Subcutaneous inoculation in the cat produces various lesions resembling the different types of human sporotrichosis.

Vegetables appear to be the usual habitat of the parasite. Living plants may be infected with it, and there are several observations which show that inoculation may take place by handling vegetables. But the mechanism of human infection does not appear to be always the same. In some cases the parasite gained entry through a cutaneous lesion, but more often the mode of penetration has

Fig. 152.—Pure culture of sporotrichum. (Monier-Vinard.)

escaped notice, and the lesions have developed without any primary lesion having been discovered. In fact, it is possible to distinguish two principal varieties of sporotrichosis, according as the mode of infection is apparent or obscure.

Sporotrichosis occurs chiefly in adults, rarely in children. The affected subjects are often in good health, but a debilitating cause favouring infection is often present, such as senility, gout, cirrhosis of the liver, syphilis and tuberculosis. The two last affections are of particular importance; for, apart from the interest of such a morbid coincidence, it shows that sporotrichosis should not be excluded because the subject is tuberculous or syphilitic.

Symptomatology.—I. *Pathogenic classification of forms.*—We have already seen that the mode of human infection is sometimes quite evident, but sometimes eludes the most careful examination. In the

first instance, the disease is accidental; traumatism opens the door to infection, which manifests itself by a local primary lesion, comparable in its mode of production to anatomical tubercle. Secondary lesions may appear in the neighbourhood of the primary lesion, but their limitation to the region primarily affected clearly distinguishes this form, which may be called *localised sporotrichosis*, from all other forms.

The term *septicæmic sporotrichosis* may be given to another form of the disease, characterised by the simultaneous appearance of numerous lesions in several parts of the body. The fact of propagation by the blood-stream is in most cases confirmed by the general symptoms which precede or accompany the appearance of the lesions; Widal and Weill, by positive culture from the blood of one of their patients, showed that the infection was truly septicæmic. As a rule, the point of origin of this general infection escapes notice. Possibly there is a latent lesion of the mucous membrane or viscera. De Beurmann and Gougerot think that the lesion should be sought for in the pharynx and tonsils. It is, however, possible that the *sporotrichum* may be a saprophyte of the natural cavities, capable of becoming virulent under unknown influences, and thus give rise to general infection without any appreciable lesion.

II. *Clinical description.*—**Localised Sporotrichosis.**—In the cases reported by Schenk, de Hektoen, Perkins and Brayton, the lesion of inoculation was a prick or cut in the finger; a contused wound of the forehead in a case of Beurmann and Gougerot; a cut finger in Domenici's and Rubens-Duval's patient. In the last case, the local infection appeared forty-seven days after the wound, which was made while picking vegetables. It commenced as a swelling of the pulp of the finger, which was transformed, after an incision, into an indolent ulcer with irregular and swollen borders; this persisted for several weeks, and gave rise to gummatous lymphangitis.

Gummatous Lymphangitis.—In the neighbourhood of the initial lesion the lymphatic vessels are transformed into large, red, hard, nodular cords, apparent to the eye as well as to palpation; here and there, their course is marked out by gummatous swellings, varying from a pea to a nut in size. The gummas are at first hard, but soften slowly, usually in a few weeks, and their contents are evacuated through a narrow opening, which discharges a seropurulent liquid containing numerous parasites. The lymphangitis may undergo complete spontaneous resolution, but more often it persists indefinitely, and these localised lesions may lead to secondary septicæmic dissemination.

Septicæmic Sporotrichosis.—This form is far more common, and is the one which has been most frequently observed in France. In many cases the lesions have been preceded by general symptoms,

sometimes severe. On account of their mild nature and ordinary characters, the manifestations were not always attributed to their true cause, and it was at first thought that the sporotrichosic eruption coincided with some catarrhal affection. The almost constant presence of these general phenomena now shows that they must be regarded as the clinical reaction of the septicæmic condition; this has also been proved by blood culture.

The symptoms are slight fever (38° to 39° C.), with general weakness and malaise; sometimes vomiting and signs of bronchitis. The last symptom, which is very frequent, possibly indicates that this diffuse infection originates in the respiratory passages. These symptoms last from three to six days, and are followed by the sudden appearance of multiple nodular lesions. The first eruption may be followed by several successive eruptive outbreaks, increasing the number of the lesions.

III. *Analysis of the lesions.*—The lesions are generally cutaneous, and include subcutaneous abscess, subcutaneous gumma, and lesions of the dermis, epidermis and mucous membrane. Lesions of the bones, periosteum and muscles are also often observed, and more rarely visceral changes. Diverse lesions are often seen in the same subject.

Sporotrichosis with Abscess Formation.—This type is rather rare. Dor observed a case in which there were large cold abscesses, having the appearance of tuberculous abscesses. Some of these abscesses contained half a litre of pus. But, as a rule, large abscesses are comparatively rare.

Disseminated Subcutaneous Gummas.—This is the form of sporotrichosis described for the first time by Beurmann and Ramond, and which has since been met with by several observers. The gummas form painless nodules, situated in the subcutaneous tissue. They are seldom less than five in number, usually twelve to fifteen, sometimes more than fifty, and vary from a pea to a walnut in size. They are generally irregularly disseminated, but sometimes symmetrical. They occur chiefly on the forearms, legs and thighs, sometimes on the deltoid and pectoral regions, less often on the face, neck and trunk. The causes which determine these localisations are unknown, but in some cases the first or the largest lesion is preceded by traumatism, such as a violent contusion or repeated pressure.

The gummas are at first hard and resistant, and may remain so for a long time, they may even disappear without leaving any trace; but more often they slowly increase in size, adhere to the subjacent tissues, undergo softening, and open externally. The skin becomes red or purple, and viscid, grayish yellow pus is evacuated through a narrow opening. The abscesses heal spontaneously or under the

influence of treatment, and leave a cicatrix which is sometimes depressed, pearly white or brown, sometimes raised and cheloidal in appearance.

Sporotrichosic Ulcers.—These sometimes result from spontaneous opening of a subcutaneous abscess, but more usually occur after incision. The borders are sometimes swollen, sometimes thin

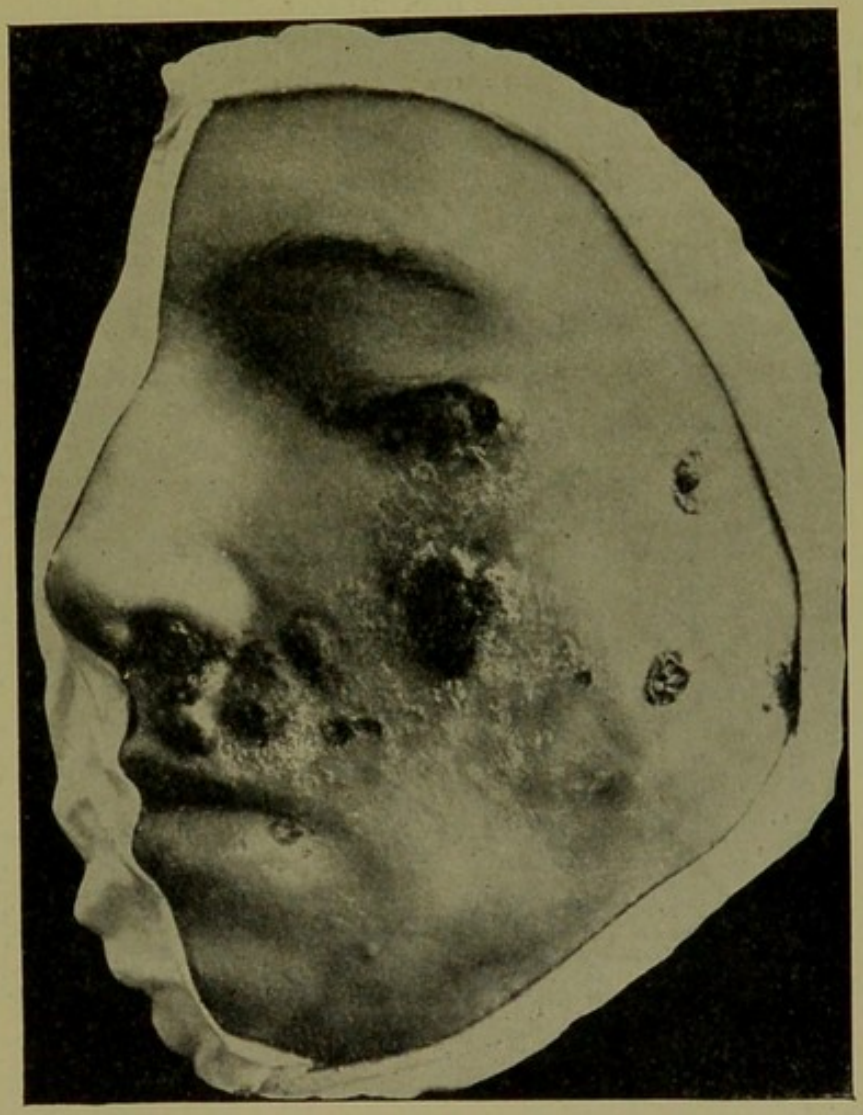


Fig. 153.—Sporotrichosis with open gummas and crusts.
(St Louis Hospital Museum.)

and undermined; the ulcer is atonic, painless, and sometimes very extensive; it closely resembles certain tuberculous ulcers. It may persist for a long time and originate secondary auto-inoculation.

Dermic Lesions.—These may be produced by two different processes; sometimes there is more or less extensive infiltration of the dermis during the evolution of a subcutaneous nodule; sometimes the dermis is affected primarily, the subcutaneous tissue being comparatively free. This type is observed in parts where the skin

is thin; on the face and flexor surfaces of the limbs. The dermic nodules vary from a millet seed to a shilling in size; they are soft, elevated, slightly moist, and of a barley-sugar colour. They may remain in this condition for some time and then extend, become confluent, and soften in the centre; or they may undergo cicatrisation without ulceration.

Epidermic Lesions.—These present two varieties: one resembling tinea circinata, the other kerion; so that the epidermic lesions produced by sporotrichosis closely resemble those due to trichophytons.

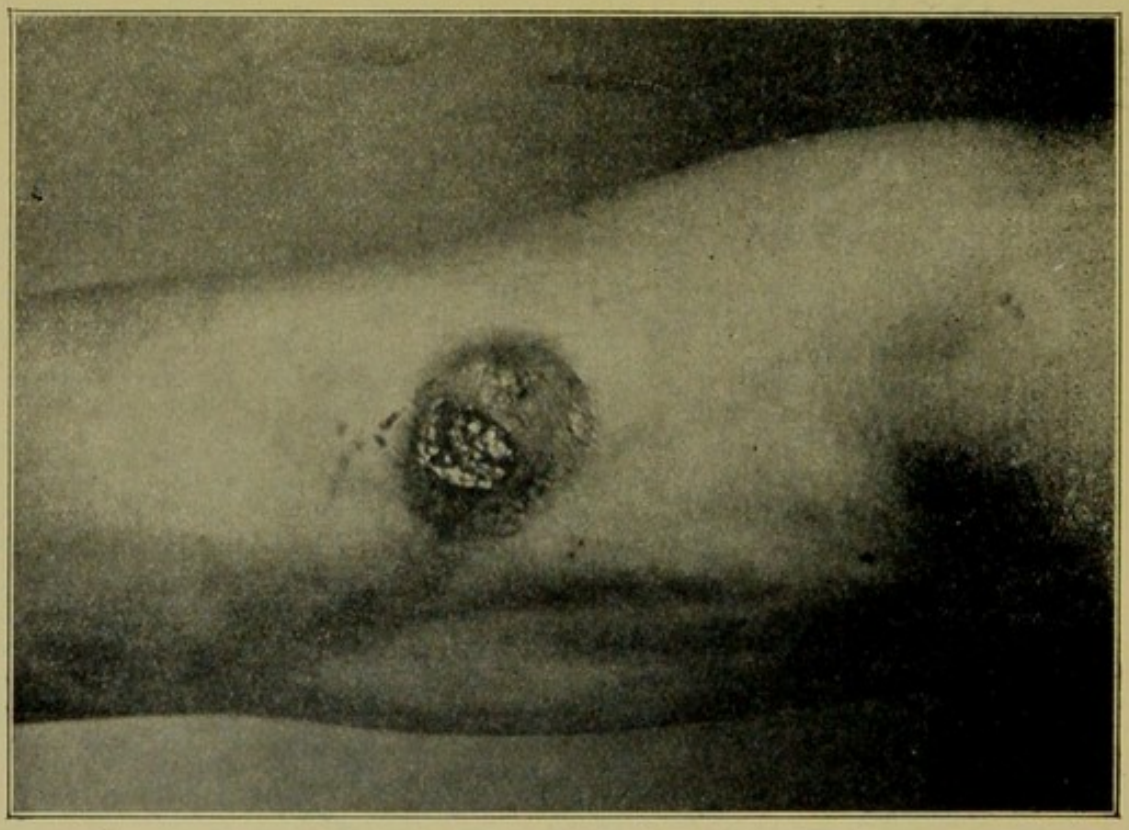


Fig. 154.—Sporotrichosis (ulcerated gumma).

The *trichophytoid form* occurs round large sporotrichosic ulcers, which are surrounded, at a variable distance, with a ring of small red papules, which become vesicular and are grouped in circles, with squamous centres.

Sporotrichosic kerion, described for the first time by Gaucher and Fouquet, was observed on the dorsal surface of the hand. The lesion was circular, about two inches in diameter, with a slightly indurated base movable over the deeper tissues, and surrounded by a red inflammatory areola. The border was raised and smooth, sloping externally, slightly vegetating, and partly undermined towards the centre of the lesion. Pressure on the internal border

caused exudation of pus. The centre of the lesion was dry, granular, and apparently in process of repair. This lesion, which resembled a suppurating trichophytic lesion, originated in an open subcutaneous gumma. There was no glandular enlargement.

Sporotrichosis of the mucous membrane. — De Beurmann and Gougerot observed a case in which there was chronic ulceration of the tonsil, the exudation of which contained numerous parasites. Brodier and Gastou observed cases in which, besides multiple cutaneous lesions, there were pharyngeal and laryngeal lesions, which were found at the autopsy to consist of red infiltration of the mucous membrane, very different to the lesions of syphilis and tuberculosis.

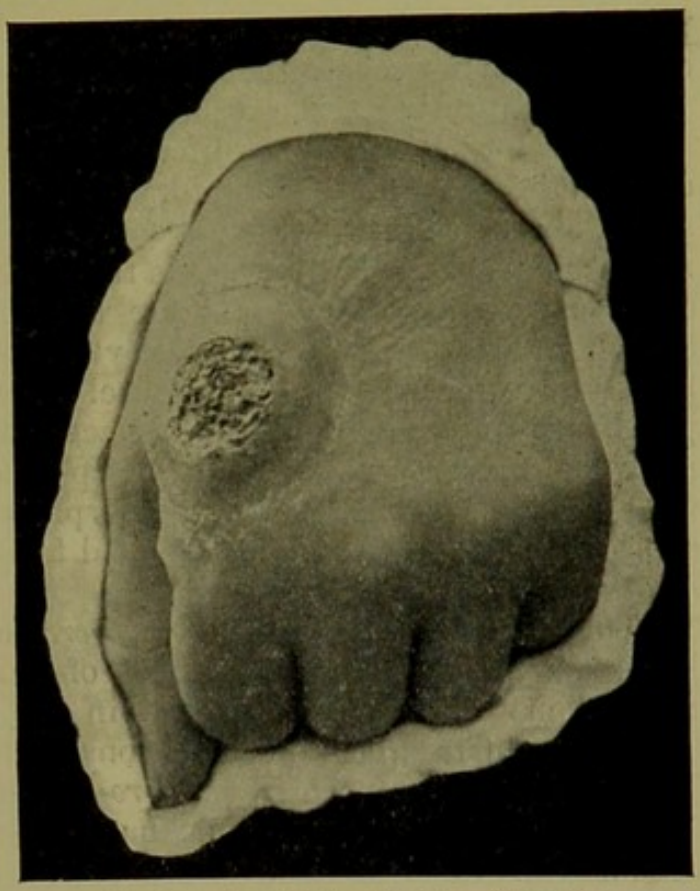


FIG. 155.—Sporotrichosic kerion. (St Louis Hospital Museum.)

Extracutaneous and Visceral Lesions. — Although less common than the preceding lesions, these are by no means rare. Sporotrichosic lesions may occur in the muscles, tendon sheaths, periosteum and bones. In a case of Rochard and Duval's, the *sporotrichum* was the only organism found in a pyonephrosis. Lastly, the sputum of patients affected with septicæmic sporotrichosis has given positive cultures.

General Characters of Cutaneous Sporotrichosis. — The cutaneous lesions of this disease have certain general characters, which are of more assistance in the diagnosis than the aspect of any individual lesion. These characters are: the usual multiplicity of the lesions; the frequent association of dermic and subcutaneous lesions; their painlessness and slow evolution, with little tendency to spontaneous evacuation, and slow cicatrisation when ulcerated; the usual absence of glandular enlargement, both in the septicæmic form and in the localised form with lymphangitis. Glandular affection, when it occurs, is due to secondary infection of the ulcers by the common agents of suppuration.

PATHOLOGICAL ANATOMY.—We shall not describe the pathological

histology of each of the cutaneous lesions of sporotrichosis; we shall only describe the structure of the most common manifestation, the *subcutaneous gumma*. As this generally suppurates in the centre, it is in reality an abscess. The contents consist of polynuclear neutrophiles, together with mononuclear vacuolated cells of the type of ordinary macrophages. The walls are very thick, and show intense vasculo-connective tissue reaction, manifested by vascular congestion and new formation, by proliferation of the fixed connective-tissue cells, and diapedesis of migratory cells. Here and there are microscopic abscesses in the thickness of the wall, containing polynuclear neutrophiles, while in other parts the hypertrophied connective-tissue cells become acidophile, and are transformed into epitheloid cells and giant cells; lastly, the leucocytes grouped round the vessels are mostly transformed into plasma cells, constituting plasmomas comparable to those of syphilis. In fact, the characters of the inflammatory process resemble those of syphilis and tuberculosis.

DIAGNOSIS.—The great polymorphism of the manifestations of sporotrichosis has for a long time led to its confusion with a number of other affections.

The large abscesses resemble *tuberculous abscesses* connected with bones or joints; but the absence of painful points and the usual multiplicity of the lesions assist in the diagnosis.

The nodular subcutaneous lesions, disseminated or lymphangitic, may be mistaken for *leprosy*, *fibro-lipomatosis*, *chronic staphylococcic abscess*, seen in young children; but especially for *syphilitic gumma*, or for the various manifestations of subcutaneous tuberculosis, especially *tuberculous gumma*. The multiplicity of the lesions, their painlessness and slow evolution, and the absence of glandular enlargement, suggest the possibility of sporotrichosis.

The same characters serve to distinguish the dermic lesions from *lupus* and certain *lupiform syphilides*, to which they have sometimes a great resemblance. Sporotrichosic ulcers have often the same appearance as *tuberculous ulcers*. Sporotrichosis is not excluded by the fact that the patient is affected with syphilis or tuberculosis, for it has been shown that the two infections may coexist.

It is in the laboratory that the diagnosis is finally settled. It is only in rare cases that a biopsy has decided the question. The pus may contain elongated granular bodies which resemble the *sporotrichum*, but these are few in number, and are difficult to distinguish from cellular debris. The chief methods necessary to establish the diagnosis are culture, agglutination, and the reaction of fixation.

The two first methods are easy to carry out. Cultures are made by inoculating Sabouraud's media (peptonised agar with maltose or glucose) with pus or fragments of lesions removed aseptically. The tubes are kept uncovered at the ordinary temperature of the labora-

tory. These two conditions are essential for the development of the parasite.

The phenomenon of agglutination in sporotrichosis was discovered by Widal and Abrami. These observers have shown that the serum of patients affected with this disease causes agglutination of the spores of the parasite. They recommend a culture about a month old. The colonies collected from the surface of the agar are ground in a mortar, and then mixed with physiological salt solution; this liquid is then filtered, to separate the fragments of mycelium from the spores, which pass through the filter. A drop of the patient's serum is added to different quantities of the filtered liquid containing the spores in suspension; after a few minutes, the spores become united together in a mass, or agglutinated. The power of agglutination is always high, especially with cultures a month old; Widal and Abrami obtained it at 1 in 800. The maximum degree of reaction should always be obtained, for, in sporotrichosis, as in typhoid fever, the patient's serum is also agglutinating for pathogenic agents allied to the one in question, although in lesser degree. For instance, the serum of patients affected with actinomycosis may also give an agglutination reaction with the *sporotrichum*, and inversely. It is therefore necessary to obtain the extreme limit of agglutinative value.

The reaction of fixation has been studied by the same observers, who showed its constant presence in sporotrichosis. In spite of its great interest, we only mention it here, for its practical difficulties debar it from ordinary use.

Prognosis.—As regards the cutaneous manifestations, the prognosis cannot be considered grave, for, as a rule, the general health is not seriously affected. Multiple lesions of the face, however, may be very disfiguring. The visceral lesions of sporotrichosis are not frequent enough for any prognosis to be formulated, but the effects of iodide treatment show that the disease may be regarded as curable.

Treatment.—Iodide of potassium is the drug to be prescribed, for many cases have been cured by this alone. Surgical intervention is not as a rule indicated, especially as sporotrichosic abscesses, when incised, often become transformed into ulcers, which heal more slowly, even under iodide treatment, than unbroken gummas. When it is necessary to evacuate a large abscess, aspiration is preferable to incision.

Iodide of potassium should be given in daily doses of 30 to 75 grains, and should be continued not only till the lesions have completely disappeared, which generally takes two or three months, but for several weeks afterwards; for new lesions may appear if the drug is discontinued too soon. Iodide of potassium may also

be applied locally to the ulcers, in the form of dressings soaked in a weak solution of the drug. It may also be injected into and around abscesses; this method is especially useful when the drug cannot be taken internally.

BLASTOMYCOSIS.

Blastomycoses are affections caused by fungi called Blastomycetes. By this term, Vuillemin includes all fungi which may appear in the form of budding elements (yeast) at a certain period of their life history. The term is therefore very vague and rather comprehensive, and has no precise botanical value. In fact, among the Blastomycetes which have been described, some certainly belong to the order of Ascomycetes, and others should be classed with the Hyphomycetes. Again, some botanists reject the term Blastomycetes. However, in medicine, the term has become stereotyped by use, so that it is better to use it, even though incorrect, than to replace it by another which in the present state of our knowledge of these parasites might be no better.

The first observation made on pathogenic blastomycetes in man is that of Troisier and Achalme (1893). These observers described a case of sore throat caused by a yeast, resembling thrush in its clinical appearance. A year later, Gilchrist in America, and Busse and Buschke in Germany, published simultaneously cases of cutaneous blastomycosis and blastomycosis with multiple foci. Since then numerous observations have been published, especially in the United States.

The cases which we shall include in the group of blastomycetic affections have not all been published under the name of blastomycosis. In America, many have been described as cases of oidiomycosis, or as affections due to coccidia or protozoa, thus causing confusion. The American observers who have described cases of dermatitis due to protozoa, are even of opinion that these cases should be classed with oidiomycosis or blastomycosis, which are equivalent terms for these authors. If the American cases were identical with those observed in Europe the question would be solved, but among the latter some correspond to the type described by Gilchrist, while others agree with the type described by Busse and Buschke, which is quite different.

These two clinical types correspond to different pathogenic agents, and for this reason it is necessary to divide the group of blastomycetic affections into two classes. The first includes all the American observations, and perhaps a certain number of European cases; it may be designated as American blastomycosis

or oidiomycosis, for the pathogenic agent in these cases was a fungus related to the *Oidium*. The second class includes the cases described by Busse and Buschke and other European observers, and constitutes true blastomycosis or saccharomycosis, for the yeasts isolated by these observers were either true *saccharomyces* or incompletely developed forms.

American Blastomycosis, or Oidiomycosis.

The cases of blastomycosis or oidiomycosis observed in America are very similar to each other. We shall give a clinical description of these cases, and then compare them with cases reported in other countries, which are difficult to classify.

ETIOLOGY.—This affection is not very rare, at any rate in America, for, since the original case of Gilchrist (1894), about fifty cases have been published in the United States. The disease has been observed at all ages, except in children. It appears to be more common in men, especially those who work in farms. The mode of contagion is generally unnoticed, but in one case the parasite appears to have been introduced by a splinter of wood; in another case a doctor inoculated himself accidentally while making an autopsy on a man who died of blastomycosis. Auto-inoculation round the original lesion is often caused by scratching.

SYMPTOMATOLOGY.—The principal manifestation is blastomycetic dermatitis. This is generally primary, and may constitute the whole affection; in other cases, the pathogenic agents invade the viscera, especially the lungs, and cause death. Sometimes infection takes place by the respiratory tract, when the disease is at first visceral and the cutaneous lesions are secondary, due to dissemination of the parasite.

Blastomycetic Dermatitis.—The lesions are usually multiple, and occur chiefly on the exposed parts of the body. The face is nearly always affected (nose, eyelids, cheeks, lips, chin and ears), and very often the back of the hand. Other parts may be occasionally affected; scalp, neck, chest, back, abdomen, scrotum and limbs.

The lesion begins as a red, indurated papule, which soon becomes a pustule; the latter becomes covered with a brown crust, under which is an irregular bluish red surface secreting a little glairy muco-pus. This lesion extends slowly, and attains a diameter of about an inch in two to six months. At the same time the surface becomes covered with large vegetations, separated by deep irregular fissures full of pus, producing a cauliflower appearance, which is one of the characteristics of blastomycetic dermatitis. The skin round the vegetations is bluish red, infiltrated, and abruptly raised above the surface of the normal skin. It presents a number of yellow

spots, from which thick pus can be expressed; these correspond to miliary abscesses situated in the epidermis and dermis.

The lesion extends peripherally, while the centre subsides. It loses its vegetating appearance and becomes granular, red and moist. Sometimes ulcers develop, some flat, others crateriform. In some cases the centre of the lesion tends to cicatrisation, and becomes dry, smooth and white.

The disease progresses by successive outbreaks, separated by periods of retrogression or stationary periods. New foci are caused by auto-inoculation. Some lesions remain small and stationary, while others extend and form large patches, which may occupy the whole shoulder or the greater part of the forearm. These patches may become inflamed and secrete sanious and fœtid pus. The lesions cause but little pain, except when they are situated in places where the skin is subject to frequent tension. The general health remains fairly good; there is no fever, but always some wasting.

Blastomycetic dermatitis most often constitutes the whole disease, which in this case may undergo spontaneous cure. Healing always begins in the centre of the lesions; the vegetations atrophy and leave a smooth cicatrical surface, the purulent secretion ceases, and the miliary abscesses disappear. The cicatrix is at first thick and indurated, with a red and granular periphery; later on it becomes thin and movable over the subjacent tissues. Sometimes it is marked by telangiectases; in rare cases it may become cheloidal.

Such is the usual clinical picture of blastomycetic dermatitis, but sometimes the lesions are much less typical, and present the form of gummas or acneiform papules. Occasionally, the disease is quite atypical, and consists of a simple pustular eruption.

As already mentioned, the lesions are usually multiple. Sometimes they are localised to the nose and median part of the face, simulating lupus or tertiary syphilis. In two cases reported there was perforation of the nasal septum, and in another case there were ulcers simulating Kaposi's serpiginous ulcerative folliculitis of the nose. A single localisation may also occur on the back of the hand, resembling verrucose tuberculosis.

We mention these diverse observations because, in the present state of the question, it seems reasonable to connect them with oidiomycosis, but with the reservation that this connection cannot be established with certainty.

Visceral Manifestations of General Blastomycosis. — The visceral manifestations are generally secondary to cutaneous lesions. The latter undergo acute exacerbation, become painful, and suppurate freely. At the same time, the glands become enlarged and the patient suffers from cough, at first dry, afterwards accompanied by muco-purulent and often blood-stained expectoration. Sometimes

there is actual hæmoptysis. There is high fever with profuse sweating, and considerable wasting. The disease thus simulates rapid pulmonary tuberculosis, but there are no tubercle bacilli in the sputum. This form is rapidly fatal. At the autopsy, multiple visceral lesions are found, especially in the lungs.

In other cases infection takes place through the lungs, the symptoms commencing with cough and blood-stained expectoration; later on, subcutaneous nodules appear, which open externally and become vegetating, producing lesions somewhat similar to those of primary blastomycetic dermatitis.

Sometimes the pulmonary and visceral infection is so severe that the cutaneous lesions have no time to develop. In one case reported, the disease resembled pyæmia in its evolution, commencing with pleurisy and painful swellings of the large joints, followed by periosteal abscess of the frontal bone and great enlargement of the subclavicular glands, muco-purulent, blood-stained expectoration, high fever, repeated rigors and profuse sweating. The patient died one month after the onset, general blastomycosis being found at the autopsy.

Pathological Anatomy.—The lesions of blastomycetic dermatitis affect both the epidermis and the dermis. The epidermis undergoes active proliferation; it not only covers the papillomatous vegetations from the papillary body, but also sends out long processes between the hypertrophied papillæ, irregular in form, dimensions and direction. These processes ramify in such an exuberant and irregular way, that, in sections, they simulate epithelioma.

Within these epidermic proliferations are small intra-epidermic abscesses, constituted essentially by polynuclear leucocytes, and eventually by migratory connective-tissue cells, which become transformed into epithelioid cells and giant cells. Within these small abscesses are found the parasites, either free or within the giant cells. The parasites are generally scanty, and take the form of round or budding bodies with a double outline: they are rather large, and may be 30 micromillimetres in diameter.

The lesions of the dermis are very complex; in an œdematous tissue, where the fixed cells are in a state of inflammatory reaction and which is infiltrated with cells of various kinds, vacuolated cells, plasma cells, mast-cells, lymphocytes, neutrophile and eosinophile polynuclear leucocytes, are found denser, scattered islets of cells. These islets consist of masses of neutrophile cells representing miliary abscesses, or small groups of epithelioid cells and giant cells, or plasma cells grouped in plasmomas. Here and there, but always in small numbers, are found the parasites, either in the miliary abscesses or among the giant cells.

Visceral lesions occur chiefly in the lungs, which appear riddled

with yellow or gray nodules, resembling miliary tubercles or abscesses. Similar lesions are found eventually in the liver, spleen, kidneys, suprarenal capsules, and sometimes in the testicles. The mesenteric glands are often suppurating. Subperiosteal or intraosseous abscesses are also sometimes found. Amyloid degeneration of the kidneys has been reported. The visceral lesions differ from the cutaneous lesions by the abundance of parasites, which may be present in such quantity that, in some places, the tissue of an organ is replaced by colonies of blastomycetes.

The *parasites* are round bodies with a refractive membrane and double outline; they are rather large, and may attain the size of 30 micromillimetres. They may occur in a budding form, or may contain a large number of small round bodies, which are probably spores. They may be cultivated on ordinary media, but thrive best on sugary media. On solid media, they form a white, downy or creamy layer on the surface. There are considerable differences in different specimens. Some parasites produce, in culture, chiefly budding forms and little mycelium, while others produce not only an abundant mycelium but also aerial hyphæ.

The first colonies obtained from pathological products are always slow in growth, and take a long time to appear. The experimental inoculation of these fungi is very uncertain; the white mouse seems to be the most susceptible.

Diagnosis.—The diagnosis depends largely on exclusion of other diseases, especially syphilis and tuberculosis. Several cases have been discovered by systematic research and laboratory examination, even when the diagnosis of lupus or verrucose tuberculosis was probable. The only means of establishing the diagnosis with certainty is by culture of the parasite.

Treatment.—Scraping, cauterisation and radiotherapy give good results in blastomycetic dermatitis. In generalised blastomycosis, treatment has proved ineffectual, although iodides have been tried in some cases.

Iodide treatment, in the present state of our knowledge, is the most rational, and, whatever accessory treatment is employed, should be instituted from the first. If moderate doses produce no effect, the doses should be increased up to 2 drachms daily.

True Blastomycosis, or Saccharomycosis.

Observations concerning this group are few in number. There are two groups of cases to be considered: one consisting of tumours in the clinical sense of the word, the other of multiple abscesses.

Blastomycetic Tumours.—Only two cases of this kind have been published. In Curtis's case, the growth was situated in the

upper part of the right thigh, at the base of Scarpa's triangle, and was double the size of the fist. There was also a large abscess in the right lumbar region, from which exuded a small quantity of thick flocculent pus. The skin was healthy and quite intact over the tumour. Incision showed that the tumour was simply a subcutaneous pouch enclosing soft and gelatinous tissue, resembling a sarcoma in a state of advanced mucous transformation.

In the case published by Blanchard, Schwartz and Binot, there was a "fluctuating mass in the right iliac fossa, dull to percussion, and slightly painful on pressure." After laparotomy, a gelatinous, yellowish white mass was found in the peritoneum; "this large mass (a litre at least) filled a pouch in the midst of which floated the cæcum and appendix."

In these two cases the gelatinous masses consisted of proliferating fungus, surrounded by a gelatinous capsule. General reaction was very slight in both cases. In the second case, infection appeared to be of appendicular origin, for the parasites were found in the wall of the appendix.

Blastomycetic Abscess.—There are three cases of this kind reported: by Busse and Buschke, by Vuillemin and Legrain, by Hudelo, Rubens-Duval, and Laederich.

The first case presented at first small ulcers on the nape of the neck and the forehead, afterwards suppurative osteoperiostitis of the tibia. The ulcers had slightly undermined borders and a flat granular base, discharging a thick red secretion; they varied from a lentil to a sixpence in size. The osteoperiostitis resembled a gumma, and contained 40 cubic centimetres of a reddish fluid, which was evacuated by incision. Later on, ulcers appeared on the face, and osteoperiostitis developed on the right ulna and the sixth rib. The patient suffered from wasting, acute abdominal pain, and vomiting, and died from exhaustion. At the autopsy, besides the cutaneous and osseous lesions, blastomycetic foci were found in the spleen, kidneys and lungs. These consisted of a whitish substance, firm in some lesions, softened or liquefied into thick pus in others.

The second case, in which infection took place by the mouth, presented submaxillary subcutaneous abscesses, from which sanious fluid containing numerous parasites was evacuated by incision. The patient lost 13 lbs. in weight in six weeks, but left hospital before he was cured. In the third case, which I reported with Hudelo and Laederich, the cutaneous lesions consisted only in some acneiform papules on the face. As in Busse and Buschke's case, there was gummatous osteoperiostitis of the tibia. It was only by a biopsy that the diagnosis of blastomycosis was made, as the lesion was regarded as tuberculous, syphilis having been eliminated by the failure of antisyphilitic treatment. Subcutaneous abscesses then

developed in the hypogastrium, fronto-parietal region, left elbow, right great trochanter, occipital and mastoid regions. Finally, osteoperiostitis appeared on the sacrum. During the evolution of the disease there were several attacks of fever and considerable wasting (33 lbs.). The patient was cured by large doses of iodides.

Blastomycosis with multiple foci is therefore a disease which is characterised by the successive or simultaneous appearance of multiple abscesses on all parts of the body, generally reaching considerable dimensions. Some are subcutaneous, others subperiosteal. The latter at first simulate tuberculous or syphilitic gummas; they afterwards soften and give rise to fluctuating abscesses, which have little tendency to spontaneous evacuation.

The rapid appearance of multiple disseminated lesions tending to persist as cold abscesses distinguishes blastomycosis from syphilis and tuberculosis, but it is more difficult to eliminate sporotrichosis, which also gives rise in a short time to numerous gummas and torpid subcutaneous abscesses. But, in the three cases of blastomycosis which we have just considered, there was rapid and pronounced wasting, which is slight in cases of sporotrichosis. In the case of a patient affected with multiple abscesses of torpid evolution, therefore, the presence of rapid wasting is in favour of blastomycosis rather than sporotrichosis.

Pathological Anatomy and Parasitology.—Blastomycetic tumours are formed entirely, or almost entirely, by exuberant proliferation of parasites. Blastomycetic gummas and abscesses also contain a considerable number of parasites, but in this case the parasites give rise to a strong reaction on the part of the organism. This is essentially a connective-tissue reaction, of the macrophagic phagocytic type. The connective-tissue cells undergo hypertrophy of their protoplasm and multiplication of their nuclei, and are transformed into giant cells and large plasmodia with vacuoles which contain the parasites. This plasmodial and giant-celled transformation is the characteristic feature of the reaction of the organism to blastomycetic infection; the remaining features, such as the diapedesis of neutrophile polynuclear cells and the presence of eosinophiles and plasma cells, are only accessory.

The epidermis showed no changes in the case which we examined, and no changes of any consequence in Busse and Buschke's case.

True blastomycosis or saccharomycosis, therefore, differs from American blastomycosis or oidiomycosis in the reaction being essentially a connective-tissue reaction of a uniform type, leading to the formation of giant cells. In oidiomycosis, there are important changes in the epidermis, manifested by irregular proliferation of the interpapillary processes and the presence of intra-epithelial abscesses. The connective reaction which is added to the epithelial

reaction is composite; giant cells, epithelioid cells, plasmomas, and miliary abscesses formed by neutrophile polynuclear cells, rendering the histological picture very complex.

In saccharomycosis the parasites are always very numerous in sections, and of small size, from 2 to 10 micromillimetres; while in oidiomycosis they are sparse, but much larger, up to 30 micromilli-

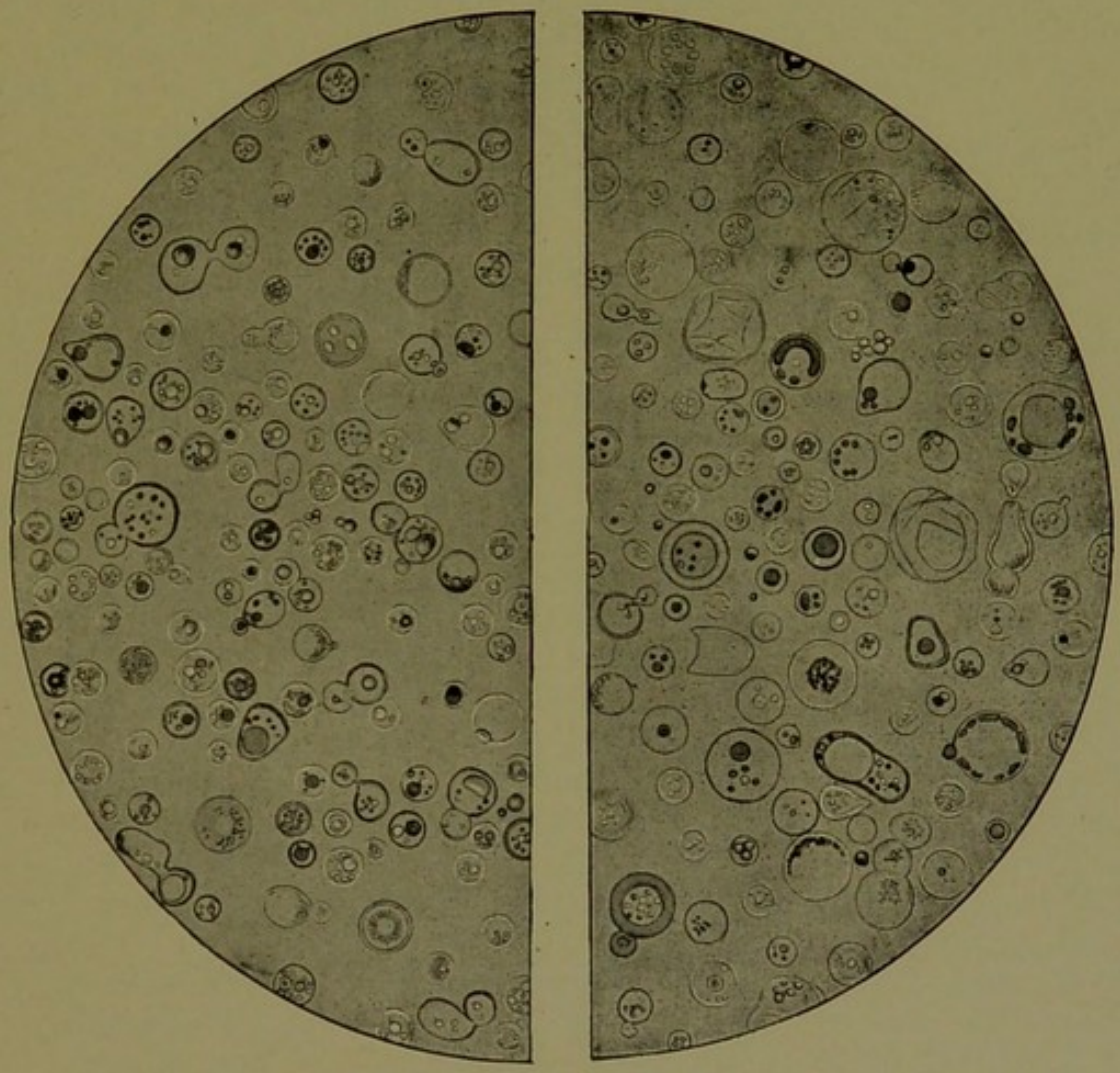


Fig. 156.—Fresh yeast from blastomycosis; culture on agar. (After Hudelo, Rubens-Duval, and Laederich.)

metres. The two affections are therefore distinct, clinically, histologically, and mycologically.

The tumour form of saccharomycosis only differs clinically from the form with multiple gummas and abscesses. In fact, in the course of comparative experimental research undertaken with Laederich, we have succeeded in reproducing sometimes tumour masses without inflammatory reaction, sometimes connective tissue inflammatory reaction of the same type as that observed in man.

The animals most susceptible to the pathogenic action of these

yeasts appear to be mice and new-born guinea-pigs. The yeast grows well on the usual media at the ordinary temperature of the laboratory; but it is especially abundant on sugary media, such as Sabouraud's (agar 1½, peptone 1, glucose or maltose 4, water 100 parts).

The cultures grow rapidly; after inoculating the medium with pus or pathological tissue, the colonies appear on or before the fourth day; in stab cultures, the line of inoculation becomes visible after twenty-four hours. The colonies are white or creamy at first, afterwards they become yellow and then brown. When grown on potato, they are blackish gray. Both in cultures and in the tissues, the yeast is always in a budding form, but some cases show traces of a mycelium.

TREATMENT.—Incision of abscesses and excision of blastomycetic masses may be sufficient to cure some cases, but it is better to regard these as only palliative measures, and always administer iodides. The latter should be given in large doses, up to 2 drachms or more daily, continued for several weeks or months.

PARASITIC DERMATOSES.

VEGETABLE PARASITES.

FAVUS.

THIS is a parasitic affection, characterised by the presence of dry, yellow crusts, depressed in the centre in the form of a cup.

PARASITOLOGY. — The parasite of favus was discovered by Schönlein in 1839; two years later it was investigated by Grüby and Remak, who gave it the name of *Achorion Schönleinii*. Microscopic examination shows that this fungus is composed of spores and mycelium. The spores are mostly rectangular instead of round, more or less elongated, sometimes cubical, and varying in diameter from 3 to 10 micromillimetres. They may be single or in pairs, or in chains of three or four. They have a characteristic double outline, are unequal in size, and form irregular masses between the mycelial filaments. The mycelium is formed of cylindrical filaments about 3 micromillimetres in diameter, very tortuous, often septate, simple or branching (trichotomous or tetrachotomous). Some of the larger ones contain spores; others are filiform, non-ramifying, and without spores.

The achorion forms yellow cup-shaped cultures on agar, similar to the cups which occur on the skin. According to Quincke, there are two species of achorion; one which develops on the scalp, the other on the smooth skin. According to Bodin, the achorion of favus is a distinct species with five varieties, which are only differentiated by culture.

ETIOLOGY.—The only cause of favus is contagion. This may result from daily contact with an infected person, as occurs in families; from inoculation of an abrasion (for instance, a patient with favus may inoculate himself through scratching); from the clothes, hair-brushes, etc., belonging to an infected person; or by the air, which conveys the spores of the fungus.

There are also certain predisposing causes. According to Hardy, some individuals escape contagion and are even refractory to

inoculation; these are robust individuals; while children, especially those of a lymphatic type, and those living in poverty and unhealthy surroundings, are easily infected. Favus is more common in the country than in towns. Favus also occurs in animals, and may be conveyed from them to man. Draper and Anderson have shown that it occurs in mice and rats.

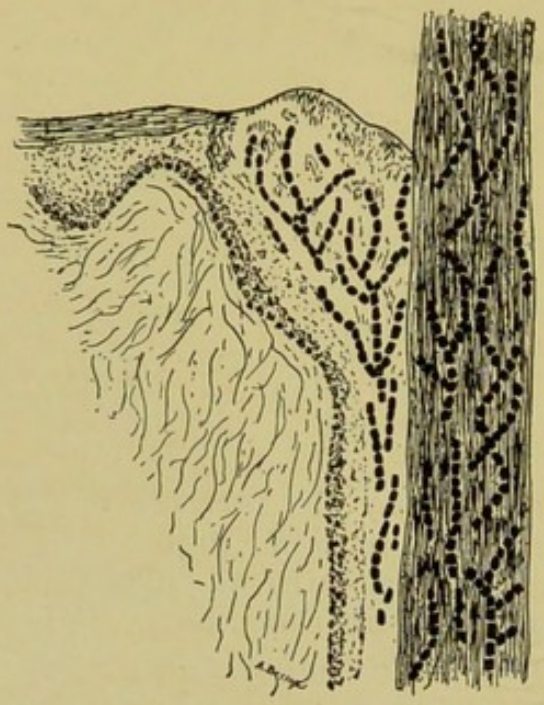


Fig. 157.—Vertical section through the centre of a hair, with part of a favus cup. (Besson.)

Favus affects chiefly the scalp, less often the skin and nails. Kundrat reported a case in which the œsophagus and stomach were affected. The fungus forms cups round the hairs. It is situated below the superficial layer of epidermis, and extends downwards in the hair sheath. According to Kaposi, it extends to the base of the hair follicle, penetrates the bulb and invades the hair from below. The infiltration of the hair is never so great as in ringworm; the spores form networks with longer meshes, and the hairs are less brittle than in ringworm.

Favus of the Scalp.

This begins in the form of red spots, which gradually enlarge and become covered with yellow, raised points, due to the parasite. These yellow points are at first very small, but by means of a lens they are seen to have a depressed centre pierced by a hair. In the course of a few weeks the characteristic dry, sulphur-yellow, cup-shaped crusts are formed, each cup being pierced by a hair. The cups may attain a diameter of nearly half an inch. They are formed of concentric layers, the oldest or central ones being white, the recent or peripheral ones sulphur-yellow. They are very adherent to the skin, and if removed reveal the red, moist depressed epidermis; under a lens the orifice of a hair follicle is seen in the centre of each depression. After a time, the thin layer of epidermis covering the cup ruptures, and the crust breaks up into powder. The crusts are rapidly replaced when they fall. Sometimes the cups are not apparent, the crusts being so thickly developed that they form a kind of carapace. When the crusts are old and discoloured they form thick raised masses, which break up and adhere to the

hairs, as in *impetigo granulata*. These three forms may coexist in the same subject.

The hairs invaded by the parasite become dull, dry and atrophied; they are easily pulled out, but do not break; sometimes they fall spontaneously. The characteristic spores are seen within the hairs, under the microscope.

Favus causes little or no itching. A characteristic sign is the

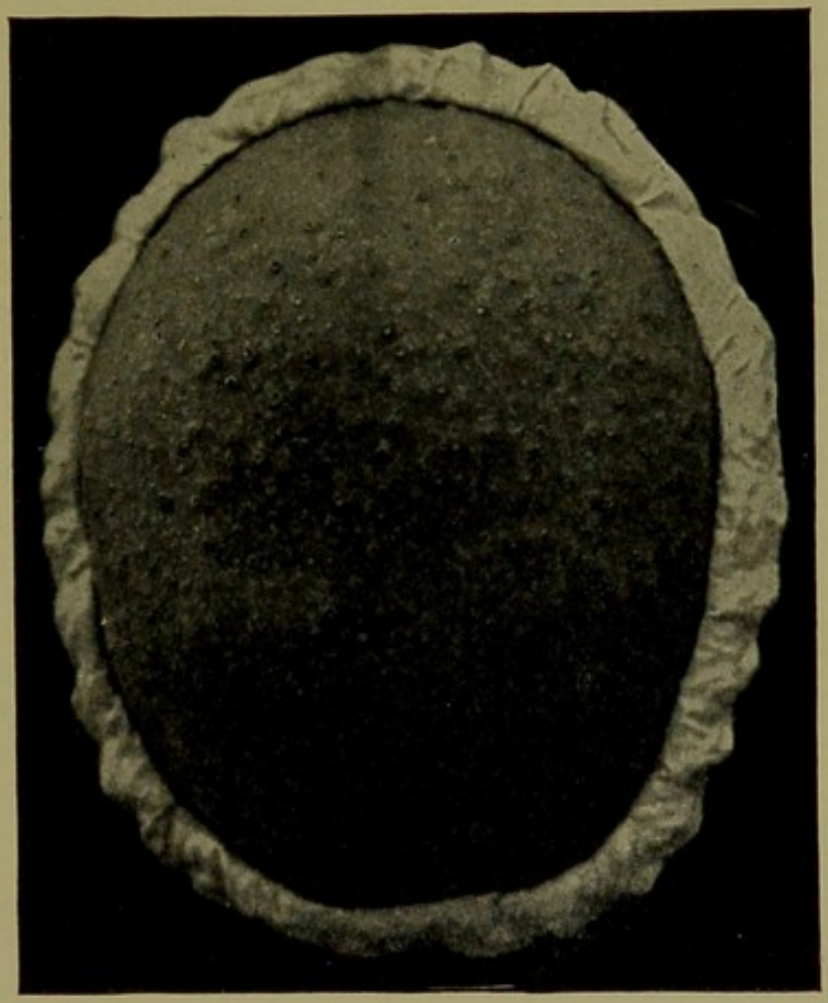


Fig. 158.—Favus. (St Louis Hospital Museum.)

mouse-like odour of the crusts, which is appreciable at some distance, and in doubtful cases assists the diagnosis.

Complications.—Secondary infection of the hair follicles may give rise to small circumpilary pustules, which soon dry up into crusts. There may also be a secondary eruption of impetigo. In predisposed subjects there is sometimes eczema. These secondary inflammations may cause cervical adenitis and suppuration, and subcutaneous abscess in the scalp. Favus may also be complicated by pediculosis.

Duration.—When untreated, favus is usually of long duration, and may last for fifteen or twenty years. However, it sometimes

undergoes spontaneous cure; but in this case the hair bulbs are destroyed, and the dermis undergoes a kind of cicatricial atrophy, characterised by a red, shiny, depressed condition of the skin, which becomes a smooth, white, indelible cicatrix. When the affection is treated early, it may be cured in a few months, without any cicatrix; but the hairs are nearly always dry and scanty.

Atypical Form.—Besnier has described a form which affects the whole scalp, and is constituted by thin, gray or yellow scales, or by a fine desquamation. On raising the scales, small yellow cups can sometimes be seen round some of the hairs. The characteristic mouse-like odour is often present, but not always. This form is accompanied by small patches of alopecia, which may be mistaken for alopecia areata, pseudo-alopecia areata, or lupus. The hairs are dry, dull, and atrophied, and are easily pulled out. The diagnosis of *atypical favus* is often difficult; it depends on microscopical examination of the hairs, and the presence of the characteristic odour.

DIAGNOSIS.—This is generally easy. In *eczema*, the eruption is more diffuse, and the crusts soft and moist.

In *impetigo*, the crusts are soft and yellow, and the hairs are not altered; but, as already mentioned, impetigo may complicate favus. In *seborrhœa sicca*, the crusts are soft and gray, and the hairs intact. The crusts of the *pustulo-crustate syphilide* differ from those of favus by their colour, form, and hardness. Lastly, *ringworm* differs from favus by its gray, finely squamous patches, and by the absence of cups; the spores are also different in the two affections.

Favus may also be diagnosed by the *serum reaction*, which was established in my clinic by Abrami. This discovery is of great interest, for it shows that superficial cutaneous affections are capable of developing changes in the blood similar to those caused by bacterial diseases. These changes are not peculiar to favus, but are also found in ringworm and cutaneous trichophytosis; they should also occur in erythrasma, pityriasis versicolor, etc.

For the serum diagnosis of favus, the best procedure is the Bordet-Gengou method of fixation. If the patient's serum is added to an emulsion of a culture of favus, it fixes the amboceptor on this emulsion, and causes absorption of a complementary serum added to the mixture. The reaction is negative with serum from patients affected with alopecia areata, seborrhœa decalvans, suppurative folliculitis; in fact, in every non-mycosic affection of the scalp.

With regard to the reaction of agglutination, this is not practicable because it is impossible to obtain an emulsion of the spores of the fungus, the spores being the only elements on which serums have a sufficiently agglutinating action.

TREATMENT.—After cutting the hair short, the crusts must first be removed by means of boracic fomentations. Further treatment

consists in epilation and the application of perchloride of mercury solution (1 in 500), or oxygenated water.

Epilation is painful and should not be performed at a single sitting, especially if it is necessary to epilate the whole scalp. It usually sets up an acute inflammatory reaction, which may be treated by emollient applications. Epilation should be repeated at the end of a month if the new growth of hair is not normal. After epilation the scalp should be treated with one of the above lotions and afterwards with some anti-parasitic ointment, such as turpeth (10 per 100). The general health requires tonic treatment.

The disease may be regarded as cured when the scalp becomes white and the new growth of hair is normal; but the case should be kept under observation for several months. Patients should be isolated during the whole course of treatment.

Favus of the Skin.

This is usually associated with favus of the scalp. It may occur on the face, back, abdomen and limbs. On the limbs, it generally affects the external surfaces, where the hairs are larger and more numerous; some cases have been reported in which it occurred on the glans penis. It presents the same characters as favus of the scalp. The cups are usually few in number, and isolated. In exceptional cases nearly the whole body may be covered. Sometimes the cups are less definite, surrounded by a bright red areola, and covered with yellow squames.

DIAGNOSIS.—This is sometimes difficult, and depends chiefly on the mouse-like odour, and on microscopic examination.

TREATMENT.—The crusts should be removed by boracic fomentations, followed by the application of antiseptic lotions, turpeth ointment, or tincture of iodine. Favus of the skin never leaves cicatrices, but only pigmented spots which slowly disappear.

Favus of the Nails.

This is chiefly observed in patients affected with favus of the scalp or skin, who inoculate themselves between the nail and the pulp of the finger by scratching. It may affect one or more nails, but never all of them. It begins underneath the nail, which becomes thickened and detached; under the nail are formed yellow masses. The nail soon becomes rough and irregular; the horny layers exfoliate, and the nail becomes thin and perforated.

DIAGNOSIS. — Favus is difficult to distinguish from *eczema* and *psoriasis* of the nails. The diagnosis is easier when there is perforation of the nail and yellow masses of favus underneath it, and when favus is present on the scalp. Microscopic examination is not always easy, but when positive removes all doubt.

TREATMENT.—The nail should be scraped down with a file till the deposits of favus can be removed with a curette, after which perchloride of mercury solution is applied (1 in 500).

TRICHOPHYTOSIS.

This term includes all affections of the skin due to the presence of the *Trichophyton* fungus: these are, ringworm, erythema or herpes circinata, trichophytic agminated folliculitis, parasitic sycosis and trichophytic onychia. The researches of Sabouraud have shown that these affections are caused by parasites of different species, but belonging to the same botanical group.

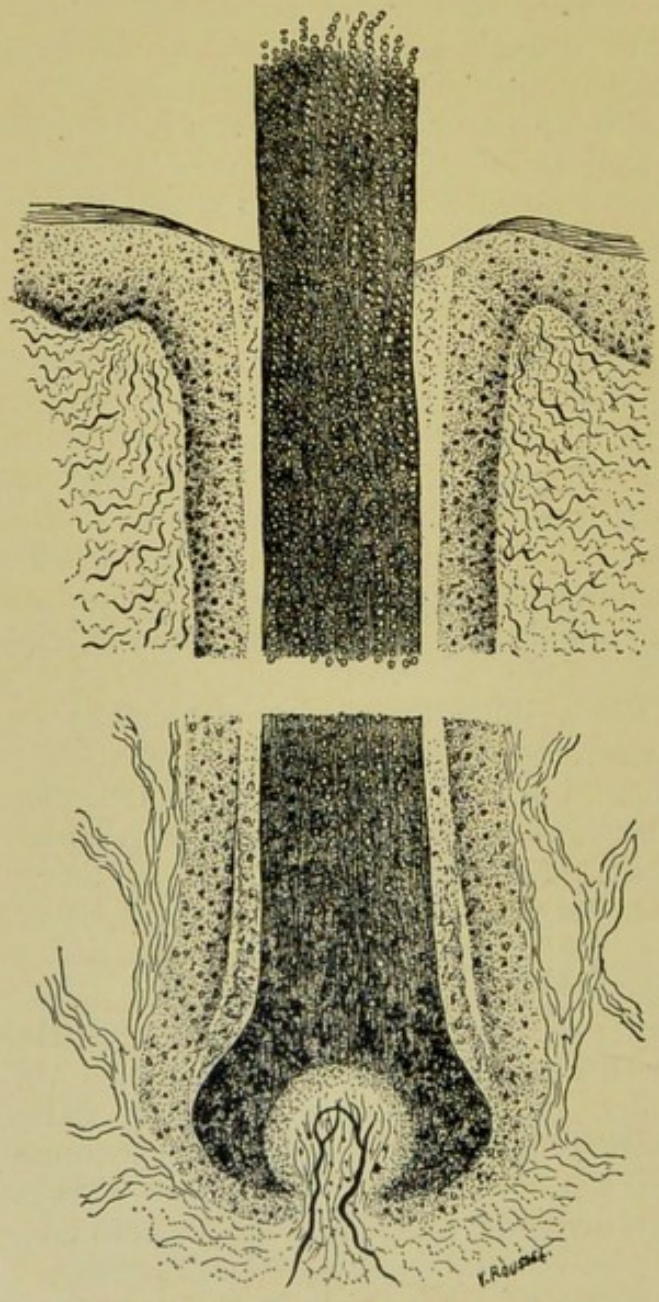


FIG. 159.—Vertical section of hair affected with trichophyton endothrix. (After Sabouraud.)

PARASITOLOGY. — There are two species of trichophyton: (1) the *trichophyton ectothrix*, of animal origin; (2) the *trichophyton endothrix*, of human origin. Sabouraud gave the name *ectothrix* to the former, because it is situated between the hair and its sheath; *endothrix* to the latter, because it is situated within the hair itself. But this distinction was already made by Grüby in 1842.

Trichophyton endothrix. —This is the cause of many ringworms of the scalp, and also of ringworm of the body (erythema or herpes circinata). To examine the parasite, the hair should be soaked in liquor potassæ, which destroys the animal matter but respects vegetable matter and spores. The parasite consists of regular mycelial filaments formed by mycelial spores, 5 or

6 micromillimetres in diameter. The spores are colourless, highly refracting, with a double outline, and are arranged in chains which follow the direction of the hair and bifurcate at intervals. This invasion of the hair by spores explains its brittleness.

When the hair is left for some time in liquor potassæ, the mycelial filaments are dissolved, but the spores remain. Under a magnifying power of 1000 diameters, the double envelope of the spores is seen, and also their mode of articulation.

The trichophyton endothrix forms a characteristic crateriform culture of a brown colour, on the following medium (Sabouraud):—

Agar-agar	1·3 parts
Peptone	0·5 „
Maltose	3·8 „
Water	100 „

Trichophyton ectothrix.—This is observed in some cases of ringworm (*kerion*) which are of animal origin. It is also the cause of all trichophytosis of the beard (parasitic sycosis), and of suppurative cutaneous trichophytosis. The parasite, instead of being situated within the hair, forms a sheath round its root. Most of the filaments adhere to the hair; but, according to Sabouraud, some of them penetrate the epidermis of the follicle and even the superficial layers of the hair. At the height of its virulence this parasite is *pyogenic* by itself.

The trichophyton ectothrix includes several species, each of which produces a special lesion in man. The trichophyton of the cat produces pustular cutaneous trichophytosis; this variety forms a snow-white culture with a downy centre and peripheral rays. The trichophyton of the horse causes sycosis, and sometimes kerion in children, and even in adults; its culture resembles that of the preceding variety, but is salmon-coloured instead of white. There is another rare species of equine origin, which forms a yellowish brown culture; this produces trichophytosis of the beard in the form of disseminated moist dermitis. Lastly, there is a variety of avine origin, which forms a red culture; this causes dry trichophytosis of the beard in the form of ichthyosis pilaris.

To resume, ringworm of the scalp and ringworm of the skin may be caused by the *trichophyton endothrix* or *ectothrix;* ringworm of the beard is caused by the *trichophyton ectothrix.*

That each of these trichophytons has its own individuality, is proved by the fact that it is not associated with another species of trichophyton in the same lesion, that it always forms the same culture, and reproduces the same form when inoculated. Lastly, in cases of contagion occurring in families and schools, the same form of trichophyton is always reproduced.

Etiology.—All forms of trichophytosis are caused by contagion, which may be due to the use of hair-brushes, combs, caps, etc., which have been used by infected subjects. Contagion is also favoured by excoriations of the skin, which facilitate the entrance of the parasite; this occurs in ringworm of the body and beard.

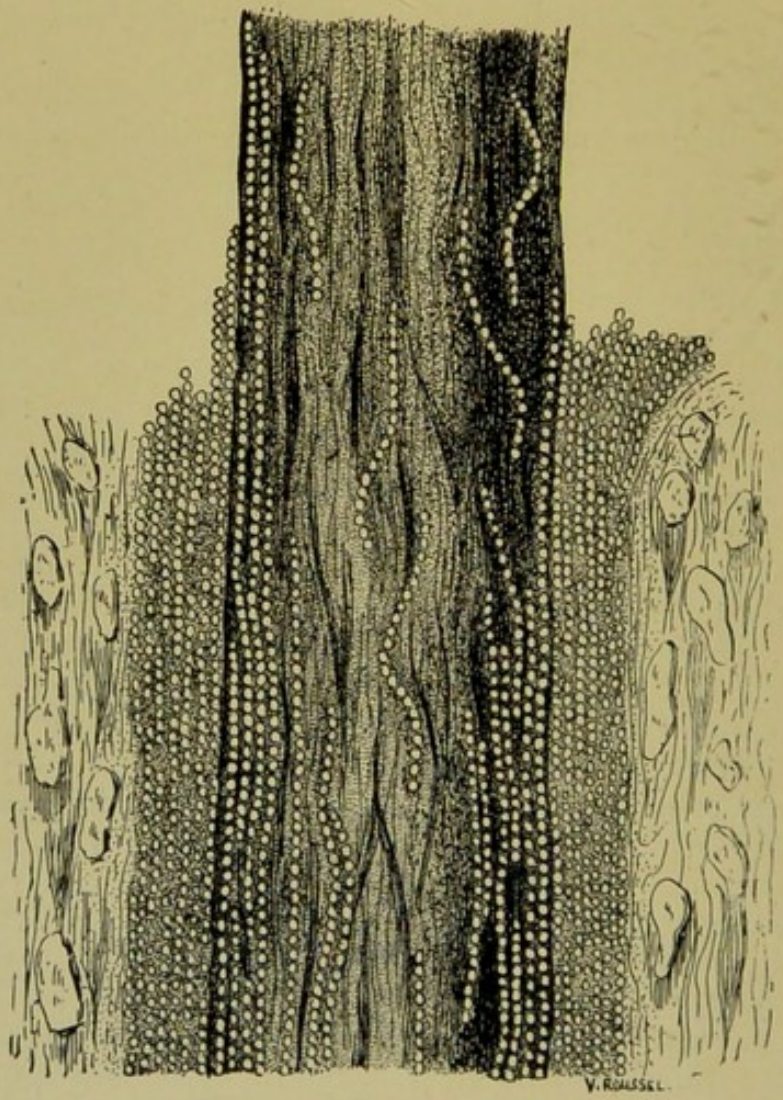


Fig. 160.—Trichophyton ectothrix round a hair from the beard. (After Sabouraud.)

The affection is common where many individuals live together, such as schools and large families. In the same family the children may be affected with ringworm, and the parents with tinea circinata. Contagion may also be transmitted from animals to man (horse, cat, calf, fowl, pigeon). But it is not improbable that trichophytosis may manifest itself otherwise than by contagion, for, according to Sabouraud, it appears that the trichophyton may have an independent saprophytic existence; in fact, this observer succeeded in cultivating

the fungus on natural media, such as vegetable mould, soil, decayed wood, and grain of all kinds.

Age is a predisposing cause in trichophytosis of the scalp, which is observed almost exclusively in children under fifteen years of age; it is seldom seen after twenty. Trichophytosis of the skin occurs both in children and adults, but more frequently in the former. Trichophytosis of the beard (sycosis) occurs exclusively in men.

The constitution of the patient plays a certain part with regard to the duration of the disease, ringworm being more rebellious in feeble and lymphatic children than in the robust.

Concerning the mode of penetration of the trichophyton endothrix into the hair, Balzer has shown that the parasite descends along the hair to the bottom of the hair follicle, and invades the hair from below.

Trichophytosis of the Scalp, or Ringworm.

This is not produced by a single parasite, as was formerly supposed, but by three. We have described the *trichophyton endothrix* and *ectothrix*, each of which may cause ringworm. Sabouraud gave these the name of *megalosporon*, to distinguish them from a third parasite with small spores, called the *microsporon*. According to Sabouraud, this *microsporon* is not a trichophyton but a parasite belonging to quite a distinct species, the *Microsporon Audouini*, discovered by Grüby in 1843.

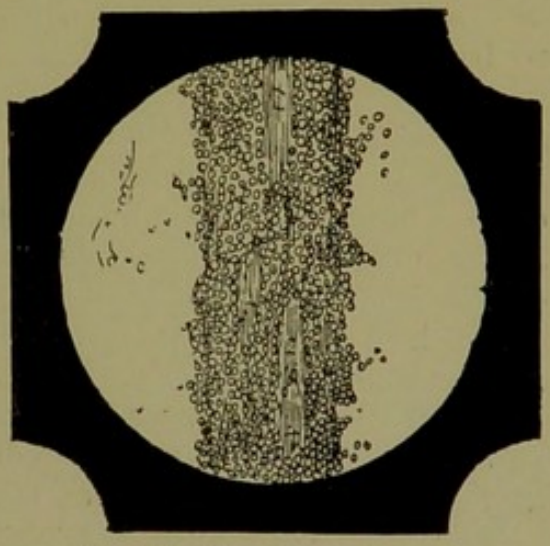


Fig. 161.—Microsporon Audouini. (Besson.)

This parasite consists of very small spores, from 2 to 3 micro-millimetres in diameter, which form a continuous sheath of spores round the root of the hair and for about one-eighth inch above the surface, without any trace of a mycelium. The microsporon forms a grayish white flaxen culture with a downy depression in the centre, very different to the crateriform culture of trichophyton endothrix. This parasite, which only affects children, also occurs in the horse during the first few years (Sabouraud). It causes the "contagious herpes" of colts.

To resume, there are three kinds of ringworm:—

(1) The large-spored ringworm, caused by the *trichophyton megalosporon endothrix;*

(2) Kerion, caused by the *trichophyton megalosporon ectothrix;*

(3) The small-spored ringworm, caused by the *Microsporon Audouini.*

Large-spored Ringworm.—This form begins as small patches, sometimes circumscribed, sometimes ill-defined, formed by the coalescence of small erythematous spots or ephemeral vesicles, and covered with fine scales. The patches cause a variable degree of itching. They increase in size and unite with each other to form the principal patch, while other small patches are developed in the neighbourhood.

At its stage of maturity, the affection is characterised by well-defined round or oval patches, slightly infiltrated, from half an inch to an inch or more in diameter, and covered with dry, discoloured, broken hairs and a few squames. Each patch with its broken hairs has a certain resemblance to a gray tonsure (hence the name *tinea tonsurans*). Along with the squames, there are sometimes crusts formed by dried vesicles, and sometimes lesions caused by scratching. As a rule, there are several patches in different degrees of evolution, situated on the nape of the neck, behind the ears, on the temples and on the vertex; but, in the midst of these, there is usually a principal patch more important than the others. If one of the patches is examined with a lens, the diseased hairs are seen to be thick and broken off very short; sometimes they appear as black dots at the orifice of the hair follicles. They are not surrounded by a white sheath, as in small-spored ringworm. If an attempt is made to pull out the diseased hairs by forceps, they break off. Microscopic examination, after treatment with liquor potassæ, shows chains of spores situated within the hair (*trichophyton endothrix*).

A hair affected with trichophyton endothrix shows changes throughout its whole length; the bulb is flattened, the hair is irregular, bent, nodular and sometimes fissured; its cortex is ruptured under the pressure of spores. The broken end is subdivided into several fibrillæ, separated by rows of spores.

Varieties.—Besides this typical form, there is a diffuse form, in which the patches are very small and disseminated. Another form simulates alopecia areata (peladoid, or bald ringworm); the hairs are broken off level with the scalp and the patches are smooth, resembling those of alopecia areata.

Sometimes ringworm is marked by other eruptions, such as eczema in predisposed subjects, or impetigo due to secondary inoculation. In some cases furuncles are present.

Evolution.—The general health is unaffected as a rule, but when ringworm has lasted a long time there may be some digestive disturbance and anæmia. When left to itself, ringworm is of long duration. At first it sometimes develops rapidly and covers a great

extent of scalp; in other cases, the patches are few in number, circumscribed and slow in growth. The evolution is always irregular, periods of increase alternating with stationary periods, the cause of which is unknown.

After a variable time, but always very long, ringworm may undergo spontaneous cure, especially in adolescents. The new hairs are at first thin and pale, but afterwards regain their normal appearance. There is never alopecia nor cicatrices. Ringworm also takes

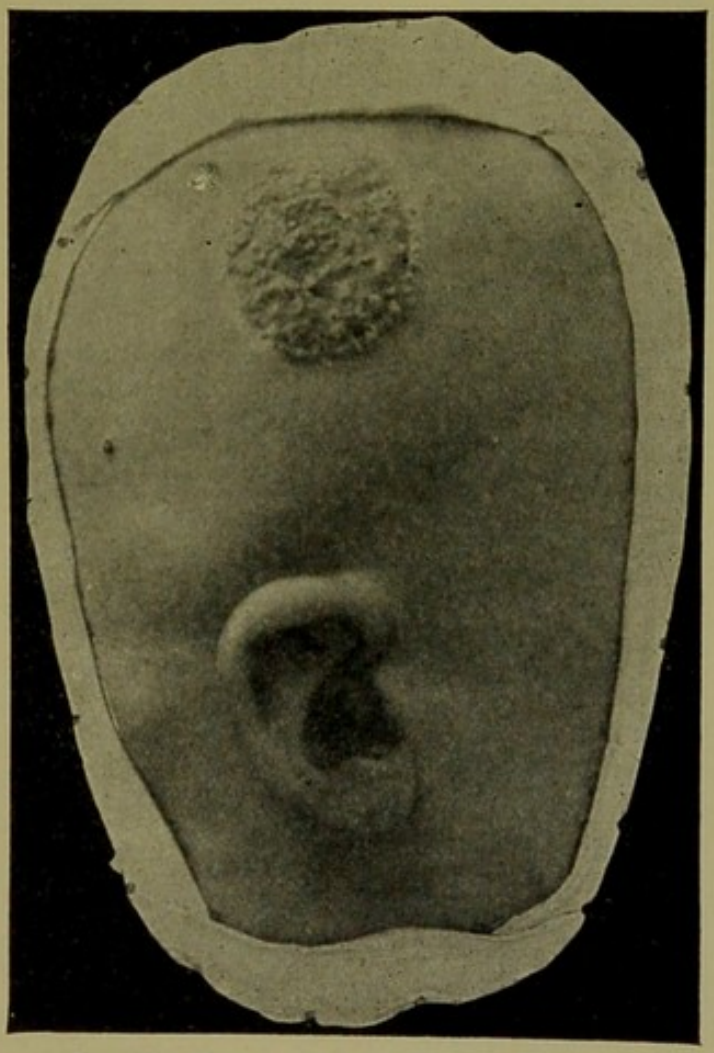


Fig. 162.—Kerion. (St Louis Hospital Museum.)

a long time to cure by the ordinary methods of treatment, the cure being delayed by the appearance of new patches.

Much reserve is necessary in declaring that a case of ringworm is cured, as some hairs may escape the most careful examination and the spores may germinate and produce recurrence. Several examinations at considerable intervals are necessary before giving a certificate of cure.

Kerion.—Kerion is suppurating ringworm. Sometimes the ringworm presents a different evolution to the above, and ends in

cicatrices and patches of permanent alopecia. In this form the patches are more raised and more infiltrated than in the usual cases. The lesion is accompanied by inflammation of the hair follicles, which suppurate and are finally destroyed, causing cicatrices and permanent alopecia.

Kerion, as already mentioned, is produced by the *trichophyton ectothrix* of equine origin (Sabouraud).

Small-spored Ringworm.—This form of ringworm presents peculiarities which require careful consideration, on account of its frequency and severity. The hairs on the diseased patches are at first coated, for about an eighth of an inch above the scalp, with a grayish white sheath formed by the fungus. Later on, they break off at different levels, and each patch becomes covered with white debris full of spores (parasitic pityriasis alba). In this form of ringworm the diseased hairs are very close together, contrary to the large-spored ringworm, in which they are further apart, so that it is difficult to extract one without several others at the same time. After a time, all the diseased hairs fall out; they are replaced by thin, scanty hairs, which after some months become thicker and more numerous.

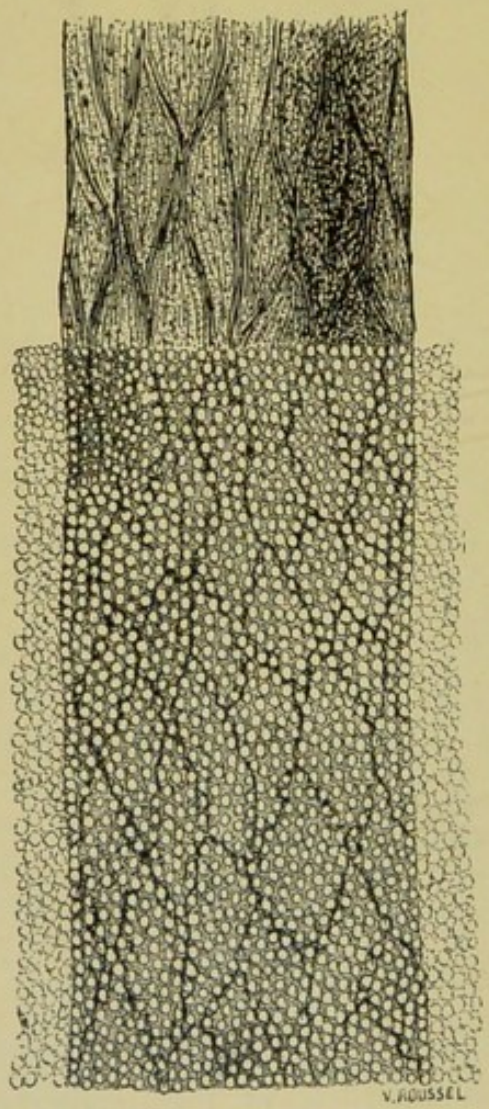


FIG. 168.—Hair from small-spored ringworm. (After Sabouraud.)

Small-spored ringworm appears to be more common than the large-spored variety. It is also the most contagious and the most rebellious form of ringworm, and may last for years. Sabouraud thought that it was never accompanied by ringworm of the skin, but Béclère has shown that this statement is too absolute, and I have myself seen several cases in which the two lesions coexisted.

DIAGNOSIS OF RINGWORM.—This depends on the presence of gray patches, covered with fine squames and broken hairs, in which spores are found on microscopical examination. However, at first sight ringworm may be mistaken for *eczema* or *pityriasis;* but in these two affections the lesions are diffuse; there are no circumscribed

patches and no broken hairs; if there is alopecia, it is diffuse and not circumscribed; lastly, the hairs do not contain spores. In *seborrhœic eczema*, there are circinate patches, but they are covered with fatty squames. In *psoriasis* the squames are thick and pearly white, and there are no changes in the hairs.

Favus differs from ringworm in the presence of the characteristic sulphur-yellow cups, underneath which are red cicatrices, which are never seen in ringworm; in the absence of broken hairs, the latter coming away entire when they fall; and in the peculiar mouse-like odour. Microscopic examination shows the spores of favus, which are irregular and different to those of ringworm.

The patches of *alopecia areata* differ from those of ringworm in being quite smooth, like ivory. The form of alopecia areata with fragile hairs may be mistaken for ringworm, but the diagnosis is decided by microscopic examination.

As regards the *distinction between large-spored and small-spored ringworm*, in the former the hairs are thick, dark, broken short and not ensheathed; in the latter they are long, fine, gray and surrounded at the base by a white sheath. Microscopic examination shows chains of spores within the hairs in the large-spored ringworm; while in small-spored ringworm they form a sheath round the hair. It is true that in ringworm due to the *trichophyton ectothrix*, the spores are outside the hair, but they do not form a sheath round the hairs, only a small collarette.

As in favus, ringworm can also be diagnosed by the *serum reaction* (Abrami), apart from microscopic examination. Whatever the variety of ringworm, the patient's serum gives a positive reaction of fixation with any one of the parasitic fungi which cause human ringworm; microsporon, trichophyton ectothrix or endothrix. The reaction of fixation thus establishes the general diagnosis of ringworm.

The reaction of *agglutination*, which is difficult to manage on account of the number of pathogenic varieties, at any rate shows whether it is a case of large-spored or small-spored ringworm. For this purpose, the agglutinating action of the patient's serum is determined for cultures of the microsporon, trichophyton endothrix and ectothrix. An emulsion of spores is used, obtained by filtering cultures made on bouillon, previously ground in a mortar. The highest value of the serum reaction indicates the variety of parasite in the patient.

TREATMENT OF RINGWORM.—This comprises prophylaxis, local treatment and general treatment.

Prophylaxis.—To prevent contagion, children affected with ringworm should be isolated. Auto-inoculation should also be prevented by frequent washing of the head and antiseptic lotions. A close-fitting cap should be worn to prevent dissemination of spores.

Local treatment.—First of all, *epilation* should be performed, after cutting the hair quite short. It is a good plan to paint the head with tincture of iodine, which shows up the smaller areas of infection which may escape the eye, and reveals the extent of the lesions. This should be done once a week.

Epilation should be performed with flat-bladed forceps. It is necessarily incomplete, on account of the hairs breaking off; but although it is impossible to extract all the diseased hairs, as many as possible should be removed, as they represent so many foci of infection. The hairs should be epilated for about half an inch round all the diseased patches. Epilation, which causes only slight redness and smarting, should be repeated several times. At the end of a month, when the hair is beginning to regrow, they should be examined for any broken hairs, and if these are present, epilation must be repeated.

After each epilation, the scalp should be treated with the following lotion (Quinquaud):—

Biniodide of mercury	½ part
Bichloride of mercury	1 „
Alcohol	40 parts
Distilled water	250 „

Epilation by the X-rays.—The treatment of ringworm by the X-rays was first tried by Freund and Schiff, and afterwards elaborated by Oudin, Gastou, Kienbock, Vieira, Nicolau, Sabouraud, Bissérié and Belot. The X-rays cause depilation of the diseased hairs, which are replaced by healthy ones.

Technique.—The current is obtained either from a static machine or from the electric-light supply, transformed by a Rumkorff's coil. A Chabaud's tube with Villard's osmo-regulator is used, and a spintermeter interposed in the circuit. Hard tubes are employed, equivalent to a spark of 9 or 10 centimetres, emitting penetrating rays which are applied for sittings of seven to fifteen minutes only. On each side of the spintermeter are placed Destot and Williams exciters, by which the tube can be hardened, while heating the osmo-regulator with a Bunsen burner softens it.

The operator is protected from the danger of radiodermatitis by surrounding the tube with an iron sheath, with a wide orifice on which metal tubes can be fixed, varying in diameter according to the size of the diseased surface, but all of the same length; the latter being calculated so that the diseased surface is always 15 centimetres (6 ins.) from the anode (Fig. 164).

The quantity of rays produced by the tube in a given time is measured by means of Holzknecht's pastilles. One of these is placed in the course of the rays at the same distance from the tube

as the patient's head, and as soon as the colour corresponds to 5 units H on the scale, the application must be stopped.

Another method of measuring the quantity of rays is by Sabouraud and Noiré's radiometer. This is based on the fact that an emulsion of platino-cyanide of barium in amyl-acetate collodion changes colour under the action of the X-rays, proportionally to the quantity of rays which it receives. Sabouraud and Noiré have made a standard water-colour tint, corresponding to that of paper painted with the platino-cyanide of barium, when the exposure to the X-rays has been sufficient to cause total depilation of an area of the scalp without producing radiodermatitis. This is called the tint B of the radiometer, and corresponds to five of Holzknecht's units

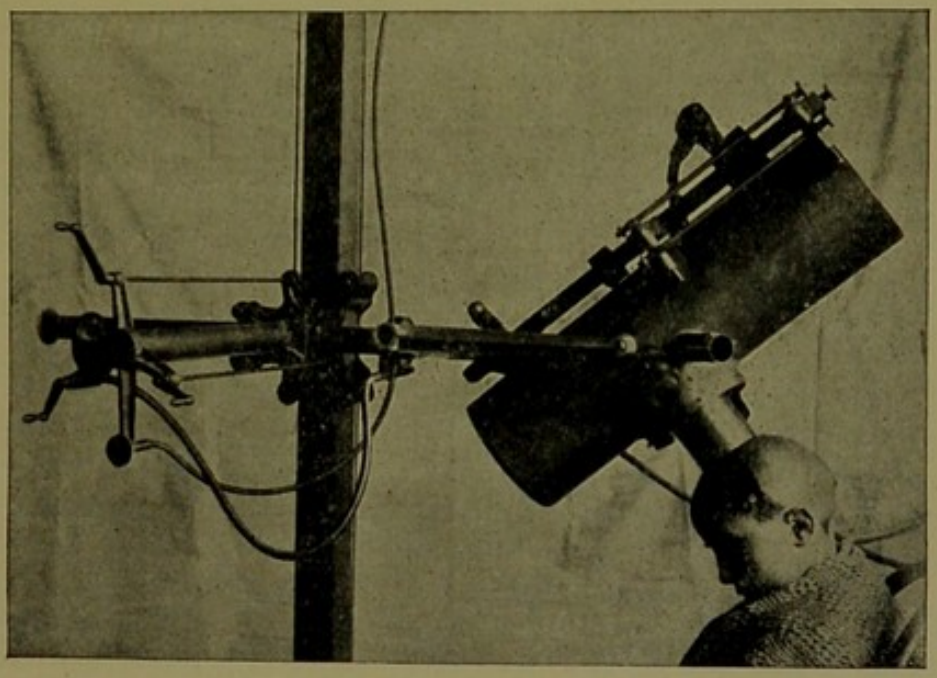


FIG. 164.—Apparatus for epilation by X-rays.

(5 units H). According to Sabouraud, this reaction fades rapidly in daylight, so that the sensitive paper should be placed in a cover of black paper. Moreover, when comparing the tint of the paper with that of tint B of the radiometer, this must be done rapidly, for in a few minutes the colour disappears.

Another disadvantage of this radiometer is that the platino-cyanide paper is less sensitive to the X-rays than Holzknecht's pastilles. While the latter is placed at the same distance from the anticathode as the part to be treated (15 centimetres), the platino-cyanide paper requires to be placed at a distance of 8 centimetres from the anticathode.

Again, according to Sabouraud, the sensitive paper should be placed, during the exposure, on a metal surface such as iron, which

is impermeable to the X-rays, and not on an absorbent surface such as aluminium; otherwise the change of colour will be less marked than it should be for the quantity of rays received.

So long as the paper, placed at a distance of 8 centimetres from the anticathode, has not reached the tint B of the radiometer, there is no danger. If the time of exposure has been long, this indicates that the source of the rays is weak. As soon as the tint B is exceeded, radiodermatitis is inevitable.

In the treatment of a case of ringworm by the X-rays, the hair should first be cut short and the diseased areas made evident by the application of tincture of iodine. The patches are then treated one after another, each patch being exposed through one of the tubes previously mentioned (Fig. 164). If there are more than five patches, it is best to depilate the whole scalp; this involves the exposure of about a dozen different areas one at a time, each area being protected by a sheet of lead after it has been treated. It is better to use exposure tubes with rectangular openings instead of circular, as with the latter large surfaces cannot be treated without leaving diseased areas between the circles, which are difficult to treat afterwards, or without overlapping of the borders of the circles, which may cause permanent alopecia. It is necessary to bear in mind that two exposures of the same area may cause incurable alopecia.

To resume, in order to cure a patch of ringworm by the X-rays, it must be placed at a distance of 15 centimetres from the anticathode, and a pastille of platino-cyanide paper must be placed at a distance of 8 centimetres from the anticathode. When this pastille has taken the tint B of the radiometer, the operation is finished. When a patch has been treated in this way, a slight erythema appears about the seventh day, which disappears in four days and leaves slight pigmentation. In fifteen days, the hairs begin to fall out, and depilation is complete in a few days more. But it must be well understood that the X-rays do not kill the trichophyton, as can be shown by making cultures from the hairs. For this reason, tincture of iodine diluted with alcohol (10 per 100) should be applied to the scalp every day, to prevent inoculation of the healthy parts.

The hairs regrow shortly; they begin to appear at the end of two months, and the new growth is complete at the end of three months. Thus epilation, which takes about two years by the usual method, is completed in three months by radiotherapy.

However, it is necessary to point out that epilation by the X-rays is not without danger, and requires very careful manipulation. I have seen three cases, treated by an expert, in which the X-rays caused *incurable cicatricial alopecia*, with atrophy of the skin and telangiectases. The cutaneous reaction is not the same

in all individuals; in the three cases mentioned, the quantity of rays absorbed at each sitting was not greater than that which had been safely used for other children of the same age.

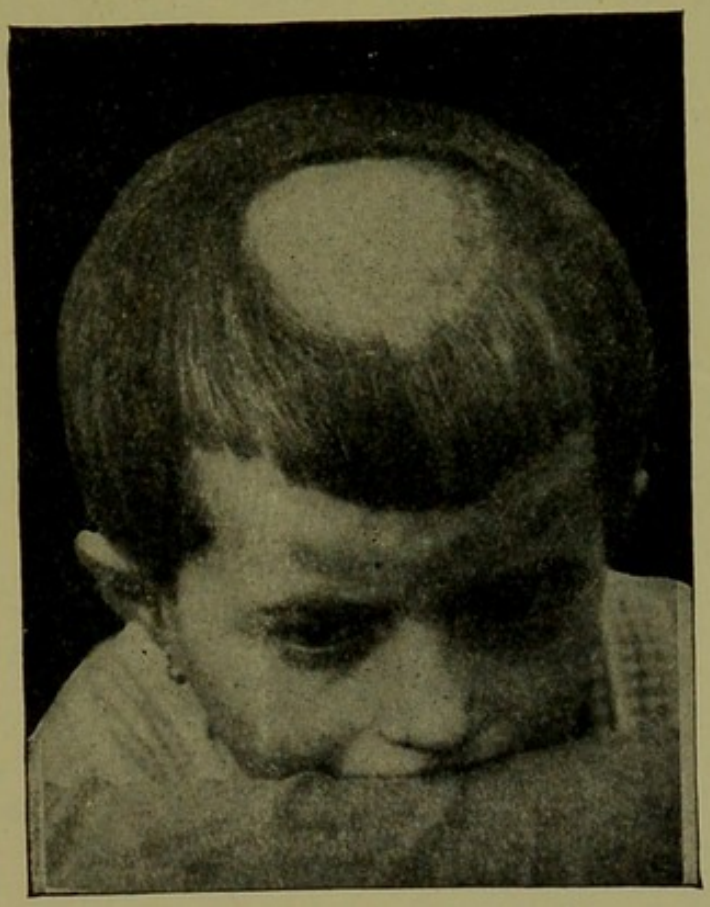


Fig. 165.—Ringworm after depilation by X-rays.

The second indication in the treatment of ringworm, after epilation, is the application of parasitic plasters to the diseased areas, to prevent dissemination of the spores. The following is Quinquaud's formula:—

Biniodide of mercury	½ part
Bichloride of mercury	1 „
Simple plaster	250 parts

This is applied after treating the scalp with Quinquaud's antiseptic lotion (p. 388) or liquor hydrarg. perchloride, and renewed after two days.

When there are only a few isolated diseased hairs, these may be destroyed separately by electrolysis (Wickham and Béclère). But electrolysis should not be used when there are many diseased hairs, on account of the alopecia produced; for it must be remembered that ringworm undergoes spontaneous cure, it is true after a long time, without leaving cicatrices or alopecia.

If eczematous or impetiginous eruptions occur during treatment, they should be treated by moist dressings or starch poultices; after which the treatment can be resumed.

In *kerion*, the diseased hairs should be removed by epilation and the inflammation treated with boiled-water dressings; after this, tincture of iodine should be applied every third day.

General treatment.—Weakly children require tonics such as cod-liver oil. A bad state of general health renders the scalp, to a certain extent, a favourable soil for the development of the parasite.

Trichophytosis of the Skin.

Trichophytic Erythema, or Herpes Circinata—Tinea Circinata.—This begins in the form of a small, red, slightly raised spot, which soon becomes squamous and causes itching. The spot extends peripherally and always retains a circular form, whatever its dimensions.

At its period of maturity, the lesion varies in size from a quarter of an inch to an inch or more in diameter. The centre is yellow and covered with fine squames; in old patches it sometimes appears normal. The edges of the patch are slightly raised. When the patches extend rapidly, they sometimes form incomplete rings, which may intersect each other and form a single polycyclic patch (serpiginous trichophytosis). In other cases, the rings are concentric, owing to the development of new parasitic rings in the centre of the primary lesion by auto-inoculation.

When the inflammation is more intense, the border of the patch is covered with vesicles which dry up and form scales; a new ring of vesicles may then develop round the first (*herpes circinata*). Sometimes there are actual bullæ, which may render the diagnosis difficult.

In other cases, the redness and infiltration are more intense, and pustules develop in the centre and on the borders of the patch. This form resembles sycosis, but differs from it in its localisation in the epidermis and its situation on the smooth skin. According to Sabouraud, this form is due to the *Trichophyton ectothrix pyogenes*, a parasite of the cat.

Situation.—Cutaneous trichophytosis occurs more commonly on the exposed parts of the body: the face, neck, hands, forearms, and especially on the back of the wrist; but it may occur on any part of the body, especially in the groins, where it may be mistaken for *erythrasma*. In children, it often coexists with ringworm of the scalp; sometimes a patch is found situated partly on the skin and partly on the scalp, forming a combined lesion of ringworm and tinea circinata. It sometimes occurs on the palms and soles, where

it has a peculiar appearance due to the thickness of the skin of these regions, and resembles the *psoriasiform syphilide.*

Microscopic examination.—If the squames are treated with liquor potassæ and stained with eosine or methyl violet, the mycelium and spores can be seen. Microscopic examination shows the presence of the *Trichophyton megalosporon endothrix*, in the case of children affected at the same time with ringworm of the scalp, and in adults infected from them. In other cases, a trichophyton of animal origin forming white cultures is found (Sabouraud).

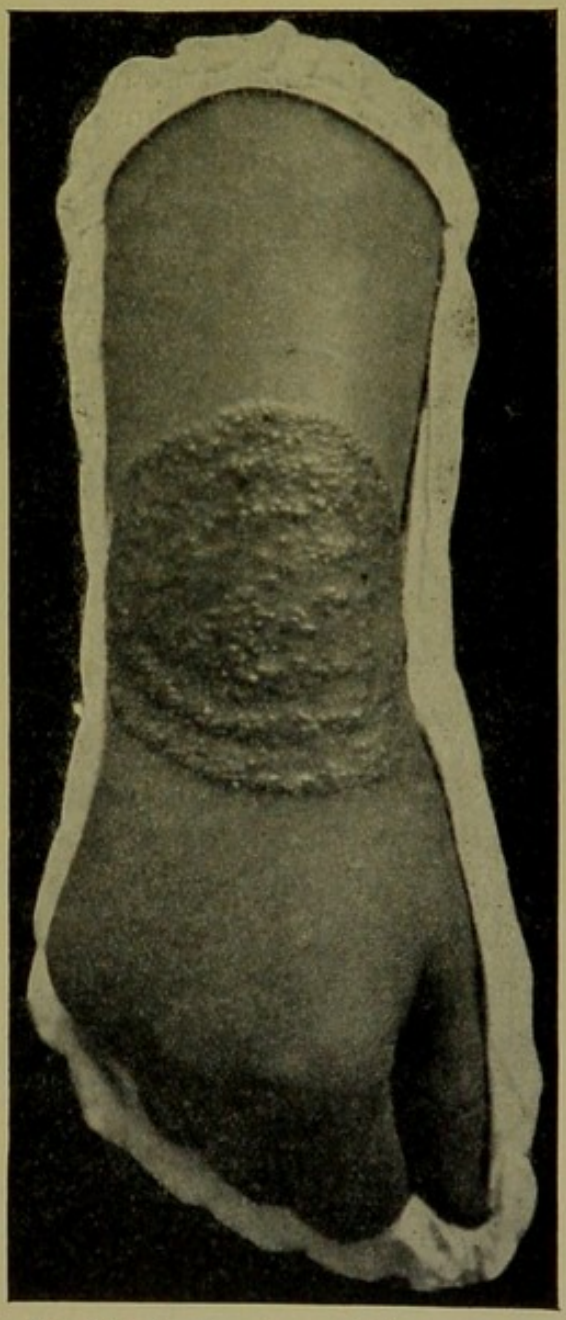


Fig. 166.—Erythemato-vesicular cutaneous trichophytosis. (St Louis Hospital Museum.)

Evolution. — When left to itself, the affection is of long duration; the patches extend indefinitely, and new patches develop. It may, however, undergo spontaneous cure, like ringworm, after a variable time.

The affection itself is not of great importance, but it is contagious and may give rise to ringworm of the scalp.

Diagnosis. — In *erythema marginata*, the colour is brighter and the edges more raised; the patches are very slightly squamous and do not heal in the centre; in doubtful cases, microscopic examination must be made. *Pityriasis rosea* is a generalised erythemato-squamous eruption; the patches sometimes form rings, but these have no tendency to extend; in pityriasis rosea, microscopic examination shows the absence of spores. *Seborrhœic eczema* and discrete patches of *eczema* are also distinguished by microscopic examination. The *circinate syphilide* causes no itching, has a characteristic coppery colour, and is usually situated on the trunk. *Lupus erythematosus* develops more slowly, and after a time a cicatricial patch appears in the centre.

In cutaneous trichophytosis, *serum diagnosis* (reaction of fixation and agglutinating reaction) is facilitated by the fact that the pathogenic fungi in question consist of two types; the *trichophyton endothrix* and the *trichophyton ectothrix*. It is sufficient, therefore, to test the patient's serum with cultures of these two varieties.

Treatment.—The best form of treatment is the application of tincture of iodine for several consecutive days; the skin desquamates and gradually resumes its normal appearance. Tincture of iodine has the disadvantage of staining the skin brown, but the stains can be partly removed by solutions of iodide of potassium or hyposulphite of soda. Instead of tincture of iodine, calomel or turpeth ointments may be used. When vesicles are present, irritating applications should be avoided, as they increase the cutaneous inflammation.

Agminated Folliculitis and Perifolliculitis. Suppurative Trichophytosis of the Skin.

This affection, first described by Leloir and Duclaux, is characterised by a raised patch, usually situated on the back of the hand or forearm, but sometimes on other parts of the body. The lesion is bluish red in colour, and its surface is studded with holes, and small yellow purulent spots which later on become holes when the pus has been discharged. On pressing the whole patch, pus exudes from the holes, and also white plugs formed by dried pus.

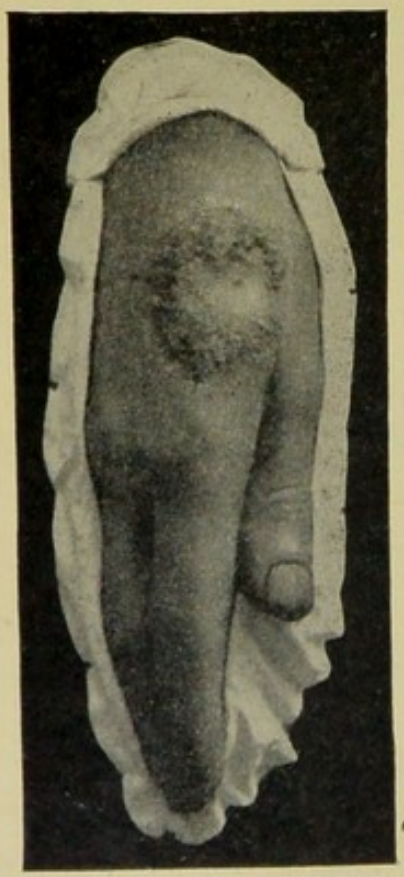


FIG. 167.—Suppurative cutaneous trichophytosis. (St Louis Hospital Museum.)

The lesion begins in the form of small red papules, which are soon transformed into agglomerated pustules. It is of limited duration, and tends to disappear in about a fortnight.

Besides this benign form, which has a rapid evolution, there is a subacute form, rebellious and slow in evolution, studied by Quinquaud and Pallier. These observers describe five varieties: (1) a common form which resembles the benign form; (2) a phlegmonous form; (3) a papillomatous form; (4) a pseudo-ulcerative form; (5) a serpiginous form.

Diagnosis.—The papillomatous variety may easily be mistaken for *papillomatous tuberculosis;* but agminated folliculitis is more rapid in development, is more suppurative, and has no tendency to sclerosis and central

cicatrisation. In doubtful cases, bacteriological examination and inoculation of the guinea-pig should be performed.

Pathological anatomy.—The lesion must be regarded as a pilo-sebaceous folliculitis and perifolliculitis. Leloir considered that the affection was not trichophytic, and described micrococci in the blood, but the researches of Sabouraud show that the lesion is caused by the *trichophyton ectothrix*, which is pyogenic by itself. Microscopic examination is often negative, but culture is always definite.

Treatment.—Tincture of iodine should be applied, or, if the lesion is deep and rebellious, the galvano-cautery.

Trichophytosis of the Beard.

This includes two forms, one affecting the epidermis only, the other invading the hair follicles. The former does not differ from trichophytosis of the smooth skin; the second form includes: (1) *trichophytic sycosis*, or *mentagra;* (2) *trichophytosis* in the form of disseminated moist dermitis; (3) a new dry *trichophytosis* in the form of ichthyosis pilaris (Sabouraud). The most important of these is *sycosis*, and the only one we shall describe here.

Sycosis, or Mentagra.—This appears at first in the form of a red, slightly squamous patch, on which the hairs are covered with a kind of rime, formed of a mass of spores (parasitic pityriasis alba). At this stage, the lesion is still superficial and epidermic, but the hairs are soon invaded by the parasite in the same way as in ringworm. The hairs are dry, dull, discoloured and brittle. The hair-follicles soon become inflamed, the skin is red and swollen, and pustules develop around the hairs.

After a time, the lesions consist of a mixture of pustules and hard nodules of various sizes. Sometimes the inflammation extends more deeply and forms small abscesses. The pustules open on the surface of the skin and discharge a sero-purulent fluid, which forms crusts and glues the hairs together. Underneath the crust are sometimes fungoid ulcerations. Furuncles may be present at the same time, also submaxillary adenitis, which may suppurate. The hair papillæ are often destroyed by the suppuration, leading to the formation of cicatrices and permanent alopecia.

The parasite is rather difficult to find. The best method is to examine a drop of pus from an unbroken pustule between two slides, without staining (Sabouraud).

Sycosis occurs most often on the border of the lower jaw, also on the chin and cheeks. It has been observed on the chest, and even on the pubic and anal regions. It causes itching at first, afterwards more or less burning sensation. There is sometimes fever with gastric disturbance, due to the suppuration.

Sycosis sometimes, but rarely, undergoes spontaneous cure; but the new growth of hair is incomplete, and there are always some bald patches. As a rule, it persists indefinitely if left untreated. It is, therefore, a serious affection, which leaves indelible traces.

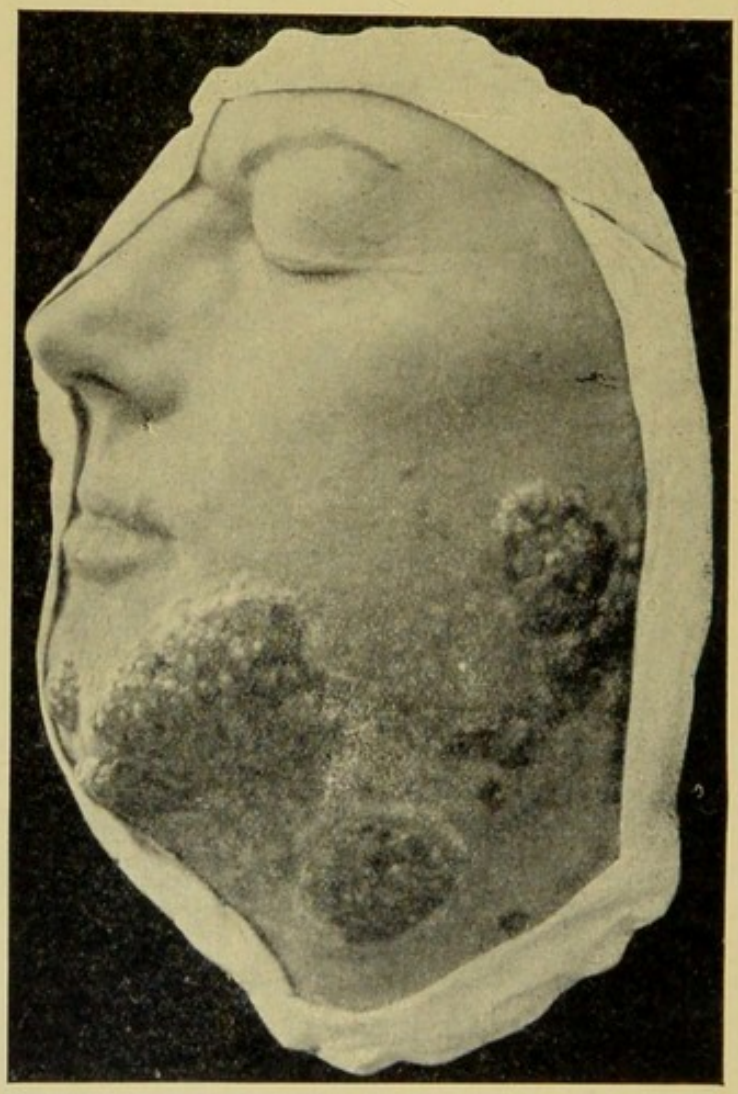


FIG. 168.—Tricophytic sycosis. (St. Louis Hospital Museum.)

Trichophytic sycosis is produced by the *trichophyton ectothrix pyogenes*, a parasite of the horse. The hair is surrounded by the parasite and its sheath crowded with spores.

Diagnosis.—The patient's occupation is an important element in the diagnosis, as sycosis is caused by the trichophyton of the horse. Persons who come in contact with horses are therefore liable to contract this affection.

Sycosis must be distinguished from *eczema*, especially *impetiginous eczema;* the latter is covered with crusts, but it is not limited to the hairs, and is not accompanied by indurated nodules. *Eczema pilaris* occurs on the upper lip in connection with chronic coryza,

and does not present the deep induration and nodules of sycosis. *Acne* differs from sycosis in chiefly affecting the smooth parts. Sycosis is sometimes difficult to distinguish from the *tubercular and pustular syphilide*, but the latter is not limited to the hairy regions; the syphilitic tubercles are hard and copper coloured, and the pustules covered with dry greenish yellow crusts. The diagnosis between parasitic sycosis and *simple non-trichophytic sycosis* is sometimes difficult; in the former, the pustules are larger, the hairs are diseased and come out easily, and sometimes other trichophytic lesions are present; in non-trichophytic sycosis, the pustules are smaller, the hairs are little changed and are still adherent; microscopic examination is sometimes necessary, but difficult to perform.

Treatment.—In the early stage, the application of tincture of iodine is sufficient. When the lesions are deeper, the crusts should first be removed by means of boracic fomentations, or starch poultices prepared with boracic lotion, or a solution of sublimate (1 in 1000); the hair should then be cut short and the diseased hairs epilated at several sittings; after epilation, the part should be treated with boracic or sublimate lotions. Pustules and dermic abscesses should be evacuated, nodules scarified, and fungosities treated with the galvano-cautery; then, after bathing with boracic lotion, an antiseptic ointment of turpeth, yellow oxide of mercury or calomel, should be applied, or, if there is much irritation, simple moist dressings.

Trichophytosis of the Nails.

This occurs in old cases of ringworm, as the result of inoculation under the nail due to scratching the diseased patches. It was formerly observed in epilators, when epilation was performed with the fingers.

The disease generally affects the finger-nails, rarely the toe-nails. The lateral borders of the nail become dull and thickened; its surface becomes irregular and covered with small white or yellow patches, and longitudinally or transversely striated. After a time the nail becomes swollen and brittle. On microscopic examination of the white and yellow patches, the spores and mycelium of the trichophyton can be seen.

Diagnosis is impossible without microscopic examination, if there are no trichophytic lesions present on the skin, scalp or beard. Ungual trichophytosis, in fact, resembles eczema or psoriasis of the nails. Favus of the nails is yellower.

Treatment consists in softening the nail by maceration in an india-rubber finger-stall, or, more rapidly, by painting it with liquor potassæ and then scraping it twice a week with pumice stone;

together with daily applications of tincture of iodine or sublimate solution (1 in 200 or 1 in 100). Sabouraud covers the nail with a piece of wool soaked in a solution of one part of iodine and two parts of iodide of potassium in 1000 parts of distilled water; over this an india-rubber finger-stall is applied.

ERYTHRASMA.

The parasite of this affection was discovered by Burchardt in 1859. Erythrasma occurs in the same situations and has almost the same appearance as *erythema intertrigo*, and is often mistaken for this dermatosis.

It consists of brownish red or yellowish red patches, varying from a five-shilling piece to the palm of the hand in size, with well-defined borders. The surface is rough, and only desquamates after scratching; the squames are always very fine. Around the principal patch are smaller patches, which may unite with it. The patches occur most frequently in the inguino-scrotal or inguino-vulvar folds and on the internal surface of the upper part of the thigh; sometimes in the intergluteal fold, on the thighs, abdomen, antero-lateral region of the chest, in the axillæ and other articular folds. The eruption is not accompanied by itching, so that it is only discovered accidentally. It persists indefinitely if left untreated, and may become inflamed. It is more common in obese and arthritic subjects.

This affection is caused by the *microsporon minutissimum*, the spores of which are situated in the horny layer of the epidermis, and are the smallest of all those which live on the surface of the skin; the mycelium is formed by very fine filaments. The parasite has been both cultivated and inoculated (Köbner, De Michele).

Diagnosis.—Erythrasma differs from *intertrigo* in being always dry; intertrigo is of a brighter red colour and is accompanied by exudation. *Eczema* differs from it in the presence of vesicles and crusts. *Pityriasis versicolor* is characterised by grayish yellow or café-au-lait coloured patches. *Tinea circinata* forms a circular red patch, extending at the periphery, while the centre becomes normal; sometimes vesicles are present on the borders; microscopic examination shows the presence of the *trichophyton*, which is very different to the *microsporon*, a higher power being necessary to examine the latter, while the spores of the trichophyton are easily seen by ordinary powers. The squames can be examined in liquor potassæ, after maceration in ether.

Treatment.—The patches should be treated with soft soap and then painted with tincture of iodine or sublimate solution (1 in 1000

to 1 in 600). When there is much irritation, sulphur or turpeth ointments may be used (10 per 100).

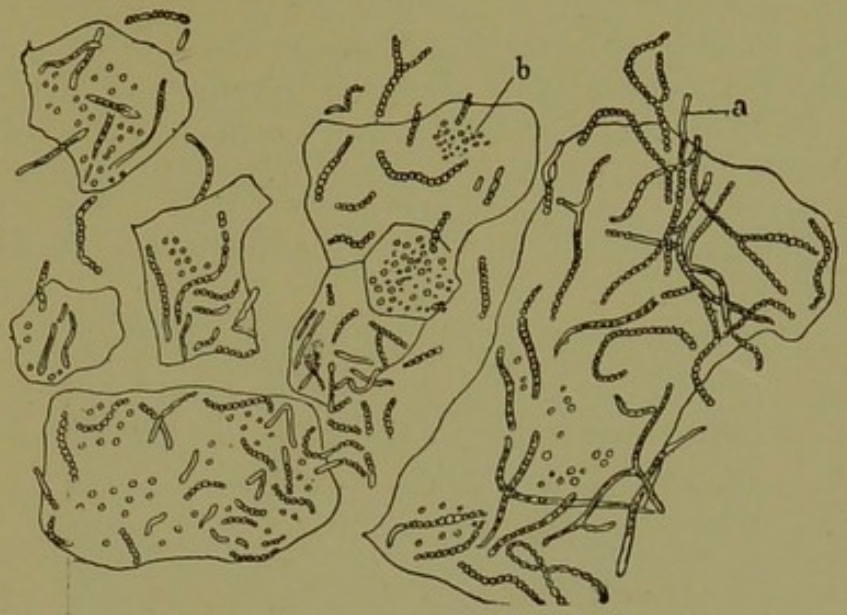


Fig. 169.—Microsporon minutissimum. (Balzer.)
a, Mycelium; *b*, spores.

PITYRIASIS VERSICOLOR.

This affection is caused by the *microsporon furfur* (Eichstedt, 1846), which consists of mycelium and spores. The mycelium is

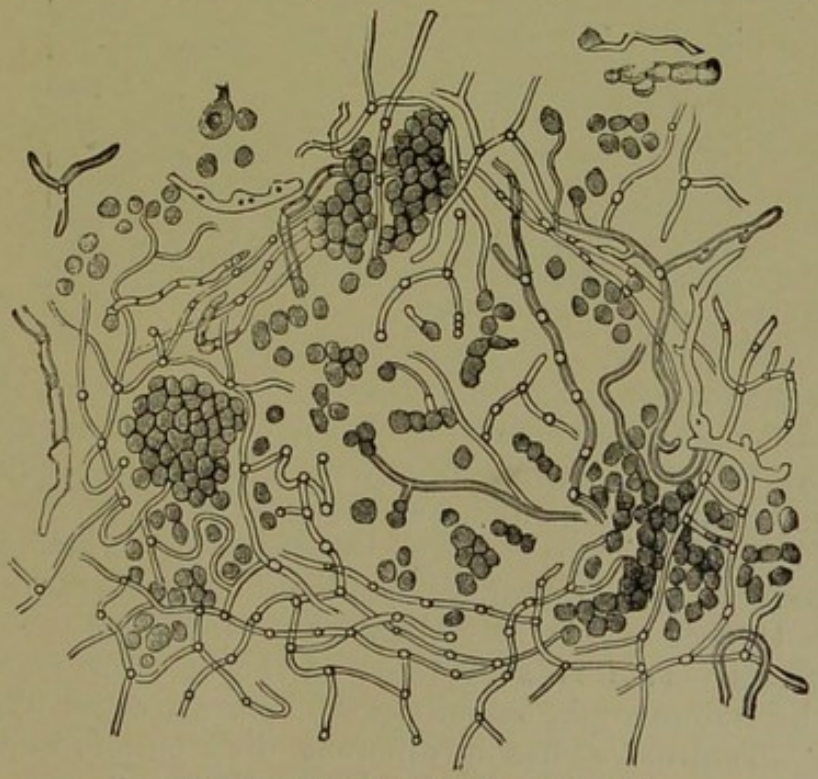


Fig. 170.—Microsporon furfur. (Kaposi.)

formed by numerous thin filaments, 15 to 40 micromillimetres in diameter, interlacing in all directions. The spores are round or oval. 23 to 80 micromillimetres in diameter, highly refractive, and arranged in groups in the meshes of the mycelium. The parasite is situated in the horny layer of the epidermis and never extends deeply. It is contagious from man to man, either directly or indirectly by clothes, especially flannel, but only in predisposed subjects: cachectic, phthisical or arthritic. It is especially common in tuberculous individuals, and in those who belong to tuberculous families.

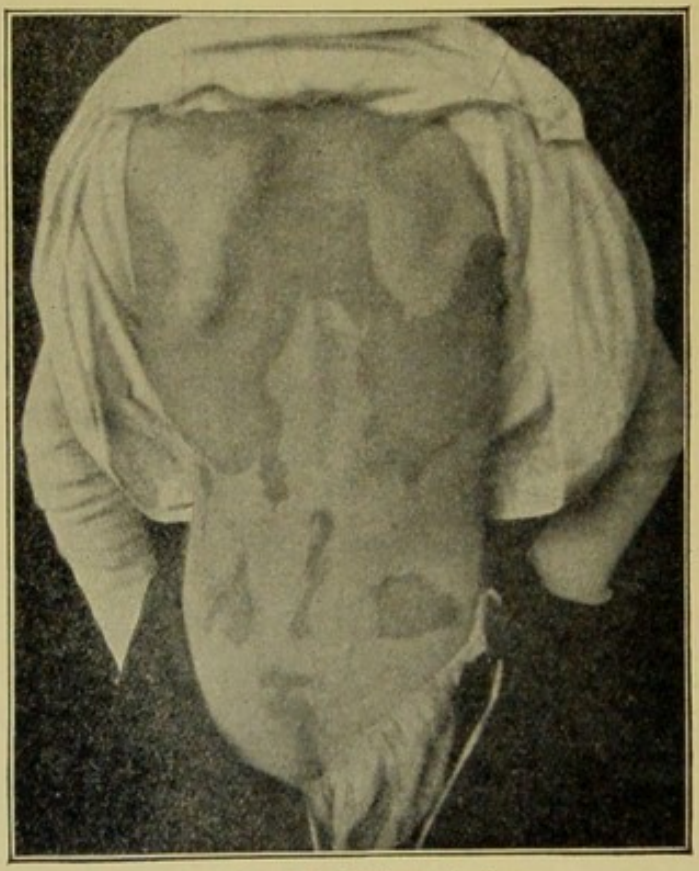


Fig. 171.—Pityriasis versicolor.

Symptomatology.—The affection is characterised by grayish yellow, café-au-lait coloured or brown patches, generally situated on the chest, but sometimes occupying the whole trunk and even the limbs, forming a continuous sheet.

Small squames can be removed by scratching, in which the parasite can be seen by microscopic examination, after soaking in liquor potassæ. Itching is generally absent, but sometimes acute.

Diagnosis.—This is very easy, as the affection can be distinguished by microscopic examination from all other hyperchromic diseases. *Erythrasma* has a different distribution, and is distinguished by microscopic examination. Fournier and Sabouraud

observed pityriasis versicolor in an infant of fifteen days, the lesions being surrounded by a bright red circinate areola, simulating a syphilitic eruption; the diagnosis was made by microscopic examination and by the presence of pityriasis versicolor in the mother.

TREATMENT.—When there are only a few patches, it is sufficient to paint them every day with tincture of iodine, liquor hydrarg. perchloride or oxygenated water. If the eruption is very extensive, a sulphur bath should be taken every day, and the parts rubbed with soft soap. Sulphur, calomel or turpeth ointments may also be used. To avoid reinfection, the patient's linen should be disinfected. The general health also requires attention.

CARATHÈS OR PINTA.

These are dermatoses characterised by the presence of spots of various colours, which disappear and give place to pseudo-vitiliginous areas. The lesions are caused by several fungi of the genus *Aspergillus*. The affection is endemic in all regions which border on the Cordilleras, such as Honduras, Guatemala, Colombia, Venezuela, and Peru.

SYMPTOMATOLOGY.—The spots of carathès have two stages: an active, hyperchromic, or erythematous stage, and a pseudo-vitiliginous stage of retrogression, which remains permanent.

Hyperchromic or erythematous stage.—From inoculations made in man, the incubation period, before the spots appear, seems to be about a month. The eruption is sometimes preceded by pruritus; it generally appears on the face and nape of the neck, then on the forearms, wrists, upper part of the chest, legs and instep. The spots increase in size and coalesce with others which appear at their periphery, forming patches of various shapes several centimetres in diameter.

In Europeans, the spots are erythematous, and soon become covered with fine squames. In coloured people, they are at first yellow, reddish or gray; at the end of about two years they assume their true colour, which is, in order of frequency, yellow, red, blue, black and white. These spots also present furfaceous desquamation. They are sometimes the same colour, sometimes of different shades.

After some years a considerable extent of skin is affected, and the bucco-lingual, balano-preputial and vulvar mucous membranes may be also covered with spots. The pruritus increases, desquamation becomes more profuse, and takes the form of large gray squames. The affected regions undergo hyperkeratinisation, as well as the palms and soles, which also present deep ulcers. The hair and nails are never altered.

Pseudo-vitiliginous stage.—When left to itself, the disease may last indefinitely, but spontaneous cure is possible. The colour of the patches disappears, and is replaced by a pigmented surface, which is only clearly marked in white subjects. The pigmentation then disappears from the centre of the patches towards the periphery, leaving smooth colourless spots.

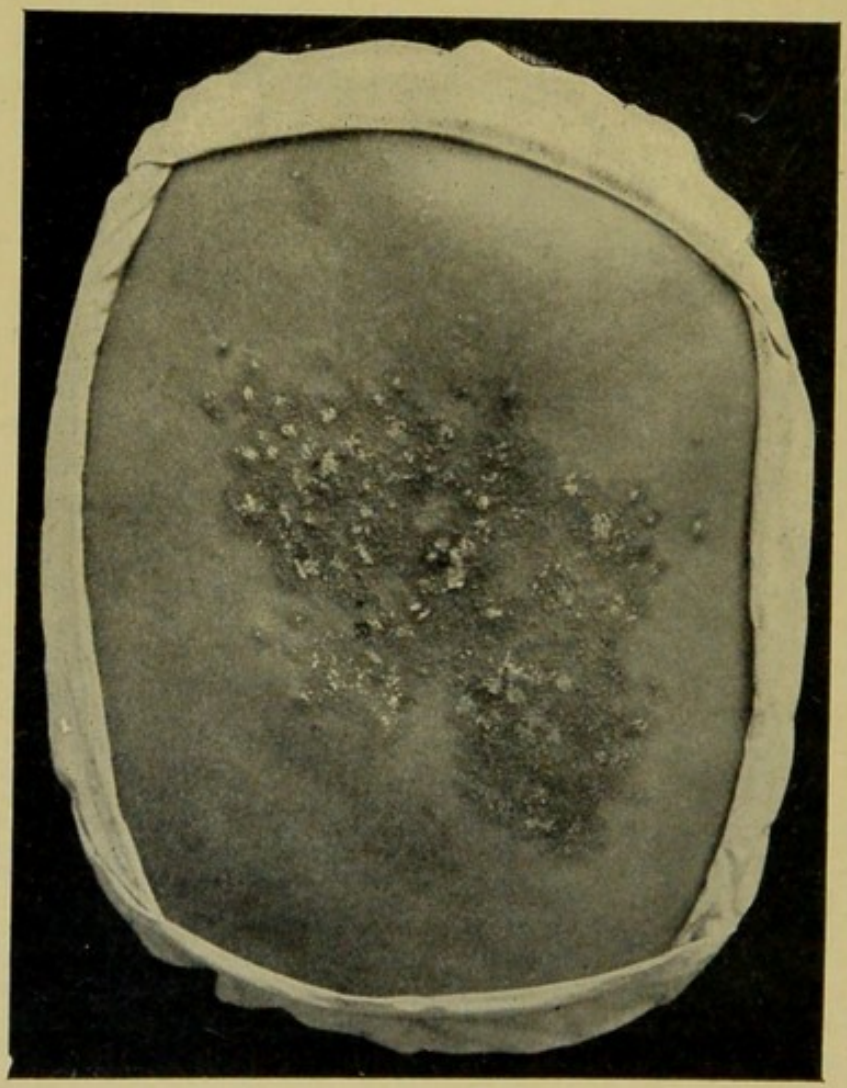


Fig. 172.—Carathès. (St Louis Hospital Museum.)

Etiology.—The disease affects all races, but occurs chiefly among half-castes. Agricultural labourers and miners are most often affected; this is explained by the presence of fungi similar to those of carathès in the grain of cereals and on many vegetables, and also in the water in mines (Montoya y Florez). It is possible that the affection is conveyed by mosquitos and bugs of the genus Acanthia.

The parasites of carathès were discovered by Montoya. They can be seen by examining squames, soaked in liquor potassæ and warmed slightly for a few seconds, in the form of very fine dichotomous

filaments between the epidermic cells. At certain points the filaments form a close reticulum, from which arise larger filaments, composed of two, three, or four fine mycelia; also a short, thick branch which terminates in a piriform enlargement crowned by a row of five or six sterigmata, each bearing from three to five spores.

The fungus can be cultivated on peptonised agar with 4 per cent. glycerine. Each variety of carathès has a special parasite with a special culture (violet, blue, black, white and yellow fungi).

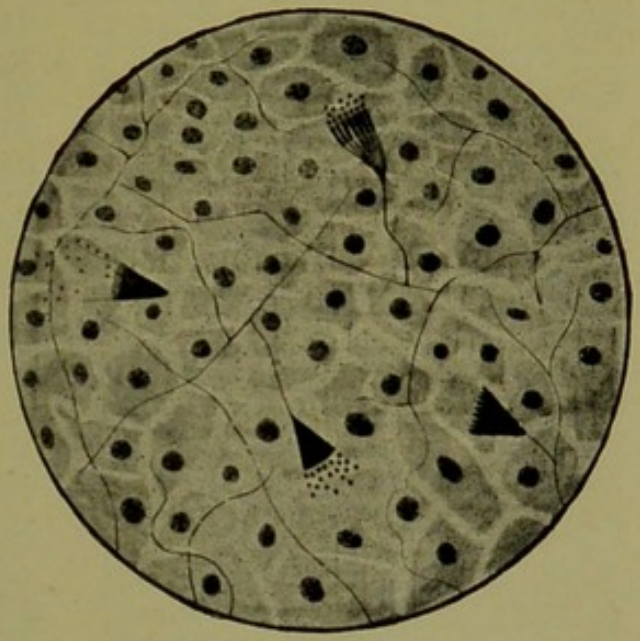


Fig. 173.—Epidermic squame from carathès. (Montoya y Florez.)

Diagnosis. — Carathès may at first be mistaken for *pityriasis versicolor*, but the latter can be recognised by microscopic examination of the squames. The spots of *leprosy* are distinguished from carathès by being anæsthetic. *Vitiligo* consists of symmetrical achromic patches which are never preceded by coloured patches.

Treatment.—Nitrate of mercury ointment, applied twice with an interval of a few days, is sufficient to cure the affection. If inflammatory phenomena arise they must be treated by emollients. This treatment applies to carathès of the face and hands. But when the patches are numerous and cover a large part of the body, the above ointment is dangerous on account of possible mercurial intoxication. In these cases the daily application of tincture of iodine will cure carathès of not more than a year's duration.

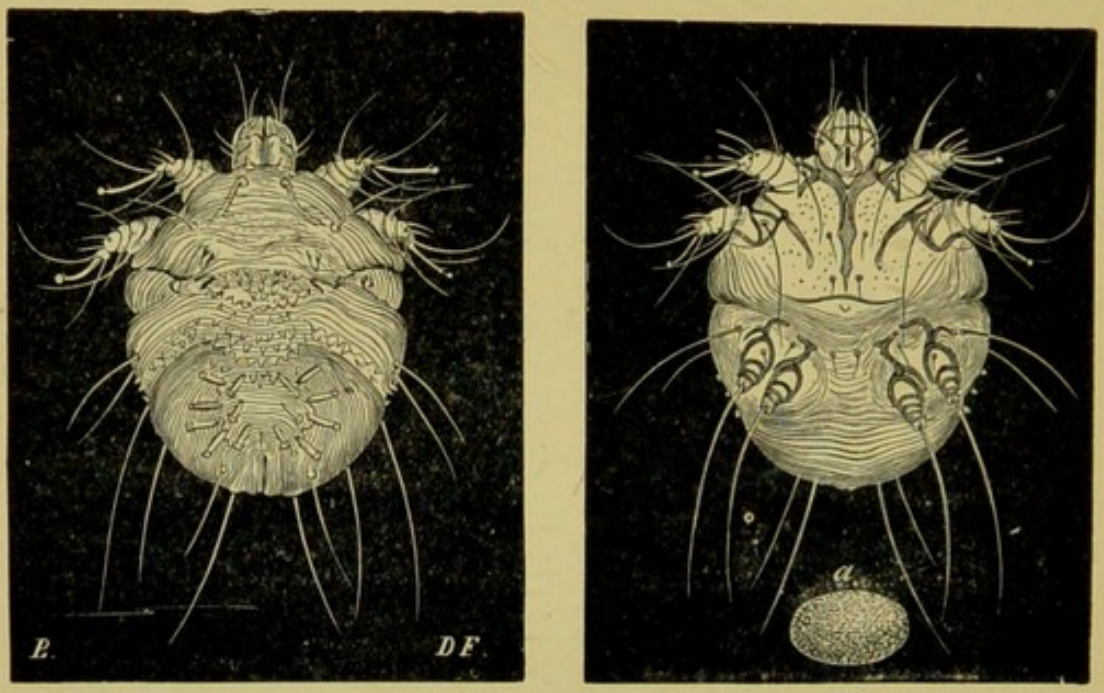
In old cases, a 10 per cent. solution of chrysarobin in chloroform may be applied every four days for a fortnight; then every week for two or three months. Only a few patches should be treated at a time, because of the irritating action of chrysarobin. Lastly, the patient's clothes should be disinfected.

ANIMAL PARASITES.

SCABIES.

Scabies is caused by the presence of the *sarcoptes*, or *acarus scabiei*, in the epidermis. This parasite, belonging to the order of

Arachnida, is white, shining and rounded. The female is larger than the male, 0·33 mm. long and 0·25 mm. wide; the male is



A B

FIGS. 174 and 175.—Acarus scabiei, female.
A, dorsal surface; B, ventral surface; *a*, egg.

0·20 mm. long and 0·16 mm. wide. The female is thus visible to the naked eye. Under the microscope, transverse striations and spines are seen on the dorsal surface; at one end is seen the head with its mandibles, at the other end the anus and genital organs. The ventral surface presents four pairs of appendages; in the female, the two first pairs terminate in suckers, the two last in spines; in the male, the two first and the fourth pairs of appendages terminate in suckers, and the third pair by a long spine. The females are much more numerous than the males; when impregnated, they penetrate the epidermis and form burrows, along which they travel without being able to retreat, on account of their spines. In these burrows, which are situated in the deeper part of the horny layer of the epidermis, the females lay eggs and then die. The eggs hatch rapidly and give rise to larvæ, which become chrysalids and finally adults. The larvæ and chrysalids live on the skin; the impregnated females

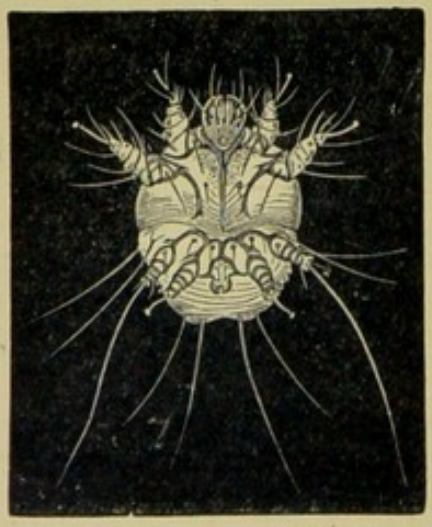


FIG. 176.—Acarus scabiei, male; ventral surface; × 250.

alone penetrate the epidermis. The males live on the skin under the squames; they die soon after they have impregnated the female.

Characters of the burrows.—The essential lesion of scabies is the

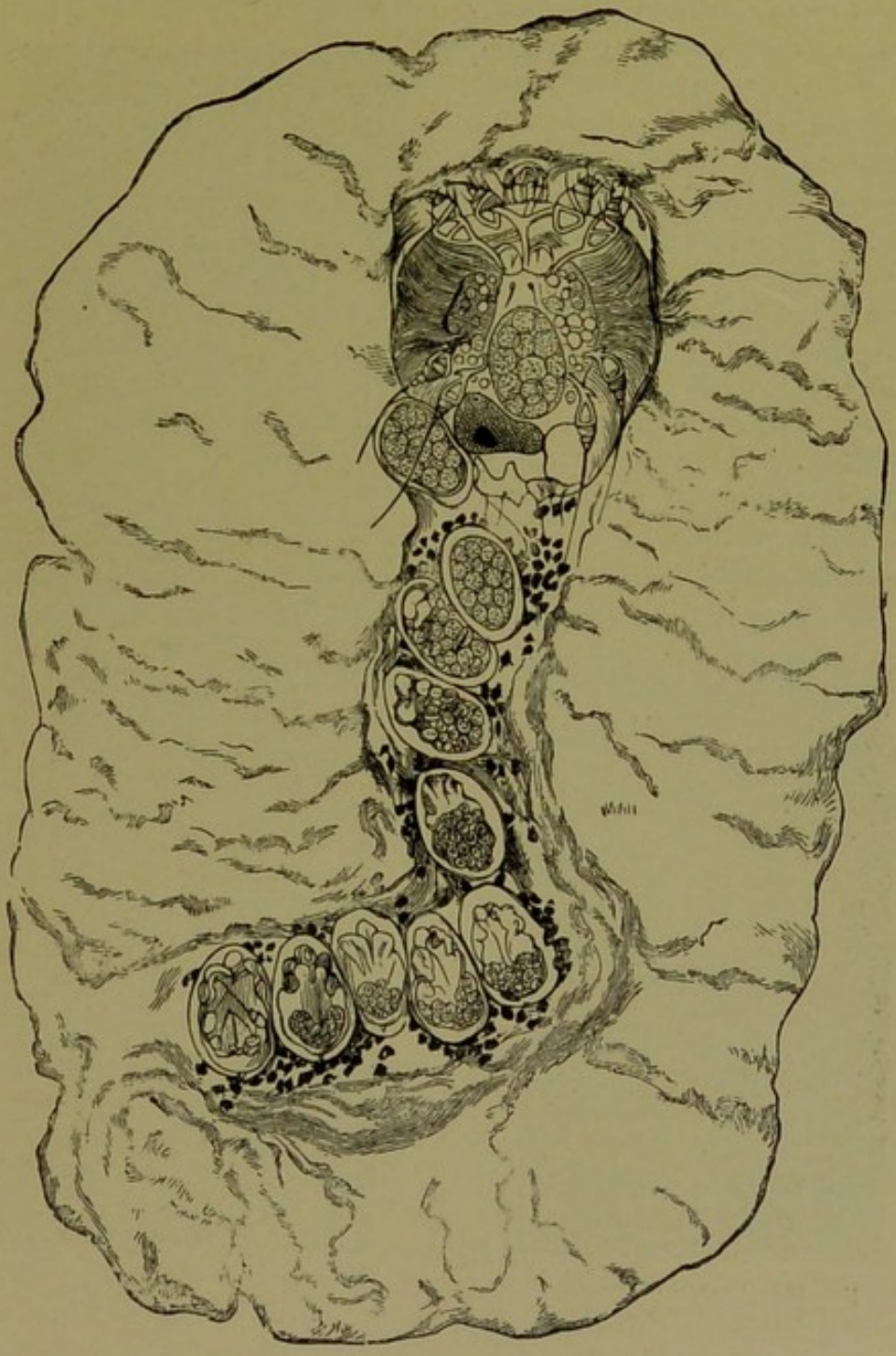


Fig. 177.—Burrow containing female acarus. (Hebra.)

The acarus contains an egg; behind it is seen a series of eggs in increasing stages of development. The black spots are the excrement of the parasite.

burrow, which is generally found on the wrists and anterior surface of the forearms, in the interdigital spaces and on the sides of the

fingers, and on the palms, when the epidermis is thin. It appears as a small gray line, more marked in uncleanly people, but does not disappear with washing. It is generally 2 or 3 millimetres in length, but sometimes reaches 3 or 4 centimetres. It is seldom straight, more usually curved or tortuous. When examined under a lens; the burrow presents two extremities: (1) the point of entry of the parasite; (2) the terminal point, which appears white and raised owing to the presence of the acarus, which can be extracted with a pin. The burrow contains eggs and excrement. It is sometimes isolated, sometimes close to a vesicle or pustule; but the parasite is never in contact with the liquid contained in the vesicle or pustule. Burrows may also occur on the feet, especially in children. They are then found on the dorsum of the foot, on the sides of the toes, and around the malleoli. They may also occur on the anus, in the axillæ, on the abdomen and thighs. In these situations the burrows are smaller and form brown lines, much straighter than on the hands. The burrows are also found

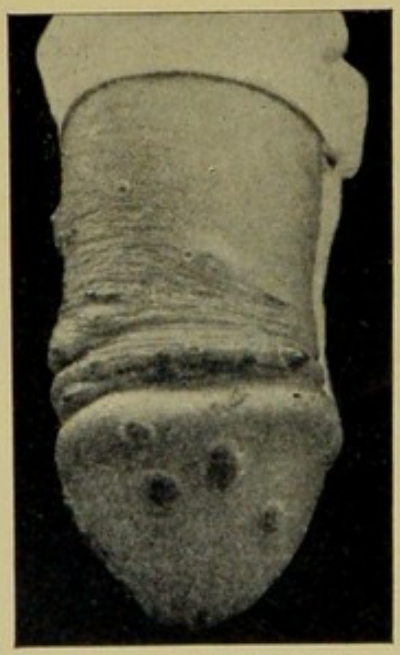


Fig. 178.—Scabies of the glans penis. (St Louis Hospital Museum.)

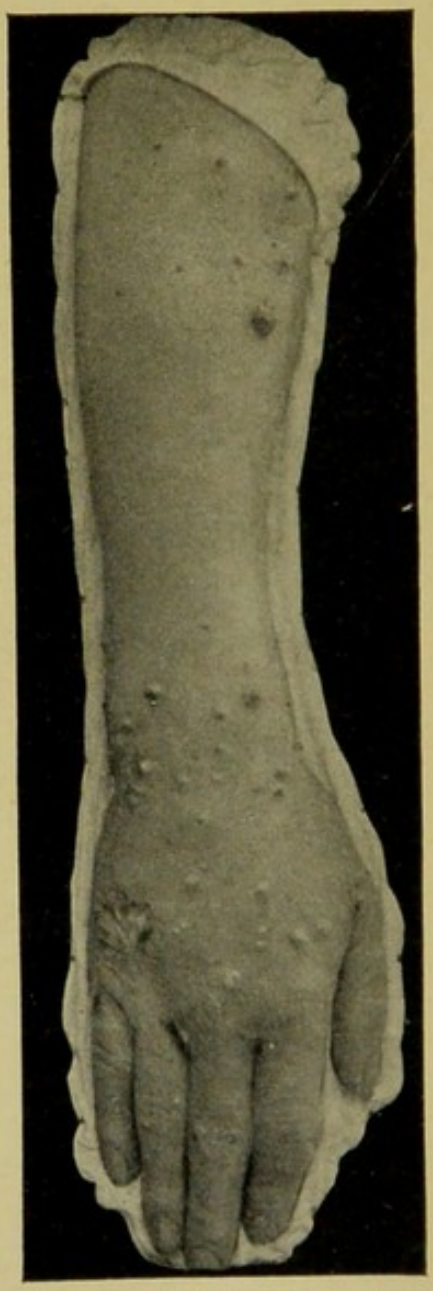


Fig. 179.—Pustular scabies. (St Louis Hospital Museum.)

on the breasts in women, and on the scrotum, glans penis, prepuce and skin of the penis in men, where their presence is pathognomonic; they are very small, sometimes straight, sometimes curved, but usually altered by scratching.

Scabies may thus affect the whole body, with the exception of the face and scalp, where any lesions which occur are secondary eruptions.

Complications.—The burrow due to the presence of the acarus is the characteristic lesion of scabies, but other eruptions are often present, due to scratching and secondary infection and to the irritation caused by the parasites.

First of all there are excoriations, then papules of prurigo covered with blood crusts (papular scabies). On the hands, vesicles are often present, the size of a hemp seed and containing clear or opaline liquid; sometimes these form actual bullæ (vesicular scabies).

Ecthymatous pustules often occur on the hands, sometimes on the feet, anus and buttocks, especially in children. More rarely, small pustules are observed, especially on the elbows, which rupture and give place to yellow impetiginous crusts; impetigo of the elbow is almost pathognomonic of scabies (Hardy).

Secondary infections may cause furuncles, dermic abscesses, lymphangitis and suppurative adenitis. All these purulent lesions result from inoculation of excoriations with pyogenic microbes.

Lastly, in predisposed individuals an eruption of ordinary eczema may develop under the influence of the cutaneous irritation.

Each of these eruptions may predominate in different cases, but the eruption is never of a single type, polymorphism being an essential character of scabies.

Scabies causes intense itching, especially when the patient is in bed, for the acarus is nocturnal in its habits. The itching may be so great as to cause insomnia, loss of appetite, and wasting.

Evolution.—The eruptions of scabies do not appear at once, but are preceded by pruritus, which gradually increases in intensity. After a time the eruptions appear; first papular, then vesicular, and lastly pustular. In persons of cleanly habits, scabies may exist for a long time without attaining any great development; but in uncleanly persons the lesions soon become generalised.

A curious fact is that scabies becomes quiescent during the course of fevers, such as typhoid, pneumonia, etc., to revive after the fever has subsided. The parasites do not thrive on a diseased subject and most of them die, but their eggs remain and hatch during convalescence.

When untreated, scabies persists indefinitely. This is still observed in some parts of Switzerland, Norway and Russia, where

scabies is characterised by the formation of thick crusts, especially on the hands and feet; the nails become broken and detached at their borders, and impetiginous crusts form on the face and scalp. This form, called Norwegian scabies, according to Méguin, is caused by a special form of acarus.

ETIOLOGY.—The sole cause of scabies is contagion, which is due to prolonged contact, from sleeping with infected persons or wearing their clothes. Scabies occurs in animals, but is not due to the same acarus; however, scabies of the cat, dog and horse is transmissible to man, but is easily cured by cleanliness and sulphur baths.

DIAGNOSIS.—This depends on the presence of the characteristic burrows. It is not necessary to find the acarus, it is sufficient to distinguish the dark lines of the burrows and the small excoriations present on the surface of the skin. The diagnosis may even be established by the polymorphism of the lesions, and by their distribution on the genital organs in men and the breasts in women, and in the absence of burrows this is often the only means of diagnosis; for instance, in bricklayers, tanners, and workmen who handle irritating substances, in whom the acarus is not found on the hands.

The presence of burrows distinguishes scabies from *prurigo*, *strophulus* and *pediculosis*, which occur chiefly on the back and shoulders. Eczema, impetigo and ecthyma complicating scabies are distinguished from simple *eczema*, *impetigo* and *ecthyma*, by the presence of burrows and the distribution of the lesions.

TREATMENT.—According to the indications given by Hardy, scabies can often be cured in twenty-four hours. The patient is first rubbed with soft soap, which softens the epidermis and opens the burrows; he then takes a bath, after which the following ointment is rubbed in energetically:—

Sulphur	2 parts
Carbonate of potassium . . .	1 part
Lard	12 parts

The ointment is left in contact with the skin for twenty-four hours, after which the patient takes another bath.

But this is not all: the patient's clothes must be heated in an oven, the under-linen and bedclothes disinfected, and the gloves burnt; otherwise recurrence is inevitable. The temperature of the oven should be 120° C.

The treatment sometimes causes considerable irritation, which may be treated by starch baths.

Another method of treatment, more suitable for private patients has been devised by Fournier. After the patient has been rubbed

with ordinary soap in a bath, he is rubbed with the following ointment:—

Sulphur	100 parts
Carbonate of soda	50 „
Gum tragacanth	1 part
Glycerine	200 parts

In young children and persons with a tender skin, also in patients affected with heart disease, albuminuria, and in pregnant women, for whom such energetic treatment is impracticable, the following ointments may be used: Styrax 20 per 100; or naphthol 1, 2, or 3 per cent., according to the state of the skin. These should be applied morning and evening for several consecutive days.

Another very simple form of treatment consists in rubbing the body for about twenty minutes with balsam of Peru, without a previous bath. The balsam is kept on the body during the night and washed off in the morning, after which the scabies is cured. This method should not be used in young children, as fatal cases have been reported due to rapid intoxication. The best treatment for children is naphthol ointment, 1 or 2 per cent. according to the age of the child and the condition of the skin; this is applied for several days.

The difficulty in the treatment of scabies is to know when it is cured. Patients often return with itching and request fresh treatment; in these cases starch baths and antipruriginous lotions should be prescribed; treatment for scabies should only be repeated when intact burrows are found.

PEDICULOSIS.

This is caused by the presence of lice, of which three species are parasitic for man; the head louse (*pediculus capitis*), the body louse (*pediculus corporis*), and the pubic louse (*pediculus pubis*).

Pediculosis of the Head.

The head louse (*pediculus capitis*) lives among the hairs. It is very common in children, especially in those with uncleanly habits and surroundings. It may occur in the eyebrows and beard in adults.

After convalescence from protracted diseases, and after childbirth, the head is sometimes full of lice; but these were present before the commencement of the disease and were unable to multiply till after convalescence.

Symptomatology. — When few in number, pediculi only cause

itching, a few papules of prurigo on the nape of the neck, and excoriations produced by scratching; but when they are more numerous, they give rise to pustular eruptions due to inoculation of the excoriations with pyogenic cocci, especially in lymphatic children.

In children, impetigo secondary to pediculosis is very common (*impetigo granulata*); when the crusts are detached and the hairs separated, the lice are seen swarming underneath. Suppuration may extend deeply and cause small abscesses in the scalp; the cervical

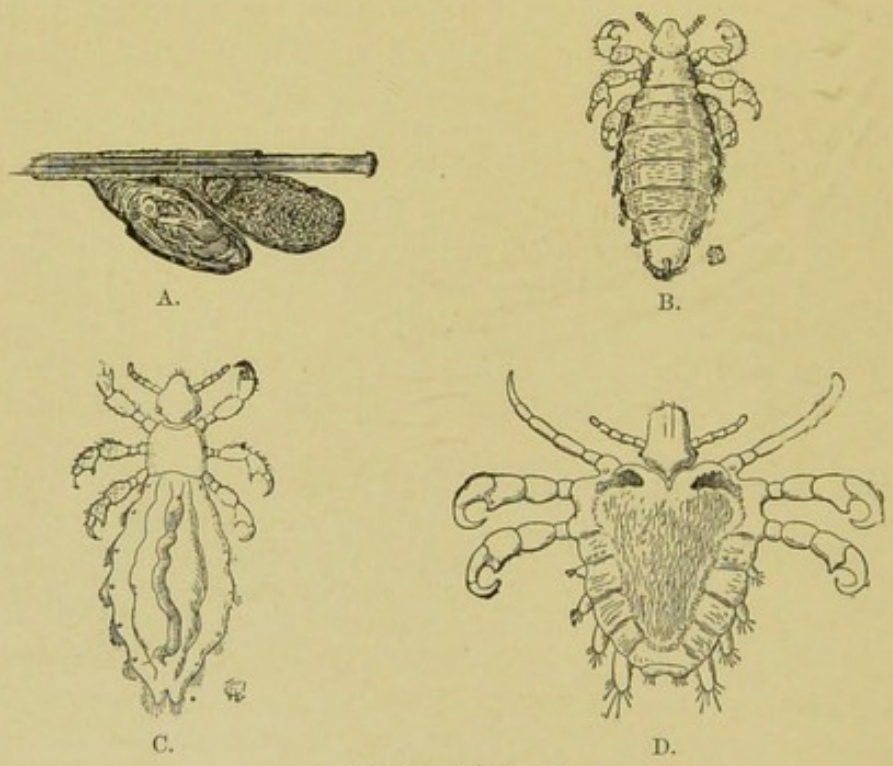


FIG. 180.—Human lice.

A, nits, or eggs glued to the hair; B, *pediculus capitis*; C, pediculus corporis; D, pediculus pubis. (Mracek-Hudelo.)

lymphatic glands are enlarged and sometimes suppurate. Lastly, the eruptions may be complicated by seborrhœa, gluing the hairs together, and producing what has been called *trichoma*, or *plica polonica*; this is common among the Jews in Russia, Polonia and Germany.

The itching and suppuration of pediculosis may cause insomnia and wasting, and in predisposed subjects the cutaneous irritation may cause an eruption of eczema.

Diagnosis.—This is easily established by the presence of the parasites, and the eggs glued to the hairs (nits); the latter must be distinguished from scales and seborrhœic crusts.

Treatment.—In children and in men the hair can be cut short, after which the scalp may be treated with camphorated alcohol or

a solution of sublimate (1 in 500), to kill the parasites and destroy the nits. In women the treatment is tedious unless the hair is sacrificed. The same lotions may be used, followed by dusting the head with powdered staphisagria. The nits can be detached from the hairs, to which they adhere closely, by means of warm vinegar. Secondary cutaneous lesions must be treated after the lice and nits have been got rid of.

Pediculosis of the Body.

The body louse is larger than the head louse, and of a dirty white colour. It is rarely found on the skin, more often on the clothes worn next the skin. The lice occur chiefly on elderly, ill-nourished persons of dirty habits; many cases of senile prurigo are nothing more than pediculous prurigo, and the so-called "vagabond's disease" is chronic pediculosis.

Symptomatology.—The lice produce papules by pricking the skin to absorb the blood; the papules cause acute itching, and lead to scratching. The excoriated papules become covered with blood crusts, or they may take the form of linear excoriations covered with dried blood. After a time the scratching produces a lichenoid and pigmented condition of the skin. The lesions occur chiefly on back, shoulders, and nape of the neck; even when no lice are found, scratch marks situated in these regions are usually due to pediculi. The excoriations may be the origin of secondary infections; lymphangitis, furuncles, ecthyma, and even subcutaneous abscesses.

Diagnosis.—As already stated, the presence of prurigo on the back and shoulders is generally sufficient for the diagnosis of pediculosis. The diagnosis can be confirmed by searching for lice in the clothes.

The pigmentation secondary to pediculosis differs from that occurring in Addison's disease; the latter is generalised, and is not associated with itching and scratch marks, and pigmentary patches are present on the mucous membranes. However, pigmentary spots on the mucous membranes have been reported in pediculosis, but even admitting the possibility of this occurrence, which seems to me very doubtful, the presence of intestinal disorders and profound asthenia is peculiar to Addison's disease.

Treatment.—This consists in disinfecting the clothes by heat, changing the linen and bedclothes, and treatment of the skin by sulphur baths and carbolic lotion (1 per 100).

Pediculosis Pubis.

The pubic louse or crab-louse is a broad, gray insect, with three pairs of appendages terminating in claws, which it fixes in the skin

so that it is difficult to detach. It is usually found on the genital region, but may occur in other hairy regions, such as the axillæ, chest, eyebrows, eyelashes and beard, but never on the scalp. It is usually transmitted during sexual connection.

Symptoms.—The chief symptom is intense itching. After a time, papules, scratch marks, eczematiform lesions, and blue spots appear, especially on the abdomen and inner surface of the thighs. The blue spots are due to a special substance secreted by a glandular apparatus in the parasite.

Diagnosis.—The diagnosis depends on the presence of the parasites, nits, and blue spots. The lice are often very adherent to the skin, and appear as black spots.

Treatment.—This consists in the application of sublimate lotion (1 in 500), or mercurial ointment, for two or three days.

VARIOUS ANIMAL PARASITES.

Besides the *acarus scabiei* and *pediculi*, the skin may be attacked by other animal parasites. These are of three kinds: some are intracutaneous or dermatozoic, like the acarus; these include the harvest bug (*leptus*), the bird-mite (*dermanyssus*), and the wood-tick (*ixodes*); others are epizoic, like pediculi; these include bugs, fleas and gnats; others belong to the hæmatozoa, and include *filaria* and *cysticercus*.

Leptus autumnalis (Harvest-bug).—This is the larva of the *trombidion*, belonging to the order of Acarina. It is a small orange-coloured larva, visible to the naked eye, and inhabiting gardens and fields. It attaches itself to the skin by its mandibles, and gives rise to papules, which cause much itching and irritation.

The best treatment consists in sulphur baths and lotions. Méguin recommends benzene.

Dermanyssus gallinæ (Bird-mite).—This is a form of acarus which is parasitic in birds and fowls. In man, it causes a pruriginous papular eruption on the hand. The application of vinegar lotion or dilute acetic acid is sufficient treatment.

Ixodes (Wood-tick).—These are Acari, the females of which puncture the skin with their proboscis and suck the blood till their bodies swell up and form small round swellings. When forcibly removed, the proboscis remains buried in the skin and causes painful nodules, but they can be easily removed entire after applying petrol or benzene.

Cimex lectularius (Bed-bug).—Punctures by bugs give rise to urticarial spots with papules of prurigo and excoriations due to scratching; sometimes to a vesiculo-bullous eruption, which may be

mistaken for dermatitis herpetiformis. Treatment consists in anti-pruriginous lotions and starch baths.

Pulex irritans (Flea).—This causes punctiform hæmorrhages, surrounded by a small red areola.

Mosquitos and Gnats.—The punctures of these insects give rise to round papules, very pruriginous and sometimes rather painful, surrounded by an erythematous patch of varied extent; sometimes there is œdema.

We may also mention bees, wasps, hornets, certain caterpillars and scorpions, which cause irritating punctures; and medusæ, which cause urticarial eruptions.

Filaria sanguinis.—Filariosis manifests itself on the skin by large, red, pruriginous patches covered with papules, which soon become vesicles, then pustules, and later on crusts (*craw-craw*). When the crusts fall off, they leave large spots of blood pigment, which slowly disappears. Microscopic examination of the fluid from the vesicles shows the presence of filaria.

Cysticercus.—This sometimes occurs in the skin, but more often in the subcutaneous tissue, in the form of cysts, which are usually multiple, round, elastic and painless unless inflamed. Diagnosis can only be made by puncture of the cyst and examination of the fluid for the characteristic hooklets.

Treatment consists in excision of the cysts, or evacuation of the liquid contents, followed by injection of a solution of iodine.

CUTANEOUS SYPHILIDES.

THE name *syphilides* was given by Alibert to the tegumentary manifestations of syphilis. These manifestations may affect the skin and the dermo-papillary mucous membranes, so that there are *cutaneous syphilides* and *mucous syphilides;* the latter being also called *mucous patches.*

In the evolution of the chronic general infection of syphilis, the cutaneous syphilides constitute, if not always the first, at any rate the most important and the most visible of the early manifestations; in doubtful cases, their appearance confirms the diagnosis of the chancre.

The exanthem of secondary syphilis follows soon after the chancre, and may be represented, in varying frequency, by all forms of syphilides. In fact, the cutaneous syphilides include eruptions of different types, which belong first of all to the secondary period. These syphilides may reappear with the same types, but with a different arrangement, in the tertiary period.

In reality, the terms secondary and tertiary *lesions* of syphilis are incorrect; there are only secondary and tertiary *periods.* Secondary syphilis includes the early lesions, tertiary syphilis the later.

Under the name of **secondary syphilis** must be understood all manifestations which are due to general infection of the body by the syphilitic virus. Syphilis is, in fact, a general or constitutional disease, in which the causal agent, the *Spirochæta pallida,* has been found in the blood. In acquired syphilis, the spirochæte is rarely found in the general circulation, as the organism defends itself against the microbic invasion; but, in fatal cases of congenital syphilis, the spirochæte is present in great numbers in the blood and in the heart.

The manifestations of secondary syphilis appear usually about six weeks after the chancre. Their extreme limits of development are: at the earliest, one month, or rarely three weeks after the beginning of the chancre; at the latest, two months, rarely more. After chancres of the lips, tongue and tonsils, the secondary exanthem

generally appears earlier, sometimes less than three weeks after the chancre. The secondary manifestations usually last several months.

At this period, syphilis may affect all the organs, the viscera and the nervous system, as well as the skin and mucous membranes. But the most frequent manifestations are the cutaneous and mucous syphilides.

Secondary syphilis sometimes develops insidiously, without any general symptoms. Sometimes the secondary symptoms are so benign, and the chancre heals so rapidly, that neither are noticed. But, in the great majority of cases, the onset of the secondary period is accompanied by symptoms of general infection, which are similar to those observed at the onset of all chronic infections.

First of all, there is *headache*, but this has special characters which at once suggest syphilis. The *syphilitic headache* is dull and continuous, with nocturnal exacerbations, which often cause insomnia. It is of great importance in the diagnosis of doubtful eruptions.

Fig. 181.—Living *Spirochæta pallida*, seen by dark ground illumination (× 500). (Dr Mucha.)

The second symptom, which is often unnoticed, is fever. *Syphilitic fever*, in the majority of cases, is transitory. Sometimes it recurs in the form of feverish attacks with slight shivering. In some cases syphilitic fever is continuous, the temperature remaining high with evening rises. This form, which Fournier has called *syphilitic typhose*, may be easily mistaken for typhoid fever.

Among other general symptoms, there may be *albuminuria*. This is a dyscrasic albuminuria, quite different to that of syphilitic nephritis; it is fairly common, but so transitory that it generally escapes notice. If the urine of syphilitics is examined systematically, albuminuria will be found in more than a third of the cases.

This albuminuria is connected with nutritive disturbances produced by syphilis, as by other chronic intoxications. These disorders of nutrition are characterised principally by incomplete transformation of nitrogenous matter in the organism, by diminution in the nitrogenous output; in other words, by a relative increase in the

quantity of extractive matters compared with that of urea in the urine.

The *function of the liver* may also be affected by the syphilitic toxi-infection. Paris and Dobrovici have shown that alimentary

FIG. 182.—Section of liver from case of congenital syphilis, showing *Spirochæta pallida*, stained by silver nitrate. (Levaditi.)

v, Portal vessel, containing cellular debris (*g*) and spirochætes (*s'*); *p*, wall of vessel infiltrated with spirochætes (*s'''*); *s*, spirochætes in the vascular endothelium; *h*, group of hepatic cells, altered by maceration, and containing spirochætes (*s''*).

glycosuria occurs at the beginning of the secondary period in nearly 50 per cent. of cases which have not been treated. Two hours after the ingestion of 150 grammes of glycose, sugar appeared in the urine. That this disturbance of the glycolytic function of the liver could

not be attributed to any other cause than syphilis, was demonstrated by its disappearance after twenty daily injections of benzoate of mercury.

These facts throw some light on the pathogeny of diabetes, which is common in syphilitic subjects, the alimentary glycosuria being no doubt transformed later on, in certain cases, into true saccharine diabetes.

The general disturbance of the organism is also manifested by changes in the epidermic products, resulting in diffuse *alopecia*. This syphilitic alopecia (called alopecia *en clairières* by Fournier) does not produce white smooth patches like those of alopecia areata, but resembles rather a growth of underwood which has been irregularly cut.

Such are the manifestations which generally accompany the appearance of the early syphilitic exanthem.

The early or **secondary syphilitic exanthem** is characterised by generalisation and dissemination of the lesions. Secondary syphilides affect the whole surface of the skin, while tertiary syphilides are grouped and localised. This is the chief and only true distinctive character between secondary and tertiary syphilis; for, if the lesions are considered by themselves, they are fundamentally the same in both cases.

In rare cases the secondary exanthem may be absent; I have seen a case, the syphilitic nature of which was proved by the subsequent appearance of iritis and tertiary syphilides, in which no secondary exanthem was observed, although the patient was examined daily.

However, the absence of secondary cutaneous lesions is exceptional, and in most cases the specific exanthem appears from one to two months after the chancre. This exanthem may present different types, in fact, all the types of elementary lesions of the skin. These different types are all characterised by the absence of pain, itching, and inflammatory reaction. These characters are important in the diagnosis of disseminated *syphiloid* eruptions which may be mistaken for secondary syphilides.

The same characters apply to the eruptions of the tertiary period. In fact, the same types of eruption may occur in both the secondary and tertiary periods, but with a different distribution. It is on the generalisation or localisation of the lesions that the chronological diagnosis of syphilis depends; the lesions are morphologically the same in both periods.

The principal types of syphilitic eruptions are the following: the erythematous type, the papular type, and the crustate type.

The *erythematous type* occurs in the form of red spots of various sizes. It constitutes the *roseola*, the most common form of secondary exanthem.

The *papular type* includes the papular syphilide and the tubercular syphilide, according to the size of the elements.

The *crustate type*, which resembles *ecthyma*, is sometimes called the ulcero-crustate syphilide.

These three principal types may be subdivided into a certain number of varieties.

In all these types of syphilitic exanthem of the secondary period, the *spirochæta pallida* is almost constantly present in the elements of the eruption.

There is another form of exanthem which appears at the end of the secondary period; this is the *pigmentary syphilide*, which will be described later. This pigmentation, the pathogeny of which has been much disputed, may persist for a long time and has an indefinite duration.

We will now consider the different types of syphilides.

The Erythematous Syphilide, or Roseola.

This is the most common form of secondary syphilide. It consists of an eruption of spots, from a lentil to a sixpence in size, or larger. The spots are generally round or oval, sometimes irregular. They are rose-coloured at first, but become yellow as they fade. They are neither raised nor squamous. The spots are generalised and occur on the whole surface of the body, with the exception of the face; but occasionally a few are seen on the forehead. Although generalised, they have a predilection for the sides of the thorax, the flanks and the anterior surface of the forearms. These seats of election are also the situations where the eruption first appears, and where it should be looked for in doubtful cases.

The roseola develops progressively and the spots increase in number for about a week, after which they persist for a variable time. In the absence of treatment, the eruption may disappear in a few days, or it may last for several months and become transformed into a papular eruption.

A curious fact is that the roseola may reappear after complete disappearance, constituting what is called *recurrent roseola*. This is similar in character to the original eruption, but is generally more discrete. I have seen a case in which roseola recurred twice; after three months in the first instance, and after five months in the second.

Sometimes the roseola takes the form of complete or incomplete rings, instead of spots. This form, known as *annular roseola*, appears later than the macular form. It has the same distribution, except that it has a predilection for the neck and upper part of the thorax. It differs from ordinary roseola only in the shape of its elements.

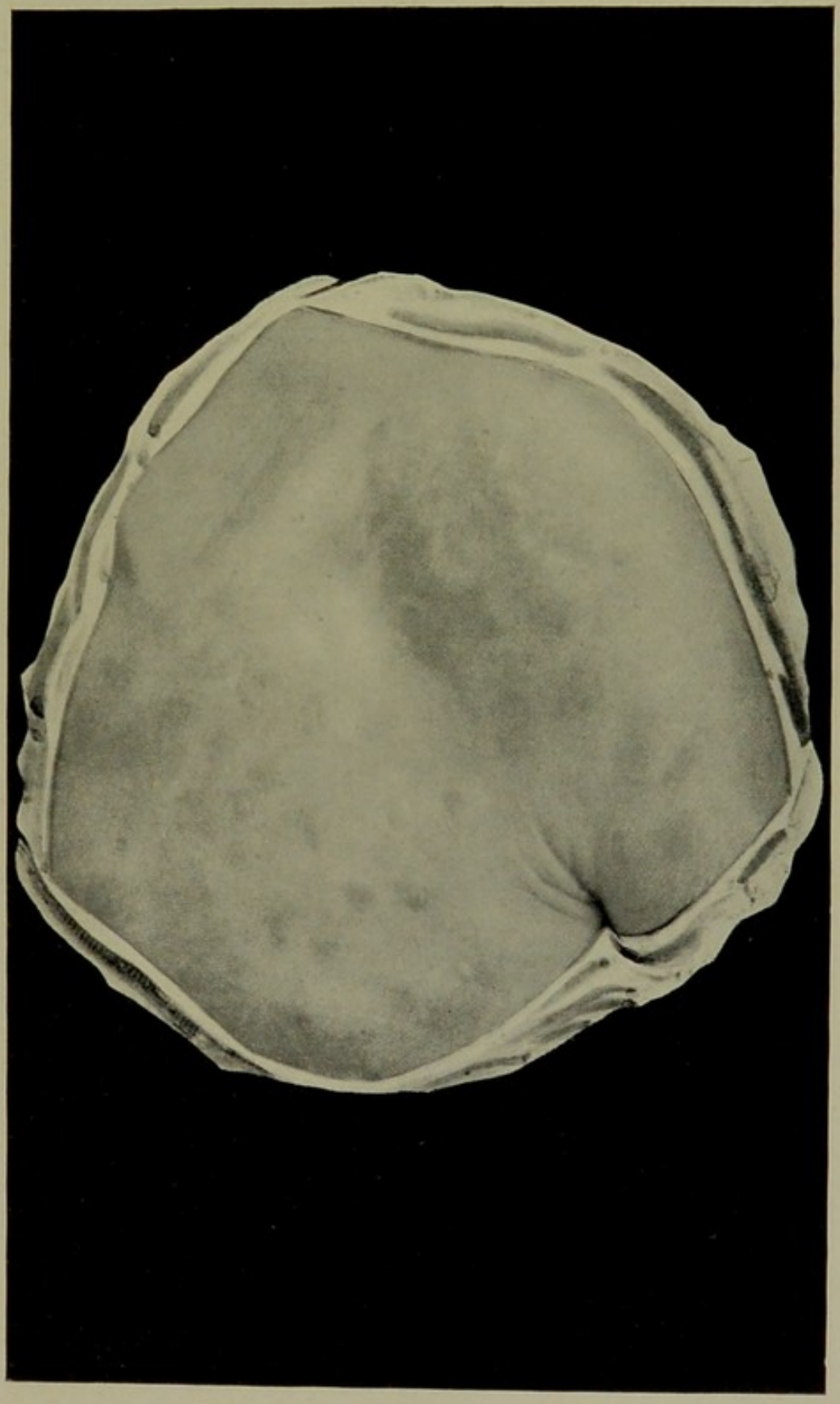


Fig. 183.—Syphilitic roseola. (St Louis Hospital Museum.)

[*To face page* 418.

DIAGNOSIS.[1]—This is generally easy, but there are some affections which present a certain resemblance to roseola, the chief of which are pityriasis rosea and medicamentous erythemas due to copaiba and antipyrin. *Pityriasis rosea* so greatly resembles syphilitic roseola that it was described by Fournier under the name of squamous roseola. However, it has a peculiar evolution, extending progressively from the upper to the lower parts of the body. It first appears on the neck and shoulders, and gradually spreads to the trunk and upper part of the thighs; on each limb it spreads from the base to the extremity. In other words, the eruption extends from above downwards on the body and on each segment of the body. In pityriasis rosea there is no trace of a chancre; while, in syphilitic roseola, the infection is too recent for the chancre to have completely disappeared. Lastly, pityriasis rosea is finely squamous; syphilitic roseola is never squamous.

Medicamentous erythemas, especially those due to *copaiba* and *antipyrin*, may be mistaken for syphilitic roseola. This diagnosis has frequently to be made, because a syphilitic patient may be taking copaiba for concomitant gonorrhœa, or antipyrin for headache.

In *copaiba erythema* the spots are raised, not flat like those of syphilitic roseola, and are chiefly situated on the wrists, elbows, and knees; lastly, copaiba erythema differs from syphilitic roseola in being very pruriginous or even painful. The diagnosis between syphilitic roseola and *antipyrin erythema* is more difficult; but, in the latter, the spots are grouped instead of regularly disseminated, larger than the macules of roseola, and irregular in outline instead of round or oval. The history of a chancre or the presence of an indurated cicatrix will settle the diagnosis.

Tertiary Erythema.—An erythematous syphilide may also occur in the tertiary period. This confirms what has already been said, viz., that the syphilides are morphologically the same at the different periods of the disease, and only differ in their distribution.

Tertiary erythema consists of elements which are similar to those of roseola, but grouped on limited regions. They may be present simultaneously on various parts of the body, but in each part they are arranged in circumscribed groups, instead of being diffuse like secondary erythema. The elements, round or annular, are also larger than those of secondary erythema.

[1] In the general diagnosis of syphilitic lesions, useful evidence is furnished by the method of serum-diagnosis introduced by Wassermann, Neisser and Bruck. This method gives positive results in the great majority of cases of primary, secondary and tertiary syphilis, during the presence of lesions, and in nearly all cases of congenital syphilis. Serum diagnosis, however, should not replace clinical diagnosis, but should be employed as an adjuvant to this in doubtful cases.—ED.

The Papular Syphilide.

Histologically, the papule is constituted by embryonic infiltration of a group of papillæ in the dermis. As regards its evolution, it is only an exaggeration of a congestive spot; in other words, the papule commences as a macule. All the tegumentary lesions of syphilis commence by localised congestion of a group of papillæ, accompanied by a slight degree of embryonic infiltration and connective-tissue proliferation. If these processes increase, the lesion, instead of remaining macular, gives rise to an elevation, which constitutes the papule. This papule forms a hard elevation of variable size. It remains exclusively papular for a short time only, and soon becomes covered with squames, so that the term papulo-squamous syphilide is better than papular.

The *papulo-squamous syphilide* may appear as such, or may succeed the roseola by transformation *in situ* of macules into papules. There are several varieties:—(1) The *common form*, which is the most frequent, consists of round or lenticular papules, with a flat surface and a reddish brown or copper colour. The papules are sometimes completely covered with squames; but, more often, the desquamation only occurs at the periphery of the papule, and presents the form of a detached ring of epidermis surrounding the papule, the centre of which remains smooth. This has been called the "collar of Biett," after the observer who first drew attention to it.

The eruption is usually generalised, and develops in successive crops. When fully developed, it occupies the whole surface of the body, including the face and scalp, and is therefore more generalised than roseola. The eruption lasts for one to three months, after which the papules subside and leave brown spots, which gradually disappear.

But syphilitic papules, when situated on the neck, may give rise to persistent changes in pigmentation. In this region there is often a small decolorised zone around the papule, which persists after it, and spreads towards the centre in proportion as the pigmented spot, which replaces the papule, contracts. I have given the name of *peri- and post-papular leuco-melanodermia* to this condition. Sometimes complete depigmentation occurs from the first, and directly succeeds the papule.

Papular syphilides are not confined to the secondary period, they also occur in the tertiary period, with the same characters. But in the latter case the papules are grouped instead of disseminated, and unite to form circles, constituting the *circinate type*. They may occur at the same time on different parts of the body, but in each region they are always grouped and localised, instead of being generalised and diffuse as in the secondary period. The papular

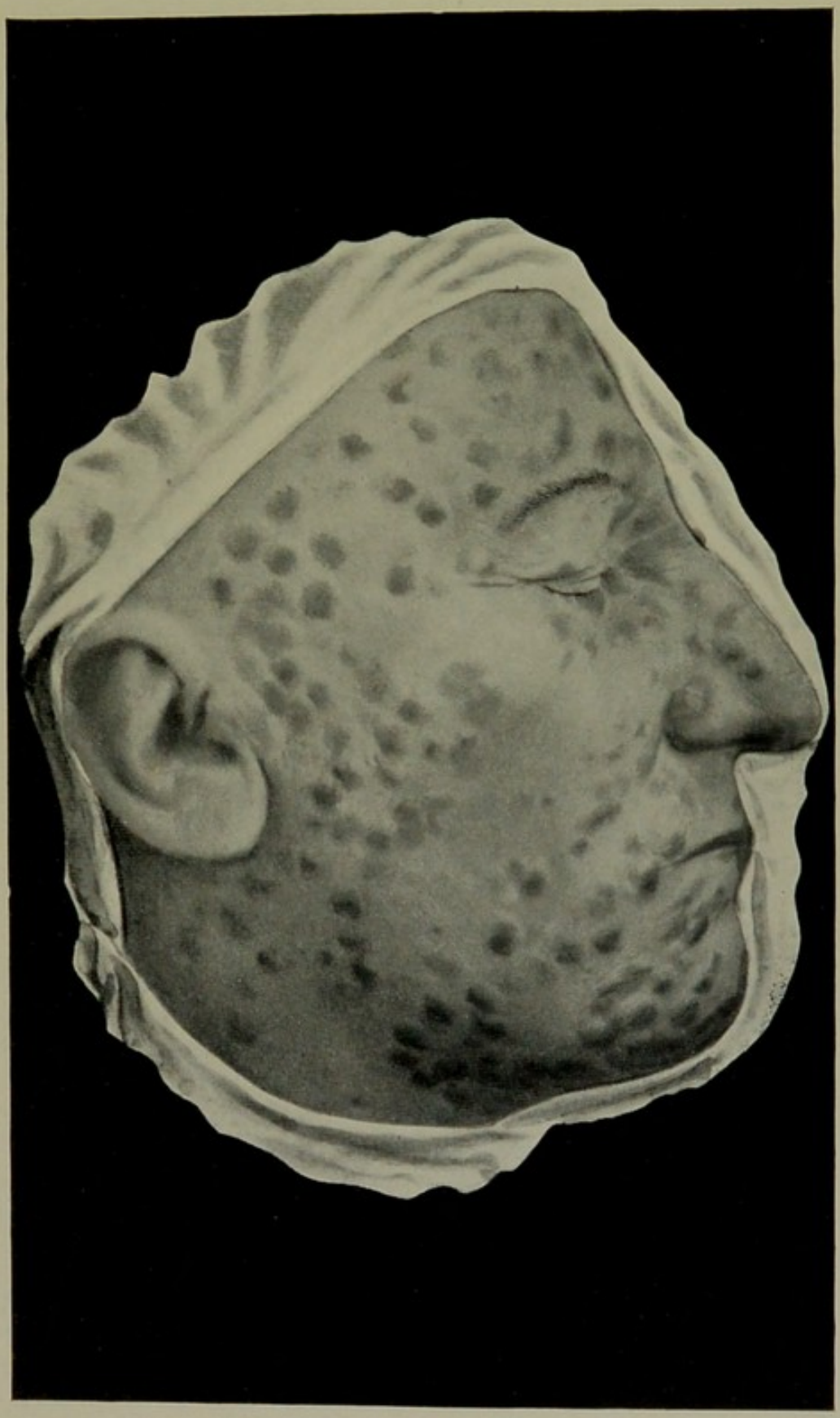


FIG. 184.—Papular syphilide. (St Louis Hospital Museum.)

[*To face page* 420.

syphilide, when situated on the forehead, has been facetiously called the *corona veneris.*

Zoniform syphilides.—In some cases, syphilitic papules, in the tertiary period, may be grouped in linear series or elongated patches on one half of the trunk or on one side of the forehead; a *zoniform* distribution simulating zona, for which they are often mistaken.

The other varieties of papular syphilides are less common, but present important peculiarities.

(2) The *miliary* or *lichenoid form* is a variety of papular syphilide in which the papules are no larger than a pin's head. The elements are more confluent and more numerous than in the preceding form. Sometimes the papules are very minute, hard and confluent, and resemble keratosis pilaris. This variety may be called *keratosic.*

The miliary papular syphilide, especially the keratosic variety, is usually rather tenacious and rebellious to treatment, and usually persists for several months. This lichenoid form may, like the others, occur in the tertiary period. The difference is always the same, the lesions being grouped and localised, instead of generalised as in the secondary period.

(3) The *papulo-tubercular form* only differs from the ordinary papular syphilide in the larger size of its elements and their greater elevation above the skin. The tubercle is, in fact, only a large papule. This form may also occur in both the secondary and tertiary periods, being diffuse in the former, circumscribed in the latter. It may also leave a persistent leucodermia.

(4) The *psoriasiform* or *squamous form* is much more squamous than the papular form. It may occur on various parts of the body, but has a predilection for the palms and soles.

This squamous syphilide, which was formerly called palmar and plantar psoriasis, is characterised by the presence of stratified, horny scales, resembling those of psoriasis. It forms disseminated islets on the palms and soles. The coexistence of palmar and plantar lesions is frequent. This syphilide is often bilateral and symmetrical, but sometimes unilateral, thus differing from plantar and palmar eczema, which is always bilateral. It is very tenacious and resistant.

The squamous syphilide is a late form, and establishes a transition between the secondary and tertiary periods. It is no longer a generalised syphilide, but one which has commenced to become localised. It belongs to the end of the secondary and the beginning of the tertiary period.

(5) The *crustate* or *papulo-crustate form* is produced by excoriation or ulceration of the papules, which become covered with crusts. This form presents two varieties: the *varioliform syphilide* and the *acneiform syphilide.* In the *varioliform syphilide* (also called

syphilitic varicella by Bazin) the eruption consists of umbilicated papulo-pustules covered by crusts, formed by the drying of secondary vesicles superadded to the papules. This form has a certain resemblance to variola. In the *acneiform syphilide* the elements are very small and resemble those of acne.

These two varieties occur chiefly in the secondary period, but sometimes also in the tertiary; being generalised in the secondary, localised or grouped in the tertiary period.

DIAGNOSIS.—When situated on the face, papular syphilides may be mistaken for *acne;* but the syphilitic papules are hard, copper coloured and without inflammatory reaction; the papulo-pustules of acne are red, inflamed and often suppurating.

The papules of the miliary papular syphilide resemble those of *lichen planus;* but the elements of the syphilide, although flat and shiny like those of lichen, have not their polygonal form and central umbilication. Moreover, the syphilitic papules are arranged in a circinate manner, which is not observed in any of the usual forms of lichen planus. The miliary syphilide differs from *keratosis pilaris* by being generalised.

The papulo-tubercular syphilide when localised, as in the tertiary period, has a circinate arrangement which may be mistaken for non-ulcerative *lupus;* but the syphilitic tubercles are flat, hard, brownish red, or copper coloured, and painless on pressure: the lupus tubercles are soft, yellow, painful on pressure, and surrounded with a red inflammatory zone, while the centre is translucent and resembles barley-sugar.

The palmar squamous syphilide differs from *palmar eczema* in the following points: it is often unilateral, while eczema is always bilateral; its borders are more sharply defined than those of eczema; it remains localised to the palm, while eczema tends to spread to the back of the hand. The same differences apply to the plantar syphilide and plantar eczema. The squamous syphilide must also be distinguished from true palmar and plantar *psoriasis.* Psoriasis may occur in these regions, but it is always atypical and does not present the usual characters. It is less circinate than the squamous syphilide, the squames are thicker and more stratified, and there are usually patches of typical psoriasis on the elbows and knees.

The varioliform syphilide may be mistaken for *variola,* and inversely. These errors depend on the hospital in which the case is seen, for there is always a tendency to diagnose the affection which is met with most frequently. It is sufficient to bear in mind the possible error in order to avoid it, remembering the temperature, the state of the tongue and the pain in the back which are characteristic of variola.

The papulo-pustular form with small acneiform elements may

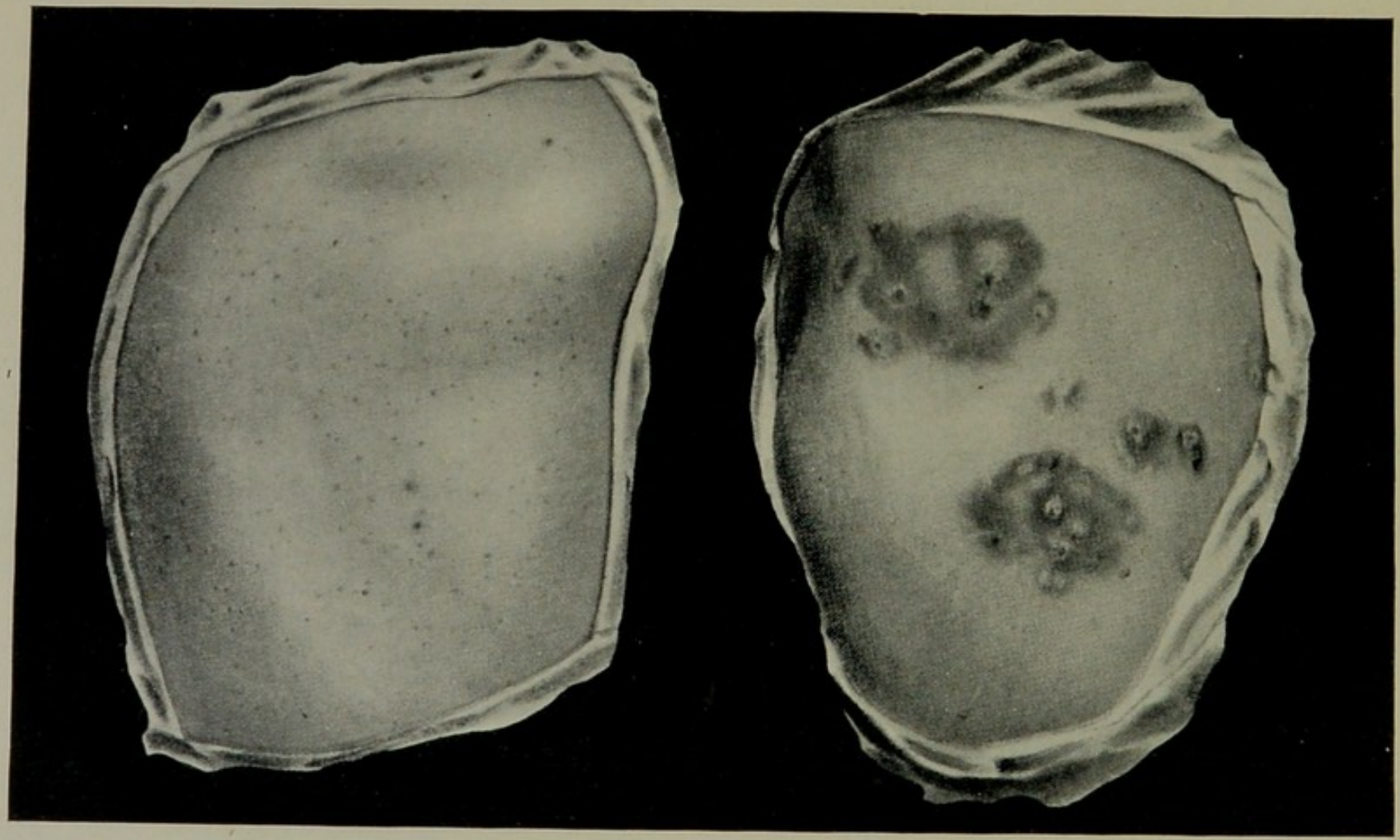


Fig. 185.—Miliary syphilide.

Fig. 186.—Papulo-squamous syphilide.

(St Louis Hospital Museum.)

[*To face page* 422.

be mistaken for *disseminated acne*, but the pustules of acne are inflammatory and painful, while those of syphilis are quite painless.

Papular and nodular cutaneous tuberculosis differs from the papulo-tubercular syphilide in the colour of the papules, which are livid or red, not copper coloured like syphilitic papules. Moreover, the tuberculides are polymorphous; they consist of papules and nodules, which ulcerate after a time and become covered with superficial crusts, thinner and less adherent than those of syphilis. Suppurating tuberculous glands are often present, which assist the diagnosis.

The Ulcero-crustate (Ecthymatous or Pustular) Syphilide.

This is formed by ulcerations of pustular origin which become covered with crusts. It is often called *syphilitic ecthyma*. The elements consist of thick, adherent, stratified brown or greenish crusts, underneath which the dermis is ulcerated. This syphilide extends deeply and always leaves cicatrices, the characters of which will be referred to later.

Syphilitic ecthyma may occasionally occur in a generalised form in the secondary period; it then results from the transformation *in situ* of a macular or papular eruption. But it is a late form of eruption which appears two or three months after the chancre, sometimes later; this generalised pustular syphilide is always preceded by a roseola, or more often by a papular syphilide. It is generally a sign of severe syphilis.

The ecthymatous syphilide occurs more often in the tertiary period, to which it properly belongs; the lesions are then localised instead of generalised, but the individual characters of the elements are the same. Sometimes, in the tertiary period, the pustules are larger and the lesions assume the type of *rupia*. The crusts of rupia are larger and thicker than those of ecthyma; they present the same dark colour, the same adherence and stratification, and sometimes resemble oyster or limpet shells.

This ulcero-crustate syphilide, localised or generalised, sometimes occurs by itself, and may be the only cutaneous manifestation of syphilis. In other cases it is associated with other types of syphilides, especially the papular type; in this case, pustules and ulcero-crustate lesions are mixed with a certain number of papules or tubercles.

All the preceding types of eruption, as already mentioned, may occur in both the secondary and tertiary periods of syphilis. The only difference between secondary and tertiary syphilides is their distribution; the secondary lesions being generalised and disseminated, the tertiary lesions localised and grouped. It must be remembered,

however, that the superficial forms, the erythematous and papular, especially the former, belong principally to the secondary period, while the deeper lesions, ecthyma and rupia, are generally met with in the tertiary period.

DIAGNOSIS.—Localised ulcero-crustate syphilides of the tertiary period have to be diagnosed from a number of affections, the chief of which are ecthyma, ulcerated lupus, epithelioma, mycosis fungoides, parasitic sycosis, acne vulgaris, and cicatricial acne pilaris.

Common *ecthyma* is more painful and more inflammatory than syphilitic ecthyma. The latter is common on the face, while ecthyma is exceptional in this region. Syphilitic ulcerations are deeper, and give rise to harder, thicker and more adherent crusts than those of ecthyma.

Ulcerated *lupus* differs from syphilitic ecthyma in the irregularity of the borders of the ulceration, and in the softness and fragility of the crusts.

Epithelioma is a papillomatous, vegetating, sanious ulceration, accompanied by glandular enlargement, which is never met with in tertiary syphilis.

Mycosis fungoides is rather a rare affection. When the tumours ulcerate, the ulcers are fungoid, irregular, and sometimes covered with a crust, but the crust is always fragile and little adherent, very different to the crusts of syphilitic ecthyma.

Parasitic sycosis occurs on the face, and may be easily mistaken for a pustular syphilide of the face and beard. But sycosis has a special localisation; it is an affection of the hair follicles and is exclusively perifollicular, a character which does not belong to syphilis. In doubtful cases a microscopic examination should be made, although a negative examination is not conclusive. In fact, it is often difficult to find the fungus in parasitic sycosis. In some cases the therapeutic test is necessary.

Pustular acne is painful and inflammatory, and can seldom be mistaken for syphilis. But this does not apply to some cases of *cicatricial acne pilaris*, in which the therapeutic test is sometimes required. This form of acne usually occurs in the naso-labial furrow, and on the forehead near the scalp; this localisation is an element in the diagnosis. Acne pilaris produces cicatrices which resemble those of syphilis, but they are deeper and more irregular.

All the preceding cutaneous syphilides — papular, papulo-crustate, tubercular, squamous, and ulcero-crustate—whether they occur during secondary or tertiary syphilis, present a uniform evolution which is always the same for each lesion. After a time the papules and tubercles subside, the crusts fall off and the subjacent ulceration cicatrises. When the syphilide is cured there

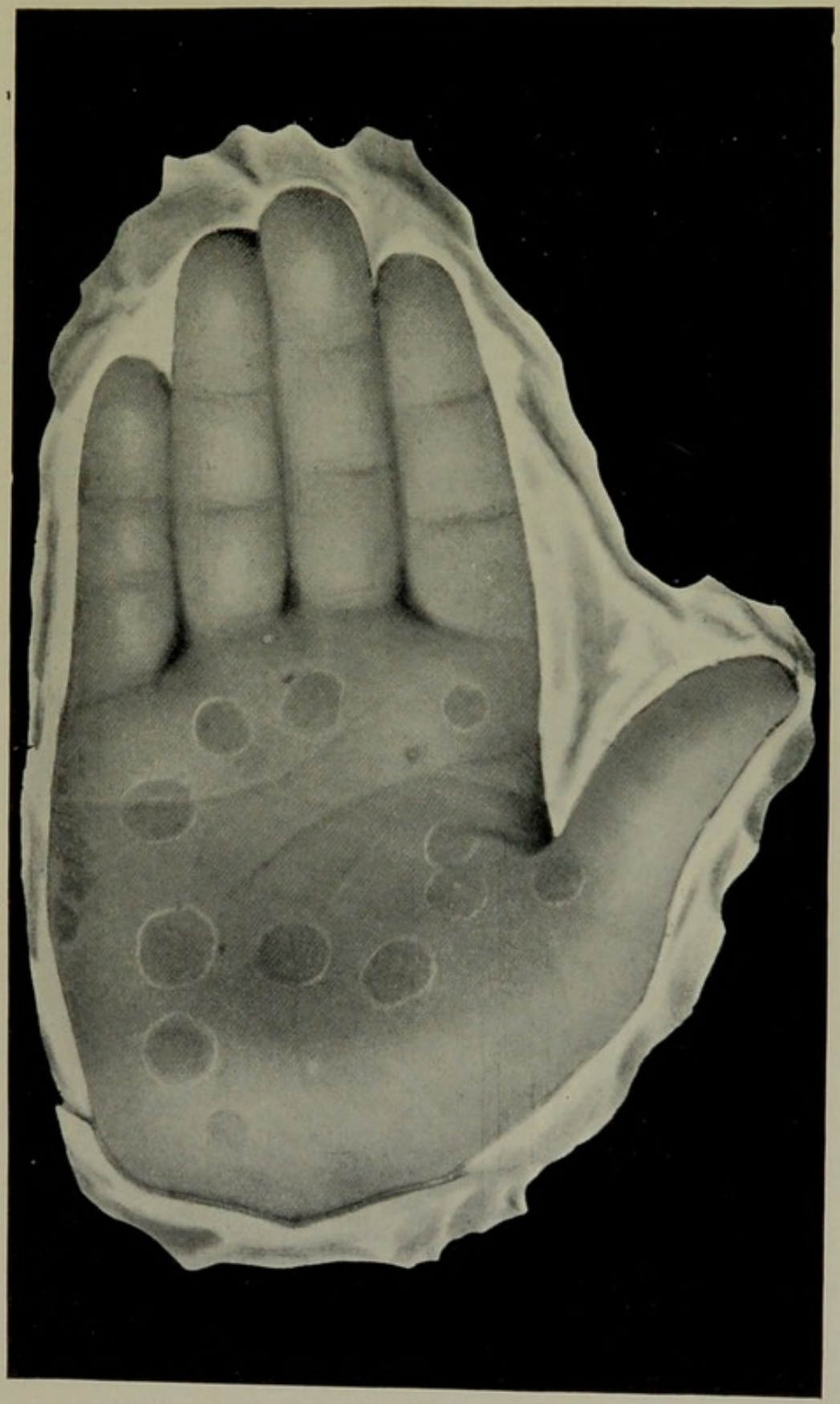


FIG. 187.—Palmar squamous syphilide. (St Louis Hospital Museum.)

[*To face page* 424.

is nothing left but brown pigmentary spots, cicatricial or not, according to the depth of the lesion. When there are cicatrices, these are round, smooth and regular.

Spots and cicatrices are equally pigmented at first, but after a time the pigmentation gradually fades from the centre to the periphery. There is soon nothing left of the original lesion, or only a very superficial, white, smooth cicatrix, very slightly depressed. In some cases, however, ulcerated syphilides have deep, indelible cicatrices, but these are always round, smooth and regular, like the simple cicatricial spots.

Having described the cutaneous syphilides, we must now consider the syphilitic lesions of the cutaneous appendages—the hair and nails.

Changes in the hair have already been mentioned (p. 417). In the same way that the syphilitic infection causes nutritive changes in the hair follicles, leading to a special form of alopecia, so may the nails be affected during the secondary period of syphilis. The nails may be affected by ungual lesions (*onyxis*) and periungual lesions (*perionyxis*).

Onyxis.—The various forms of onyxis are all trophic changes, resulting from disturbance in the nutrition of the nail, produced by the syphilitic toxi-infection.

As in all general infective diseases, there may be a *transverse furrow* resulting from arrest in the nutrition of the nail. In the early period of syphilis the free border of the nail is often *brittle*. In other cases the nail becomes *thickened*, rough and uneven, especially at the free border. This lesion and the preceding one are temporary. Of greater importance are the two following lesions: *ulceration*, and *detachment* of the nail.

Ulceration of the nail is a loss of substance in the region of the lunula, a kind of dry ulceration situated on the bed of the nail, with irregular borders and an oval shape.

Detachment of the nail may be complete or partial. In partial and temporary detachment, the nail becomes detached from its bed from the free border towards the root, for about a third or half its length. The lesion is soon repaired by growth of the nail. In total detachment the nail is completely separated from its bed, and falls off. A new nail grows, regular or irregular, sometimes before the first is completely separated.

Perionyxis.—This is a lesion affecting the soft parts round the nail. Fournier distinguishes three forms: dry, inflammatory and ulcerative perionyxis.

Dry perionyxis consists simply in horny thickening of the epidermis surrounding the nail, forming a hard horny swelling, which may become excoriated, fissured and painful.

Inflammatory perionyxis forms a painful swelling round the nail,

resembling a whitlow; but it is always hard and firm and never suppurates. Sometimes the nail falls off.

Ulcerative perionyxis may be secondary to the preceding form, or may occur by itself. It forms a deep ulcer, partly or completely surrounding the nail in the form of a trench, with raised borders and a sanious base, sometimes vegetating. This lesion may be complicated by inflammation when it affects the toes, especially the great toe. The nail always falls off. Under treatment, the nail grows again partly or completely, but the end of the digit is usually deformed.

Local treatment is necessary in the inflammatory and ulcerative forms of perionyxis. It includes local baths, mercurial ointment, iodoform, tincture of iodine, or solution of nitrate of silver (5 per 100). When the nail is partly detached, it should be removed.

The Pigmentary Syphilide.

This is a peculiar form of cutaneous lesion observed during the secondary period. According to Fournier, the pigmentary syphilide is a primary pigmentation of the skin of the neck, which develops progressively without any preceding macules or papules, in the form of light brown spots which extend into pigmented bands, interlacing at different angles and enclosing islets of normal skin. The lesion forms a sort of yellowish network, similar to chloasma in colour, with small areas of normally coloured skin in its meshes.

The pigmentary syphilide may take a few weeks or a few months to develop, but when fully developed it lasts indefinitely. It does not appear to be influenced by treatment, and for this reason Fournier includes it among the parasyphilitic lesions.

To demonstrate that the areas of skin enclosed in the meshes of the pigmentary network are not achromic, as some authors have stated, but have the same colour as the rest of the skin, Fournier employs the following method:—A card with a hole in it is placed over the pigmentary syphilide so that the hole corresponds to one of the portions of normal skin. The pigmented area is thus concealed, and the eye can judge that the colour of the unpigmented area is the same as that of the rest of the skin of the neck.

In fact, the true pigmentary syphilide must be distinguished from lesions formed by a mixture of pigmentation and depigmentation, which are sometimes observed after papular syphilides, especially on the neck. This lesion, which I have named *peri- and post-papular leuco-melanodermia*, differs completely from the pigmentary syphilide in the fact that, instead of being formed by areas of normal skin enclosed in a pigmentary network, it is formed by white decolorised spots, to a certain extent cicatricial, which

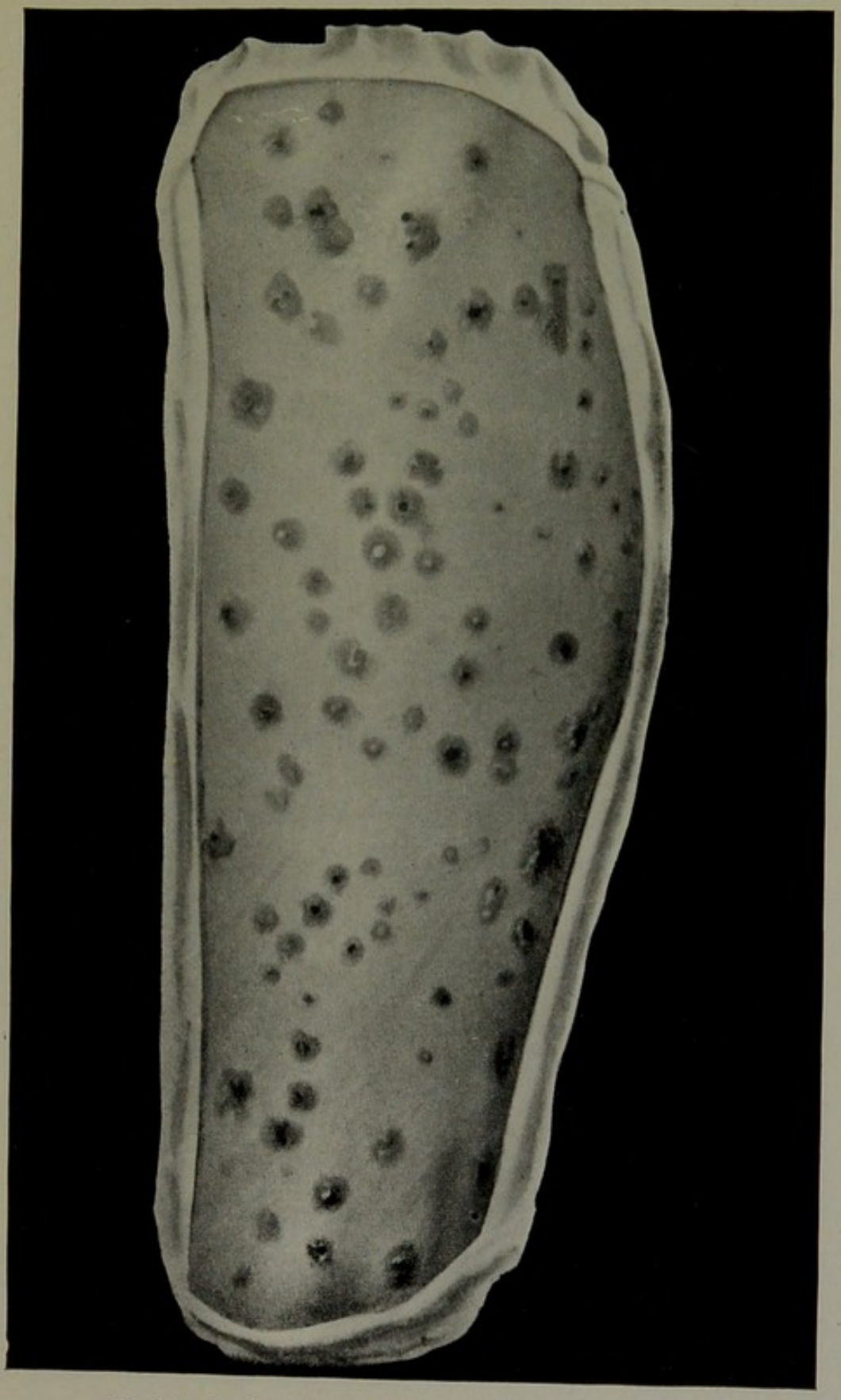


Fig. 188.—Papulo-crustate syphilide. (St Louis Hospital Museum.)

[*To face page* 426.

succeed *in situ* papular syphilides, while the peripheral region undergoes hyperpigmentation, as often occurs in cicatrices of any kind, especially syphilitic cicatrices; this hyperpigmentation extends over nearly the whole of the neck.

From these characters it is practically impossible to confuse leuco-melanodermia with the pigmentary syphilide. As to the etiology of the latter, this is a matter of opinion. According to Fournier, the true pigmentary syphilide is a primary pigmentation; according to others, it is secondary to certain forms of roseola, which are more extensive than usual and spread to the neck. The partisans of the latter view regard the lesion, not as a simple pigmentation, but as a mixture of achromia and hyperchromia.

I think that both these views are partly correct and partly erroneous. First of all, the pigmentary syphilide must be completely separated from leuco-melanodermia; on the other hand, the pigmentary syphilide, in my experience, is not primary, but secondary to roseola. Therefore, both clinically and etiologically, there may be two different forms of pigmentary syphilide of the neck, one *macular*, the other *areolar;* both of them being distinct from leuco-melanodermia.

These two varieties, apart from differences which I shall shortly point out, have certain characters in common. Both occur in the secondary period of syphilis, but at a comparatively late date in this period; in fact, they are seldom seen before six months after the chancre. The pigmentation gradually becomes darker, passing from light brown to dark brown, or almost black in dark-skinned subjects. It generally occupies the whole extent of the neck and nape, from the chin and hair above to the clavicles below. The upper and lower limits are not clearly defined, and the pigmentation gradually fades into the normal skin. The face is always exempt, but in some cases the pigmentary syphilide may descend below the clavicles and extend to the arms, shoulders, axillæ and upper part of the thorax. Some generalised cases have even been reported, but it is doubtful if they were cases of true pigmentary syphilide.

The lesion persists indefinitely for months or years, becoming attenuated in time or disappearing spontaneously; but sometimes it persists without any change. The pigmentary syphilide is much more common in women than in men, especially in young women. It has been facetiously called "Venus's collar."

As already mentioned, the pigmentary syphilide may present two varieties, the *macular* and *areolar*.

In the *macular variety*, there are round or oval brown spots, disseminated on the skin of the neck, and quite isolated. From my own observations, this variety of the pigmentary syphilide succeeds the spots of ordinary roseola affecting the neck.

The *areolar variety* corresponds to the pigmentary syphilide described by Fournier, the characters of which have been already mentioned. This variety, in the same way as the preceding form, succeeds a special form of *annular roseola*, as I have several times observed.

To resume—apart from pigmented macules with progressive eccentric decolorisation, which are observed after papular, tubercular, pustular or crustate syphilides on all parts of the body, there are pigmentary lesions which occur after syphilitic eruptions on the neck, and are usually limited to this region. These present the following types:—

(1) The *true pigmentary syphilide* (formerly called primary) which succeeds roseola of the neck. If the spots of the roseola are of the usual round form, the pigmentary syphilide is *macular;* if the roseola is annular, the pigmentary syphilide is *areolar.*

(2) *Peri- and post-papular leuco-melanodermia,* which succeeds a papular syphilide of the neck, and is a mixture of *depigmentation* and *hyperpigmentation,* the former occupying the situation of the papular elements, the latter surrounding it.

It is the first type only, especially its areolar variety, which corresponds to the pigmentary syphilide of Fournier.

Treatment.—The local treatment of cutaneous syphilides is usually of little importance. The best applications for localised syphilides, tubercular, squamous or ulcerative, are mercurial plasters and calomel ointment (10 per 100).

MUCOUS SYPHILIDES.

Mucous syphilides, commonly known as *mucous patches,* are moist in comparison with the cutaneous syphilides, which are dry. The *Spirochæta pallida* is almost constantly found in these lesions.

Mucous patches are not exclusively localised to the mucous membranes, they also occur on the skin, with the same appearance as on the mucous membrane; both forms are, in fact, moist and exudative syphilides.

Fournier describes four types of mucous syphilides:—

(1) The *erosive type,* which is very superficial.

(2) The *ulcerative type,* which is deeper than the preceding and extends to the dermis.

(3) The *papulo-erosive type,* which corresponds to the papular cutaneous syphilide; but the latter is a dry lesion, while the papulo-erosive mucous syphilide is moist. This type is also called *condyloma latum.*

(4) The *hypertrophic* or *vegetating type,* which is only an exaggeration of the preceding type.

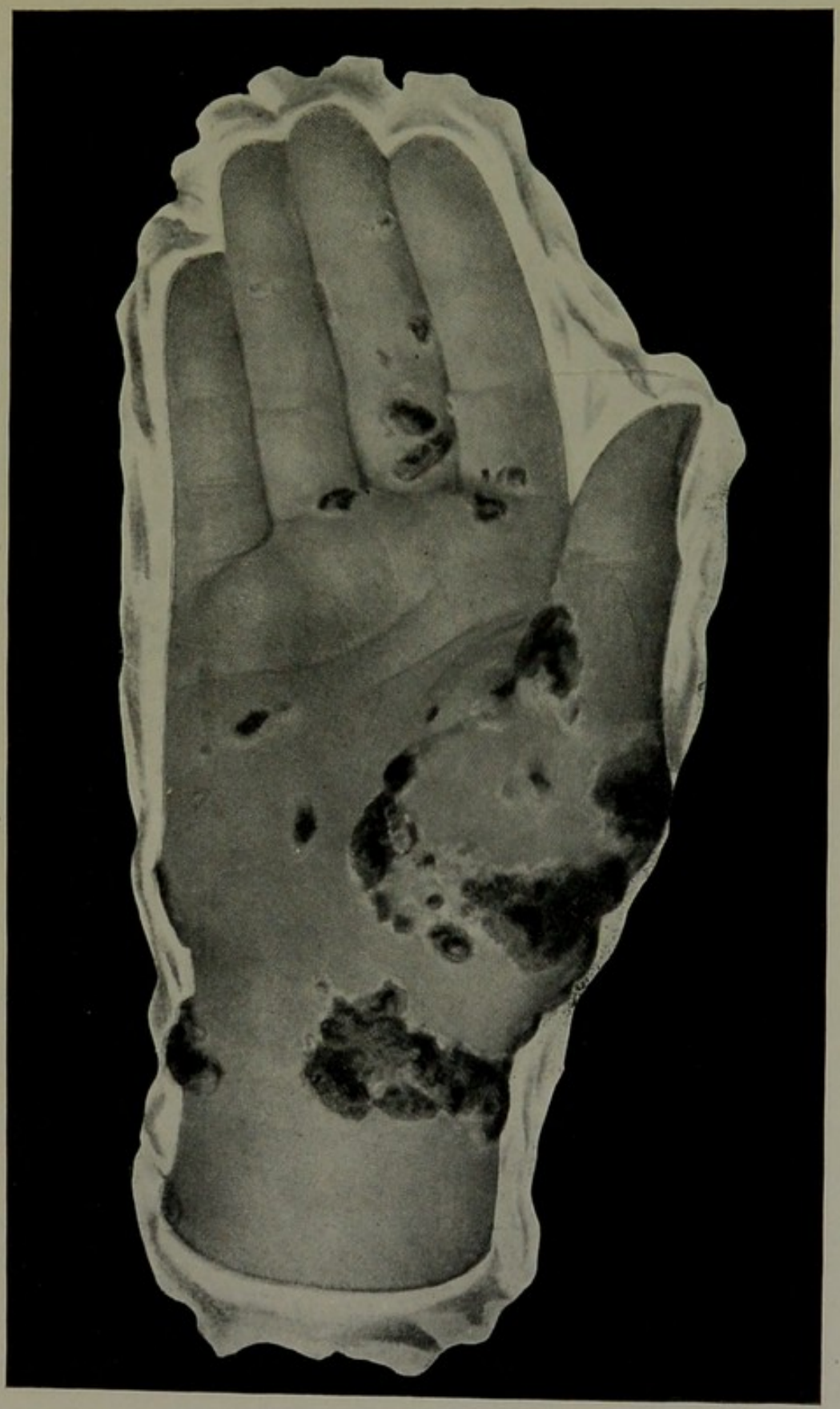


FIG. 189.—Palmar papulo-crustate syphilide. (St Louis Hospital Museum.)

[*To face page* 428.

In these four types the lesions are always moist. They occur on the mucous membranes, and also on the skin in regions where the epidermis is thin and approaches in structure the epithelium of mucous membranes, as on the lips, anus, glans penis and prepuce. Mucous patches may also occur on the ordinary skin when two cutaneous surfaces are apposed to each other, and in all regions where the cutaneous secretions are abundant and cause continual moistening.

Mucous patches do not occur on all mucous membranes. They occur exclusively on the dermo-papillary mucous membranes, which are derived from the external layer of the blastoderm (epiblast), like the skin itself, and present the same histological structure as the skin. The dermo-papillary mucous membranes include those of the lips, mouth and pharynx, tongue, eyelids, larynx and nasal fossæ. Mucous patches are absent on all the other mucous membranes which are derived from the internal layer of the blastoderm (hypoblast). The pathological differences in mucous membranes derived from the epiblast and hypoblast are thus in accord with embryology.

The regions of the skin where mucous patches occur are: the cutaneous covering of the lips, the glans penis and prepuce, vulva, anus, vagina and cervix uteri, the perineum, inner surface of the thighs and inguinal folds, the interdigital spaces of the toes, the umbilicus, axillæ and submammary region.

Mucous patches are always early manifestations of syphilis, and are the first lesions to be looked for after the roseola. They sometimes appear at the same time as the roseola, rarely before. They heal quickly, but are apt to recur during the first year or two, and may appear again long after their first disappearance, eight or ten years after the onset of syphilis. I have seen mucous patches in a case of syphilis after eighteen years. There is one region where they recur with great pertinacity, this is the mucous membrane of the tongue. In the same way as there are some patients who suffer for years from palmar syphilides, so there are others who are subject to mucous patches of the tongue for several years, in spite of treatment. This explains why syphilis is often transmitted by the mouth. In fact, the mucous patch is the usual source of syphilitic contagion. The chancre is a lesion of short duration, during the evolution of which the patient usually avoids sexual connection: the mucous patch is an insignificant lesion which recurs indefinitely, and patients who would hesitate to transmit syphilis by a chancre often convey it unconsciously through the medium of mucous patches.

Sometimes mucous patches recur without any obvious cause; in other cases, recurrence is favoured by accessory causes which set up constant irritation of the skin or mucous membrane. For example, on the genital organs, want of cleanliness is the chief cause of

recurrent mucous patches, and this is why they are more common in women, on account of the frequent presence of vaginal discharge. In the mouth and throat, the most common cause of recurrence is the use of tobacco. This habit should be discontinued during the first years of syphilis, not only for the benefit of the patient, but also so as to avoid the danger of conveying infection to others by the constant occurrence of fresh mucous patches.

Want of cleanliness and the use of tobacco are, therefore, the most usual causes of the recurrence and persistence of mucous patches. This accounts for the frequency of these lesions in the mouth and throat, and on the anal and genital regions.

We will now study the four types of mucous patches and their peculiarities in the different situations in which they occur.

I. **The Erosive Mucous Patch.**—This is the most common form, and the one that occurs in ordinary cases of syphilis. It is a small superficial erosion of variable shape, round, oval or irregular, sometimes linear or fissured, especially on the borders of the tongue and lips. The colour may be red, gray, or opaline. In fact, it varies greatly in appearance, and has different characters in different situations.

(1) *The mouth and throat.*—The mucous patch is usually bright red, and generally varies in size from a lentil to a threepenny piece. Its size depends on the degree of secondary irritation, which increases the extent of the lesion. Sometimes it is a small insignificant lesion, which heals rapidly and may not be noticed. Its usual situations are the tongue, tonsils, and inner surface of the lips.

The colour of the mucous patch is not always red, but varies in colour according to its situation. On the tonsils and lips it is often grayish or opaline. On the tonsils, this gray colour may present another aspect, by the formation of a *diphtheroid exudation.* Another variety met with in the mouth is the *fissured* mucous patch. This is a linear erosion, or rhagades, chiefly observed on the borders of the tongue and the commissures of the lips. Lastly, there is a special variety which occurs on the back of the tongue, in the form of a flat, smooth erosion, where the papillæ seem to have been destroyed. This is sometimes called a bald patch.

To resume—mucous patches of the mouth and throat may present five different aspects: a red erosion, an opaline erosion, a diphtheroid erosion, a fissured erosion, and a bald patch.

Diagnosis. — Mucous patches of the mouth and throat must be diagnosed from herpes, aphthæ and diphtheria.

Herpes is a painful lesion, while mucous patches are usually little sensitive, except when multiple and extensive. Bucco-pharyngeal herpes is often accompanied by a characteristic eruption round the mouth. Lastly, herpetic erosions are rounder and more regular than mucous patches, and soon heal spontaneously.

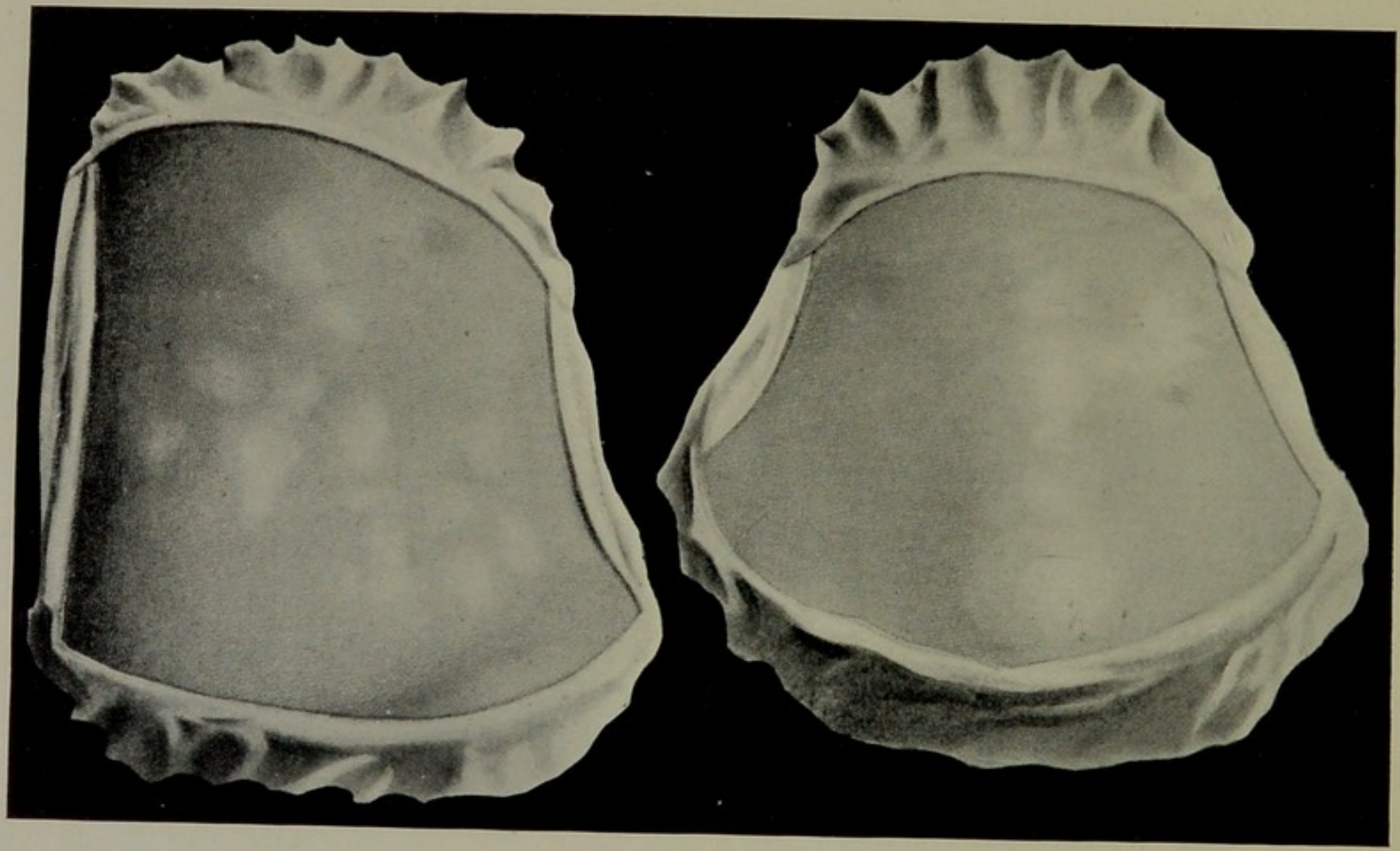


Fig. 190.—Post-papular leuco-melanodermia of the neck.

Fig. 191.—Pigmentary syphilide of the neck.

(St Louis Hospital Museum.)

[*To face page* 430.

The diagnosis between herpes and mucous patches is important, for some syphilitics are affected with recurrent herpes during the first years of the disease, the erosions of which occur on the borders of the tongue and are liable to recur on the least irritation. This peculiarity must be borne in mind, in order to avoid errors in diagnosis.

Aphthæ are painful, punched-out ulcerations, covered with a grayish white exudation, and accompanied by fever and gastric disturbance.

Diphtheria differs from the diphtheroid mucous patch in the presence of fever and severe general symptoms. However, in some cases these symptoms are present in syphilis, and the diagnosis may then be difficult. Numerous errors in diagnosis have been committed. In these cases a bacteriological examination should be made for the Klebs-Loeffler bacillus.

(2) *The ano-genital region.*—On the *vulva*, mucous patches may occur on the inner surface of the labia majora, labia minora, and vestibule, etc. They are superficial, flat, red erosions, varying in size from a lentil to a threepenny piece, resembling simple excoriations. They are usually associated with other types of mucous patches, especially the papular type, which facilitates diagnosis.

Mucous patches often occur on the *cervix uteri*, where they may be easily mistaken for common erosions. They can be recognised by their eccentric situation with regard to the external os, and their circular or semicircular form.

On the *male genital organs* the erosive type is the most common. The mucous patches may be situated in the balano-preputial furrow, on the glans, or on the inner surface of the prepuce. They are small, round, red erosions.

On the *anus*, the erosive type is almost the only one observed. In this situation they form superficial, linear erosions or fissures. The anus must be stretched, in order to see them properly.

Diagnosis.—Mucous patches on the male and female genitals must be diagnosed from *herpes*. Mucous patches are always insignificant erosions, while herpetic ulcers are sometimes cup-shaped erosions, isolated and forming distinct circles, sometimes aggregated to form larger ulcerations, which have a polycyclic outline. Anal mucous patches are painful, like all fissures of the anus, but less painful than simple *fissure of the anus*, which causes intolerable pain during defæcation. Anal mucous patches differ from *eczema* by the absence of inflammation. The fissures which accompany eczema of the anus are surrounded by a pruriginous inflammatory zone, with exudation or vesicles.

II. **The Ulcerative Mucous Patch.**—This lesion is no longer a simple erosion, like the preceding type, but an ulceration which

affects the dermis to a greater or less extent, but never so deeply as tertiary ulceration. The borders are soft, and not infiltrated. The absence of induration and infiltration distinguishes them from syphilitic chancre and tertiary ulcerations.

Ulcerative mucous patches vary in size from a lentil to a sixpence; they are generally larger than erosive mucous patches. Their colour is usually red, sometimes gray or yellow. This type is less common than the erosive form, and also occurs later. Like the erosive type, it presents different characters in different regions.

(1) *The mouth and throat.*—In this region, ulcerative mucous patches occur inside the lips, on the borders of the tongue, on the pillars of the fauces and soft palate, in the form of red or gray ulcers, varying in size and depth.

(2) *The ano-genital region.*—On the *vulva*, ulcerative mucous patches are rare, but they sometimes occur on the inner surface of the labia majora, on the labia minora and vestibule. They are always multiple, and vary in size from a lentil to a sixpence. This type of mucous patch is rare on the male genitals, and still more rare on the anus.

Diagnosis.—Ulcerative mucous patches on the borders of the tongue may be mistaken for *dental ulcers*, due to the irritation of carious teeth. But the mucous patch is seldom isolated; there are usually other mucous patches in other parts of the mouth. Moreover, the mucous patch is almost painless, while the dental ulcer is painful. In doubtful cases, for instance in a syphilitic with dental caries, the diagnosis is settled by the evolution of the lesion after removal of the tooth.

Ulcerative mucous patches of the vulva are often difficult to diagnose from *simple chancre*. The diagnosis is based on the following points: in simple chancre the adenopathy is monoglandular and often suppurative; in syphilis it is polyglandular, and hardly ever suppurative. In simple chancre there are secondary ulcers (satellite chancres), due to auto-inoculation; mucous patches do not multiply, while the number of simple chancres often increases by inoculation of the neighbouring parts. The ulcer of simple chancre has more sharply defined, punched-out borders, often undermined. Simple chancre is yellow and suppurating; the mucous patch is red and exudative, but not suppurative. In doubtful cases, examination should be made for the bacillus of Ducrey and the *Spirochæta pallida*.

III. **The Papular or Papulo-erosive Mucous Patch.**—This type differs only from the cutaneous papular syphilide in the papule being moist and excoriated. The histological structure is identical in the two cases, viz., embryonic hyperplasia of the papillæ and superficial part of the dermis.

The papulo-erosive mucous patch is a flat papule, the size of a lentil, sometimes as large as a sixpence. Its surface is denuded of epithelium, smooth, and red or whitish in colour.

(1) *The mouth and throat.*—In this region the papulo-erosive type of mucous patch is rather rare. When it occurs, it forms a flat papule, excoriated or superficially ulcerated. Sometimes it is quite flat and smooth, sometimes irregular and vegetating.

(2) *The ano-genital region.*—The *vulva* is the seat of predilection for papular mucous patches, which are also called *condylomata lata* in this situation. They occur on any part of the vulvar mucous membrane; on the inner surface of the labia majora, on the labia minora, sheath of the clitoris, vestibule and fourchette. They are usually associated with dry cutaneous papules, situated on the cutaneous part of the labia majora and neighbouring parts; the groins, inner side of the thighs, perineum and anus. In fact, as already mentioned, the cutaneous papule and the papular mucous patch are formed by the same lesion, with the only difference that the lesion is dry in the one case, moist and eroded in the other. Moreover, even on the cutaneous surface, the papules are often moist and excoriated and assume the papulo-erosive type, on account of the constant moistures of these regions, especially in uncleanly women.

These mucous patches, or condylomata, are flat papules, with a moist surface and pink or gray colour. They are always multiple, sometimes isolated, but often confluent, forming extensive patches with polycyclic borders. The exudation from these patches has a characteristic fœtid odour. In spite of their extent, these patches are easily cured by internal treatment, combined with simple local treatment and cleanliness.

On the *male genital organs*, papular mucous patches are much more rare, and never so large and confluent as on the female genitals. They may occur on the glans and penis, balano-preputial furrow and scrotum. Sometimes they are grouped behind the scrotum along the median raphe. In the same regions a special variety of mucous patch may be found, of a grayish colour, due to the presence of a slight diphtheroid exudation.

Around the *anus*, in the perineal and intergluteal regions, papulo-erosive mucous patches may also occur. This localisation is common in women, and is associated with condylomata of the vulva. In man, peri-anal mucous patches are much less common.

IV. **The Hypertrophic Mucous Patch.** — This is only an exaggeration of the preceding form. The elementary lesion is a large papule, possessing the same characters and the same histological structure as the papular mucous patch, from which it differs only in size. There is, therefore, no essential difference between the two forms.

(1) *The mouth and throat.*—In this region, the hypertrophic mucous patch is quite exceptional, and always succeeds papulo-erosive mucous patches which have been subjected to irritation by tobacco, etc. The papules are large and sometimes vegetating. They occur almost exclusively on the posterior part of the tongue, which presents a characteristic embossed appearance.

(2) *The ano-genital region.*—The *vulva* and its neighbourhood are the most common situations of the hypertrophic mucous patch. It occurs in dirty women, in the form of sessile or vegetating tumours, which are, however, never so arborescent as simple vegetations. The lesions may be isolated, or agglomerated in masses, forming extensive raised patches. The exudation from these patches has a fœtid odour.

On the *male genital organs* hypertrophic mucous patches are much less common. They seldom occur on the penis, but sometimes on the scrotum and in the genito-crural folds. Sometimes they form vegetating, indurated masses, which may give the scrotum an elephantiasic appearance. As in women, these lesions are due to want of cleanliness.

Around the *anus*, hypertrophic mucous patches occur in association with similar lesions about the vulva or scrotum, but they never occur on the mucous membrane of the anus. They sometimes form large vegetating masses or fungoid tumours around the anus.

Diagnosis.—Hypertrophic mucous patches must be diagnosed from simple *vegetations*. The latter are more subdivided or digitated than mucous patches, and are not exudative. In doubtful cases other syphilitic lesions must be looked for, especially in the mouth.

Treatment.—Mucous patches of the *mouth and throat* should be cauterised with a solution of nitrate of silver. The silver nitrate crayon should never be used, on account of the danger of conveying contagion to others. I have seen a case in which syphilis was contracted in this way. The silver nitrate solution (20 per 100) should be applied every three or four days. When the mucous patches are multiple and superficial, tincture of iodine may be applied. Mouth washes of chlorate of potash or diluted oxygenated water are also useful.

In mucous patches of the *ano-genital region*, attention to cleanliness and the application of a dusting powder of zinc oxide and calomel (2 to 1) are usually sufficient. Cauterisation is seldom necessary, except in the case of vegetating condylomata, which may be touched with the above solution of nitrate of silver, or in rebellious cases with acid nitrate of mercury. An ointment of calomel (10 per 100) is useful in some cases of erosive or ulcerated mucous patches, especially when situated about the anus and glans penis.

GUMMATOUS SYPHILIDES.

So far, we have studied the manifestations of syphilis on the skin and dermo-papillary mucous membranes, the lesions being situated in the dermis and papillary body.

There are other lesions which commence in the subcutaneous tissue, and constitute syphilitic neoplasms. These are all localised formations, primarily perivascular, like all neoplasms of infective origin, and formed by proliferation of the connective tissue. This proliferation is irritative in nature, and due to the action of the syphilitic virus on a vascular territory. Syphilitic neoplasms are, in fact, primarily constituted by an accumulation of embryonic cells. When the neoplasm is formed, the embryonic elements follow two different lines of evolution: (1) fibrous transformation; (2) necrobiosis.

In the first case, the embryonic elements develop into connective tissue. The nuclei become cells, the cells give rise to fibres, which form fibrous bundles and finally constitute fibrous tissue or sclerosis.

In the second case, the newly formed cells are insufficiently nourished, owing to their being accumulated in considerable numbers and pressed against each other. They then undergo a cadaveric process, which ends in necrobiosis, softening, and destruction of the cellular elements. This necrobiotic evolution characterises the *syphilitic gumma.* In both cases the lesion commences round a vessel; the vascular wall is the point of origin of the neoplasm.

To resume—all syphilitic neoplasms are formed primarily by an accumulation of embryonic elements around a vessel, resulting from irritation of the vascular wall and perivascular tissue by the syphilitic virus. According as the nutrition of these elements is perfect or imperfect, the evolution tends either to *sclerosis,* or to *necrobiosis* or *gumma.*

Some syphilitic neoplasms are exclusively sclerous, others exclusively gummatous; but the two processes are often combined, resulting in the formation of *sclero-gummatous tissue.*

When the syphilitic neoplasia occupies the interstitial tissue of the viscera, such as the liver and lung, the lesion is nearly always sclero-gummatous. The same process occurs in the tongue, constituting *sclero-gummatous glossitis.*

Sclero-gummatous formations also occur in the skin, especially in the lips, giving rise to *hypertrophic syphiloma of the lips,* sometimes called *syphilitic leontiasis.* This lesion, which will be referred to later, is characterised by great hypertrophy of the lips, which are thickened, infiltrated and fibrous, with ulcerations in places resulting from

softening of small gummas mixed with sclerous tissue. Syphilitic sclerosis, whether in the lips, tongue or viscera, is much more rebellious to treatment than gumma.

In accordance with the preceding facts, we may define the **syphilitic gumma** or **gummatous syphilide** as follows:—The syphilitic gumma is a localised nodosity, formed by an accumulation of embryonic cells, which die and disintegrate, causing softening of the nodosity, with a tendency to open externally.

The gumma is always a late lesion, and belongs to the tertiary period of syphilis. According to Fournier's statistics, gummas are usually observed after the third year of the disease; but they may occur ten, twenty or forty years after infection (even forty-five years in one of my cases).

The gumma is regarded as the type of tertiary lesions. However, we must remember that, strictly speaking, there are no secondary and tertiary lesions, but only a secondary period, during which the lesions are generalised and disseminated, and a tertiary period, in which the lesions are localised and grouped. This explains why, in certain cases, gummas may appear at an early stage, either on account of special virulence of the disease or want of resistance in the patient. In fact, gummas may occasionally develop during the second year, or even earlier. I have seen a case in which a periosteal gumma of the orbit developed before the chancre had cicatrised, and at the same time as the roseola; another case of retro-orbital gumma, which developed during the secondary period, soon after the roseola; and a third case, in which a frontal gumma appeared at the same time as a papular roseola.

The gumma may thus be an early manifestation, which sometimes appears before the chancre has healed; but, as a rule, it is a late lesion and a manifestation of tertiary syphilis.

The *Spirochæta pallida* is rarely found in the lesions of tertiary syphilis, especially in gummas. This is in accordance with the usual non-contagiousness of tertiary lesions. According to Bosc, however, the spirochætes are present at first, but disappear later. Schaudinn considered that they might be present in gummas in the form of a resting stage. In the present state of our knowledge, it appears to me that the microbe of syphilis should not be met with exclusively in certain eruptive elements or syphilitic formations, to the exclusion of other lesions, but that if it exists in certain forms of eruption in the secondary period, it may be absent in similar elements during the tertiary period. In other words, as already mentioned, there are no secondary or tertiary elements, but only secondary and tertiary periods, and, very probably, the spirochætes which are present in all lesions of the secondary period, at the time when syphilis is generalised and still virulent, are absent or few in number in the

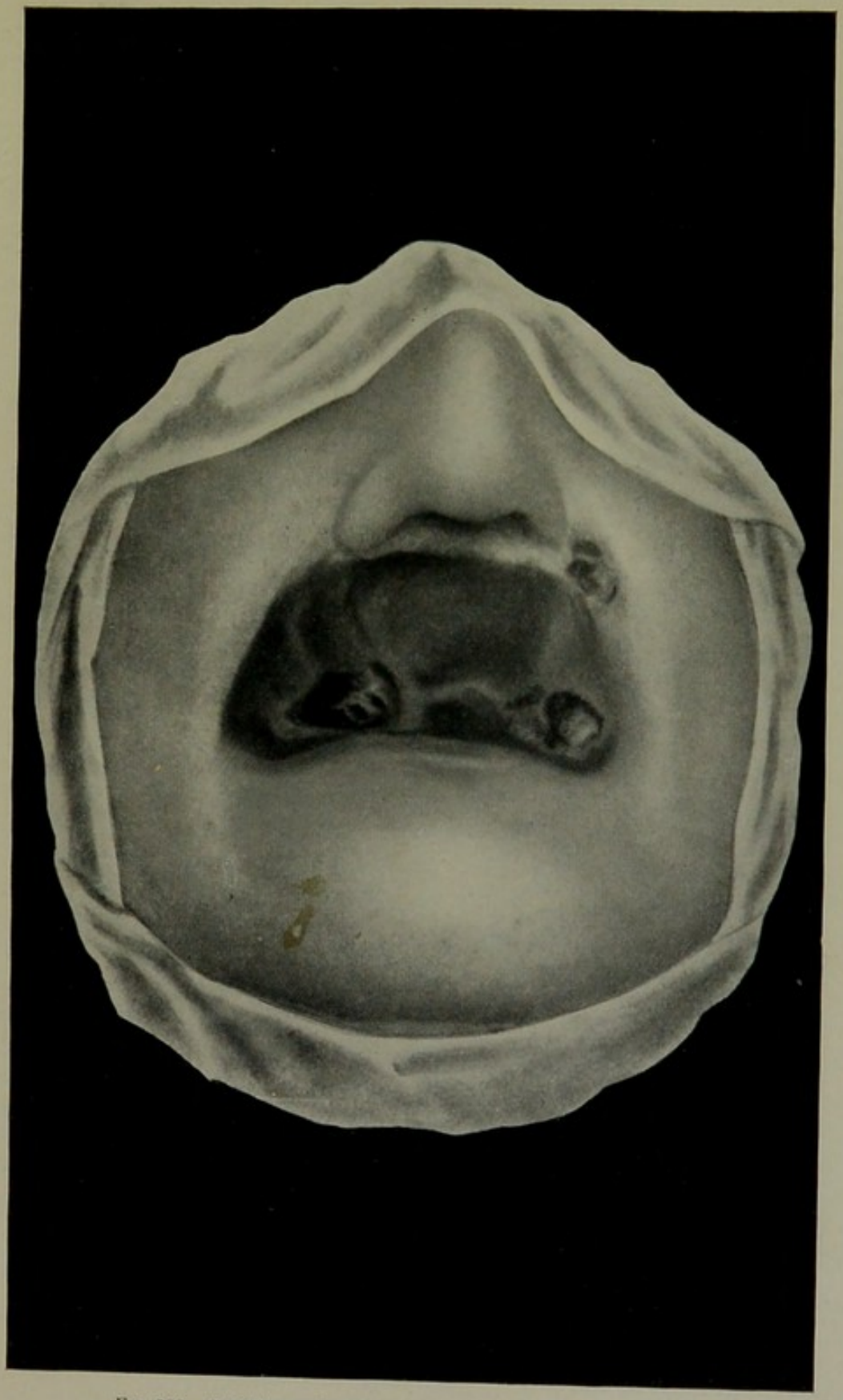


Fig. 192.—Syphiloma of upper lip. (St Louis Hospital Museum.)

[*To face page* 436.

same kind of lesion when this occurs in the tertiary period, when the disease is attenuated.

Gummas may be situated wherever there is connective tissue; in the subcutaneous and submucous tissues, in the interstitial connective tissue of the viscera, meninges, periosteum, etc.

Subcutaneous Gumma.—This may occur in any region of the skin, but has a predilection for the lower limbs, in the same way that submucous gumma has a predilection for the soft palate. In both cases, whether subcutaneous or submucous, the syphilitic neoplasia begins in the subcutaneous tissue and not in the dermis, and thus differs from the syphilides which we have previously studied, which always begin in the papillary layer of the dermis.

Fournier describes four stages in the evolution of syphilitic gumma: (1) maturation; (2) softening; (3) ulceration; (4) cicatrisation.

(1) *The stage of maturation.* — The mature gumma forms a nodosity or tumour. It varies in size from a pea to an egg, or larger; but the average size is that of a nut. It is generally circumscribed, except in cases where an accumulation of neoplasms gives rise to more or less extensive infiltration. In the latter case, the gummas are often associated with sclerosis, forming hard, raised, irregular, nodular patches, each nodule corresponding to a gumma. These sclero-gummatous infiltrations may occur in any part of the body, but they are most commonly met with in the lips, constituting the *hypertrophic* or *diffuse syphiloma*, or *syphilitic leontiasis.*

This *sclero-gummatous infiltration of the lips* appears to be sometimes caused by the abuse of tobacco, or by affections of the teeth. It is rather a rare lesion, usually appearing at an advanced stage of syphilis; between the tenth and fifteenth years. It consists of a diffuse infiltration of the skin and subcutaneous tissue, affecting one or both lips, but more often the lower lip only. The lip is thickened, and may be twice or three times its normal size. The lip retains its normal shape; it simply projects forwards, and may even have a smooth surface. When both lips are affected, they project forwards, causing a deformity which somewhat resembles a lion's muzzle; hence the name syphilitic leontiasis. As the neoplasm distends the mucous membrane more easily than the skin, the labial mucous membrane covers nearly all the syphiloma which is visible. The mucous membrane is dark red and papillomatous, while the skin of the lip preserves its normal colour. There are often islets of gummatous infiltration, which soften and open externally, giving rise to ulcerations. Labial syphiloma is often accompanied by lesions of the tongue and inner surface of the cheeks; mucous patches, tertiary ulcers, or leucoplakia, but especially deep sclerous glossitis. The lesion is essentially chronic, taking months to develop and persisting almost indefinitely. It is very rebellious to treatment;

the lip seldom regains its normal size, and usually remains more or less hypertrophied.

To return to the general description of the *gumma*. This is at first hard and firm, usually painless, and is not accompanied by inflammatory reaction. In some cases, however, the gumma may be painful; for instance, when situated over a bone, such as the frontal bone, or when it is adjacent to nerves. The period of maturity is of very variable duration; a gumma may exist for a long time before its nature is recognised.

(2) *The stage of softening.*—After a variable time, the gumma softens and becomes fluctuating, as if it contained pus; but when punctured (which, however, should never be done when the nature of the lesion has been recognised), no pus escapes, but a thick, viscous, sanious liquid, sometimes yellow and puriform, but never actually purulent. The contents of gummas have the same characters in both acquired and congenital syphilis, and these characters are useful in diagnosing congenital syphilitic gummas from tuberculous lesions. It is, therefore, important to remember the characters of the gummatous liquid; a viscous liquid, sometimes yellow, sometimes puriform, but never actually purulent. It is the viscous and gummy nature of this liquid which has given these lesions the name of gumma.

(3) *The stage of ulceration.*—The ulceration of gumma differs from that of abscess. The skin becomes perforated in several places, through which the gummy liquid exudes; this liquid gradually becomes more puriform on account of secondary infection. Microscopically, the contents of the gumma are found to consist of degenerated cells and fatty granules arising from disintegration of cellular elements. This shows that the gumma is an embryonic neoplasm which has undergone necrobiosis.

After evacuation of its contents, the gumma does not disappear completely, for its cavity is surrounded by a fibrous shell. This fibrous shell exists even in cases where the syphilitic neoplasm is purely gummatous, and not associated with sclerosis. Around the local embryonic accumulation which constitutes the gumma there is always peripheral hyperplasia, which forms a fibrous envelope around the neoplasm, and remains after evacuation of the contents.

The original small openings become united to form a larger orifice, which gradually extends at its borders. By this opening, not only is grumous liquid evacuated, but also an actual core, comparable to that of a boil. This core consists of necrosed connective tissue, and resembles a piece of lint soaked in pus. It is a dermic slough, which is either eliminated in layers, or becomes disintegrated and is gradually expelled in fragments.

After the contents have been eliminated, the gumma leaves an

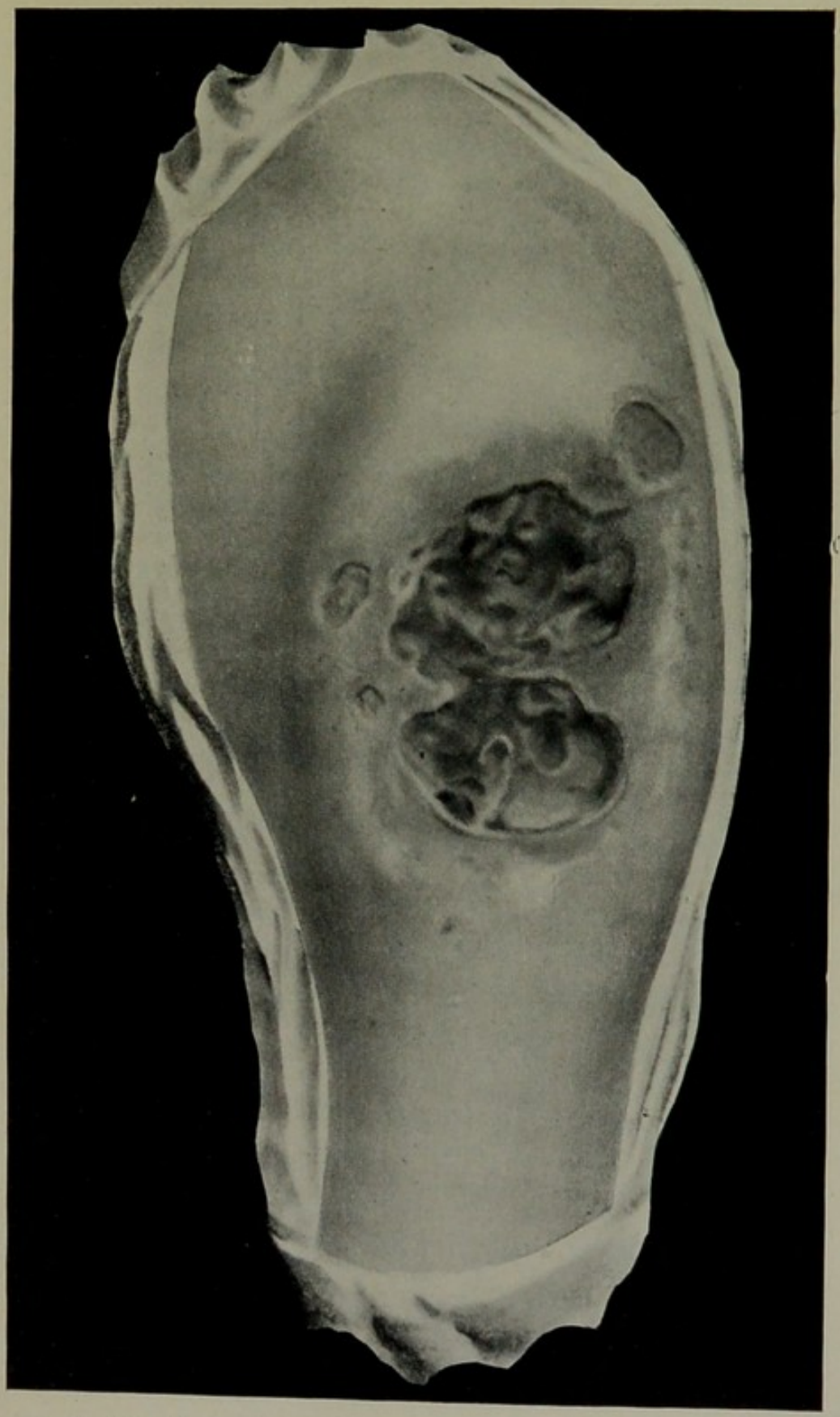


Fig. 193.—Ulcerated gumma of the shoulder. (St Louis Hospital Museum.)

[*To face page* 438.

ulceration, which is deep at first, but becomes gradually shallower, constituting the gummatous ulcer, the characters of which we have now to consider.

Gummatous Ulcer.—First of all, it is necessary to describe the peculiarities of ulcerated gummatous infiltration, or hypertrophic syphiloma.

These sclero-gummatous infiltrations open in several places, but while the necrosed tissue is eliminated, the peripheral sclerosis, due to normal evolution of irritated connective tissue, continues, and forms an indurated zone around the ulcers. This sclerosis persists much longer than the gummas themselves, on which treatment has a more rapid effect.

The *gummatous ulcer*, whether arising from a gumma or from sclero-gummatous infiltration, has the following characters:—It is circular in form, sometimes polycyclic when arising from the confluence of several neighbouring gummas. It is rather deep, not superficial like mucous patches. Its borders are usually hard, sharply punched-out, and not detached. Its base is irregular, and covered with a yellow creamy exudation. The surrounding skin is of the peculiar brown colour common to the cutaneous manifestations of syphilis, and forms a pigmented areola round the ulcer.

Gummatous ulcers, when untreated, vary considerably in duration; some heal rapidly, while others persist indefinitely. Some heal at one point while they extend in other directions; this often occurs on the legs, especially when there are varicose veins, which favour extension of the ulcer.

When gummatous ulcers are situated over bony surfaces, the destructive process may extend to the bone. This occurs in gummas situated on the frontal, presternal, and anterior tibial regions. A gumma situated over the clavicle may extend to the bone and cause spontaneous fracture.

Gummatous ulceration may also become *phagedenic*. It then extends rapidly, both superficially and deeply, in spite of treatment. This *tertiary phagedena*, like gummatous ulcers themselves, may occur on any region of the skin, but has two seats of predilection: the genital organs and the face, especially the nose. In this way, part of the face may be destroyed in spite of proper antisyphilitic treatment, which takes a long time to cure the lesions, or even to arrest their progress.

All the above characters apply equally to the gummas of congenital syphilis, which have the same clinical peculiarities, and follow the same evolution as the gummas of acquired syphilis.

(4) *The stage of cicatrisation.*—The cicatrisation of gummatous ulcers takes place in the same way as in other ulcers. The cicatrix is at first red and presents nothing peculiar, but after a time it shows

special pathognomonic characters, which are often sufficient for the retrospective diagnosis of syphilis. The red colour is soon replaced by brown pigmentation; which becomes modified in a peculiar manner. The pigmentation fades after a variable time, but in a special manner, disappearing from the centre towards the periphery, so that between the stages of uniform pigmentation and depigmentation there is an intermediate stage, when the cicatrix is white in the centre and pigmented at the periphery. This appearance may be regarded as pathognomonic of syphilis. In its last stage the cicatrix is smooth, uniform, and completely depigmented.

Such are the essential characters of the syphilitic cicatrix, which remains permanently, and can always be recognised, owing to its smooth and uniform surface; characters which do not exist in the same degree in any other lesion.

DIAGNOSIS.—(1) In the *stage of maturation*, gummas may be mistaken for many other tumours.

Lipoma differs from gumma in its doughy consistence. At this stage the gumma is hard, and only becomes doughy when it softens; at this period the skin is red and on the point of ulcerating, so that a tumour of doughy consistence cannot be a gumma if the skin covering it is normal.

Tuberculous gumma resembles syphilitic gumma very closely at this stage. Cutaneous tuberculous gumma with impending ulceration may be easily distinguished from syphilitic gumma, but the diagnosis of subcutaneous gumma is much more difficult. This often depends on the history of the case, the presence of other symptoms of tuberculosis or syphilis, or on the therapeutic test. At a later period, during the stage of ulceration, the diagnosis becomes easy, as the evolution of the two lesions is very different. The tuberculous ulceration is irregular, with detached borders, discharges a sanious liquid, and evolves in a torpid manner. The tuberculous cicatrix is irregular, bridled, and cheloidal, instead of smooth and uniform like the syphilitic cicatrix.

Cancer, in the form of disseminated cancerous nodules, may sometimes resemble gumma. But the cancerous nodules are adherent to the skin, even when the skin is intact; they are also painful, irregularly nodular, instead of round like gummas, and accompanied by enlargement of the neighbouring lymphatic glands. In doubtful cases, the therapeutic test is often necessary; but this should always be by mercury, for it has been proved that alkaline iodides hasten the evolution of epithelioma. Mercury is not contra-indicated in cases of cancer, and may sometimes even act favourably.

(2) At the *stage of ulceration*, the diagnosis of syphilitic gumma is often very difficult. It is sometimes impossible to distinguish

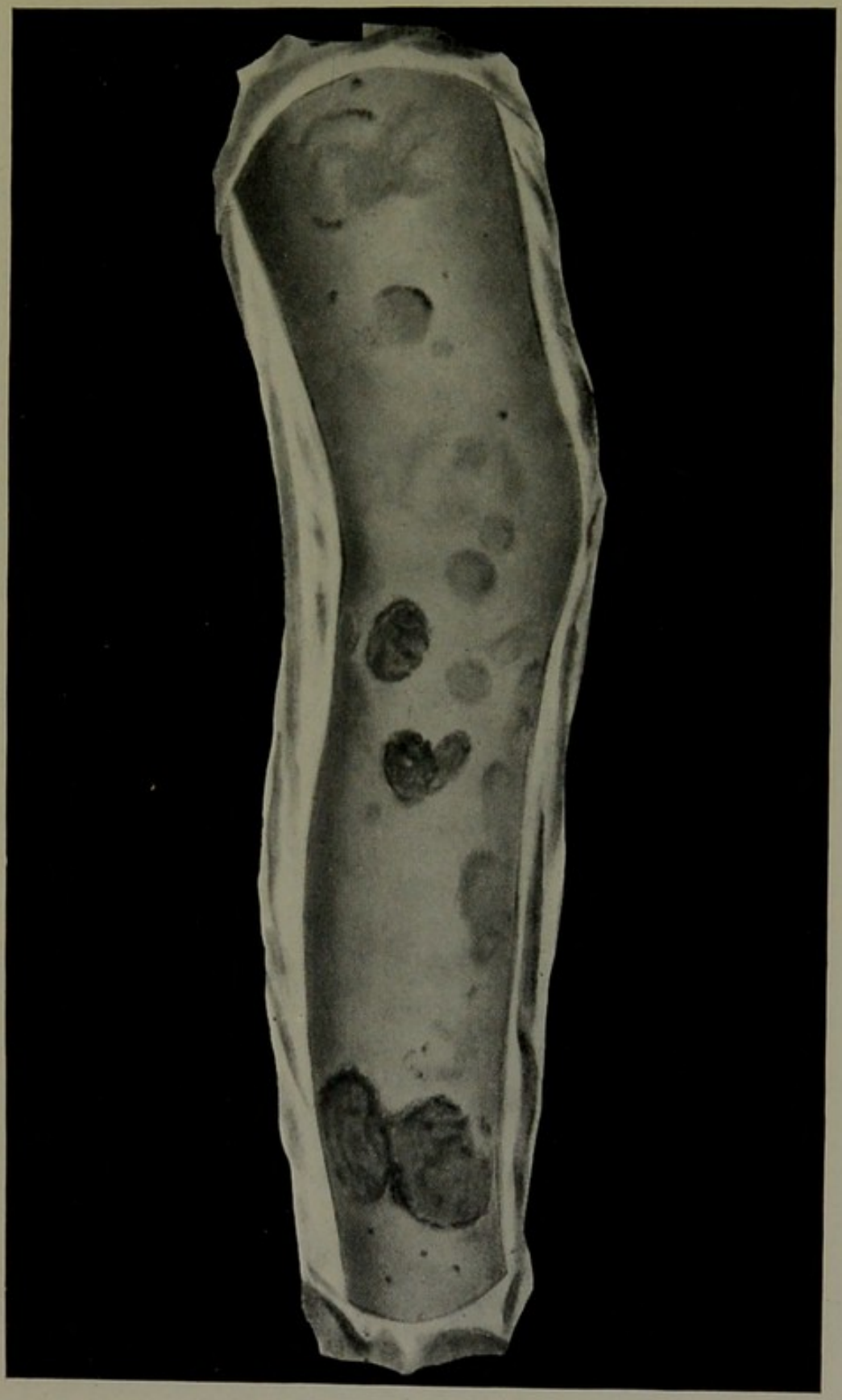


FIG. 194.—Gummatous ulcers of the leg. (St Louis Hospital Museum.)

[*To face page* 440.

syphilitic from *varicose ulcers*. The latter sometimes have an extraordinary resemblance to gummatous ulcers of the legs, and Fournier has shown that many ulcers of the legs, called varicose ulcers, are often syphilitic, and heal rapidly under specific treatment.

The distinctive characters of these two kinds of ulcer are as follows:—The gummatous ulcer has often a more *circular form* than the varicose ulcer. The syphilitic ulcer has a *brown areola;* but this pigmentation may occur around a varicose ulcer, on account of its situation on the lower limbs, where all cutaneous lesions have a tendency to become pigmented; hence this sign is not absolutely characteristic. Gummatous ulcers are often *multiple*, while there are seldom more than one or two varicose ulcers.

Therefore, if there are multiple ulcers of a circular or polycyclic form, not accompanied by varices, they may be safely diagnosed as syphilitic. But gummatous ulcers often occur in persons affected with varices. Again, gummatous ulcers are sometimes atypical, and cannot be diagnosed by their objective characters alone. In any case, mercury and iodide of potassium should be prescribed for cases of varicose ulcer occurring in persons who have had syphilis.

Lastly, certain *chancriform gummas* may be mistaken for *syphilitic chancres*, especially when situated on the glans penis or labia. The most important element in diagnosis is the absence or presence of *adenopathy*, gummas being never accompanied by glandular enlargement. The depth of the lesion is also different; gummas are much deeper than chancres.

TREATMENT.—A syphilitic gumma should never be opened nor punctured. Internally, mixed treatment is indicated. Iodide of potassium alone may cause gummata to disappear, but it has no action on the evolution of syphilis; since the presence of gummas shows that the disease is not yet cured, mercurial treatment is necessary.

The local treatment of ulcerated gummas is the same as for ordinary ulcers, and consists in antiseptic lotions and dressings. In most cases, however, moist dressings soaked in boiled water are sufficient.

GENERAL TREATMENT OF SYPHILIS.

All the cutaneous and mucous manifestations of syphilis and all its visceral manifestations, apart from some special indications, are amenable to the same general treatment.

I have no intention of analysing all the methods of treatment which have been recommended for syphilis, and shall only describe the treatment which I employ myself.

Two drugs are used in the treatment of syphilis: **mercury** and **iodide of potassium**. These two drugs have by no means the same value; iodide of potassium is only an accessory drug, an adjuvant to mercury. It has a resolvent action, not only on syphilitic neoplasms, but on neoplasms in general. Iodide of potassium is not a specific for syphilis; the only drug which is truly specific is mercury, which has a direct action on the syphilitic virus.[1]

Employed alone, mercury may be sufficient to cure all the manifestations of syphilis; iodide of potassium, on the contrary, when employed exclusively, can only cure a small number of syphilitic lesions, chiefly gummas. But gummas are generally late manifestations which appear when the syphilitic infection is already attenuated. Again, the histological structure of gummas resembles that of the neoplasms on which iodide of potassium has a manifest resolutive action. Gummas may, therefore, disappear, like other embryonic neoplasms, under the influence of iodide of potassium alone; but what must be borne in mind is that iodide does not act on the syphilitic virus itself, and consequently does not prevent the development of fresh gummas or other specific lesions. Therefore, to act against the virus, it is necessary to prescribe mercury at the same time as iodide. In fact, mercury may be prescribed alone for all syphilitic lesions.

The capital drug in the treatment of syphilis, therefore, is **mercury**.

The three principal methods of administering mercury: (1) by ingestion; (2) by inunction; (3) by subcutaneous injection.

(1) **Ingestion.**—I shall not mention all the forms of mercury which have been recommended for ingestion, but only those which I use myself.

I prefer the *soluble salts*, both for ingestion and for subcutaneous injection. There is an old pharmaceutical adage, "*Corpora non agunt nisi soluta*," and it is on account of their solubility and easy absorption that the superiority of the soluble salts depends. Insoluble salts, in order to be absorbed, must be transformed into soluble salts in the digestive tract. This transformation is very irregular, and liable to so many variations that the insoluble salts may be very imperfectly absorbed.

The soluble salts may be prescribed in the form of pills or in solution; the two salts which I employ are bichloride of mercury and neutral lactate of mercury.

Bichloride of mercury may be given in the form of pills contain-

[1] Prof. Gaucher considers the use of arsenic both useless and sometimes dangerous; "for example, the preparation of arsenic called *atoxyl*, which causes disturbance of vision and even blindness" (*Annales des Mal. Vénériennes*, June 1909).—Ed.

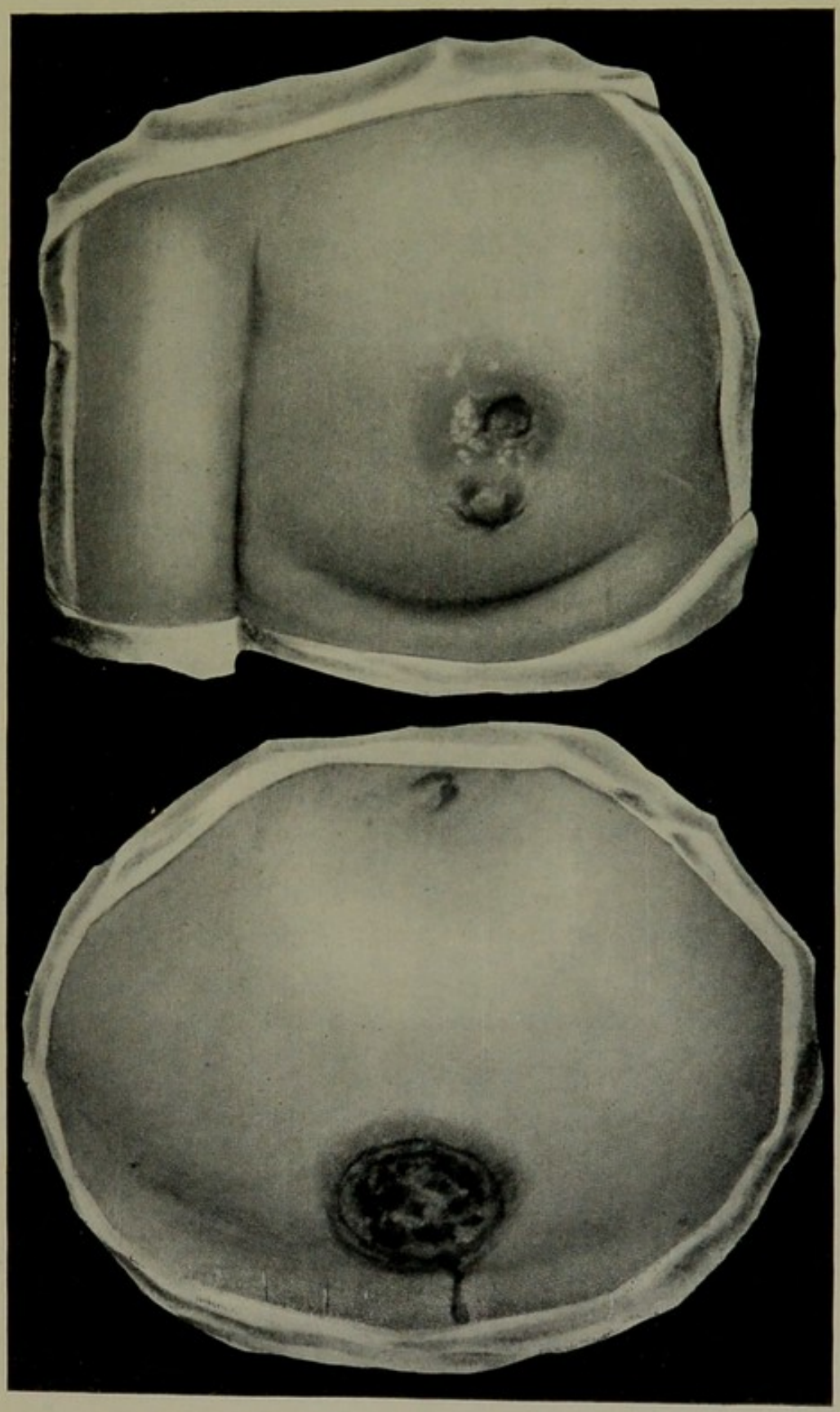


Fig. 195.—Ulcerated gumma of breast. Fig. 196.—Chancriform ulcerated gumma of abdomen.
(St Louis Hospital Museum.)

[*To face page* 442.

ing $\frac{1}{6}$ grain of the salt with an equal quantity of extract of opium. In order for pills to be easily absorbed, they should be soft, not hard as they usually become when kept for a time. Again, for an irritating substance like bichloride of mercury, the pill mass should be large enough for the drug to be distributed as much as possible when the pill becomes dissolved on the mucous membrane. For these reasons, I use the following formula :—

Bichloride of mercury Extract of opium	. . .	aa $\frac{1}{6}$ grain
Powdered soap	. . .	$1\frac{1}{2}$ grains
Glycerine	. . . .	Q.S.

These pills remain soft for a long time, and may be softened again when they become dry by mixing them with a drop of glycerine. Some chemists use mannite as an excipient; this also remains soft for a long time.

In ordinary cases of syphilis, two of the above pills daily are sufficient, taken after meals; but in severe cases, three pills may be given daily, taking care to test the **elimination of mercury by the kidney**. To test the permeability of the kidney, three procedures are necessary: (1) test for albumen; (2) estimation of the mercury eliminated in the urine (by means of Smithson's pile); (3) estimation of the nitrogenous excretion.

The proportion of the nitrogen of urea to the total amount of nitrogen, including that of the extractive matters, is normally from 85 to 90 per cent., or 87·5 per cent. on the average. When this figure falls below 85 per cent., and especially when it falls below 80 per cent., it is necessary to take great precautions in the administration of toxic drugs, especially mercury.

Bichloride of mercury may also be prescribed in solution, in the form of Van Swieten's liquor [1] (bichloride of mercury 1, alcohol 100, water 1000). Four teaspoonfuls of this may be given daily in milk: this is equivalent to two of the bichloride pills.

Owing to the disagreeable taste of this preparation, I have lately employed *neutral lactate of mercury* in a solution of 1 in 1000, four teaspoonfuls of which are given daily.

To resume, I recommend two drugs for the administration of mercury by the stomach: (1) *bichloride pills*, containing 1 centigram ($\frac{1}{6}$ grain), two pills daily; (2) *mercuric lactate*, 1 in 1000, four teaspoonfuls daily.

(2) **Inunction.** — This method consists in rubbing mercurial ointment into the skin. The mercury is absorbed in two ways:

[1] The nearest equivalent to this is the Liquor Hydrargyri perchloridi of the B.P., which is given in doses of 1 to 2 drachms, two or three times a day.—Ed.

through the epidermis by glandular assimilation, and through the lungs, the mercury being volatilised by friction.

In order to be efficacious, inunction should be practised methodically. At each rubbing, 5 grammes of ointment (about 75 grains) should be rubbed in for fifteen to twenty minutes with the bare hand. It is better for the patient to do the rubbing himself, as it may cause mercurial stomatitis in the operator. Before the rubbing, the skin is washed with soap, to render absorption more easy. The best regions for inunction are the groins and axillæ, where the gland ducts are more open. The rubbings are made daily, each region being taken in turn. The ointment is left on during the night, and washed off in the morning.

When properly carried out, inunction is a good method of administering mercury, but it should not be continued very long, because it soon causes salivation, and may also give rise to cutaneous eruptions. Twenty consecutive rubbings are usually enough.

(3) **Subcutaneous injection.**—For this method, as for ingestion, I prefer the *soluble salts of mercury.* The objection to these salts is that they necessitate daily injections; but it is not absolutely necessary for these to be performed by the surgeon himself.

I employ the soluble mercurial salts in 1 per 100 solution, 1 or 2 cubic centimetres of which are injected daily, according as the dose to be given is 1 or 2 centigrams. The salts which seem to be best tolerated are the *benzoate* and the *biniodide* of mercury. *Benzoate of mercury* is only soluble in water in the presence of sodium chloride or an alkaline benzoate. The following is the formula which I use:—

Benzoate of mercury . . .	1 gramme
Chloride of sodium . . .	2·5 grammes
Sterilised water	100 c.cms.

The chemist should prepare the benzoate himself, by treating yellow oxide of mercury in acid solution with benzoate of soda; the salt thus obtained should be washed for a long time in distilled water, till the washings are no longer acid. The commercial benzoate of mercury is generally impure, does not easily dissolve, and becomes precipitated from its solution after a few days.

The usual dose of benzoate of mercury is 2 centigrams daily ($\frac{1}{3}$ grain), or 2 cubic centimetres of the 1 per cent. solution. Larger doses cause stomatitis; but this can be prevented by the simultaneous administration of sulphur, which aids the elimination of mercury. Sulphur waters allow large doses of mercury to be tolerated.

Biniodide of mercury is only soluble in water in the presence of iodide of sodium. A 1 per cent. solution is used:—

Biniodide of mercury Iodide of sodium } .	aa 10 centigrams
Distilled water . .	to 10 c.cms.

A cubic centimetre of this solution contains 1 centigram of biniodide. Two cubic centimetres usually are injected daily, containing 2 centigrams ($\frac{1}{3}$ grain).

Bichloride of mercury may also be used for injection, according to the following formula:—

Bichloride of mercury . . .	1 gramme
Chloride of sodium . . .	75 centigrams
Sterilised water . . .	100 c.cms.

Bichloride is more painful than benzoate and biniodide, and should only be used when the others cannot be obtained. Only 1 centigram can be injected daily.

Injections of soluble salts should be made into the *subcutaneous tissue* of the buttock, not into the muscles, with a very fine needle about an inch long. Longer and larger needles are useless, painful and dangerous, for they may penetrate the muscles and cause sloughing.

Mercurial Stomatitis.—Whatever the mode of administration of mercury, the patient should pay great attention to his mouth, to avoid mercurial stomatitis. Carious teeth and stumps should be removed or stopped, and the teeth should be cleaned morning and evening during the whole duration of treatment, with a dentrifice such as the following:—

Chlorate of potash Prepared chalk Powdered cinchona	aa 15 parts
Salol	2 „

A mouth-wash of chlorate of potash (2 per 100), or oxygenated water, diluted with three parts of water, should also be used frequently.

Indications for the different Methods.—In the generality of cases, mercury may be given by the mouth, in the form of pills or solution. It is generally absorbed easily, and this mode of administration is usually much more acceptable than inunction or injection.

The two latter methods are more rapid in action and more efficacious than the administration of mercury by the mouth. Inunction and injection are equally active, but inunction is a dirty method, and is often difficult to carry out properly. For this reason, injections are generally to be preferred to inunction, in cases where rapid and intense mercurial treatment is indicated. These cases are as follows: (1) phagedenic chancres; (2) exuberant chancres, such as those of the chin; (3) ulcerative malignant syphilides; (4) all persistent and rebellious syphilides, which resist treatment by pills and constantly recur in spite of this treatment, especially palmar or lingual syphilides, which are always very tenacious; (5) visceral

syphilis, especially in the tertiary period; (6) syphilis of the nervous system, where it is necessary to act promptly. Lastly, it is sometimes necessary to use injections in normal cases of syphilis, when the drugs are badly tolerated by the stomach and give rise to dyspepsia and diarrhœa.

Sulphur.—In certain cases, especially when large doses of mercury are indicated, *sulphur waters* are an excellent adjuvant medication, in the form of baths, douches and drinks. These allow intense mercurialisation, owing to the formation of sulpho-mercurial compounds, which are easily eliminated. Sulphur may even set at liberty old accumulations of mercury which have become inactive; for instance, after calomel or other insoluble injections.

When mercury has disappeared in the urine after mercurial treatment, it often reappears after the administration of sulphur waters. This often accounts for the benefit which syphilitics derive from treatment at sulphur springs. The sulphur waters create, in a way, a constant and rapid current of mercurialisation through the organism. The sulphur utilises the mercury and favours its elimination. Sulphur waters also have a general tonic action.

Iodide of Potassium.—The second adjuvant to mercurial treatment is *iodide of potassium*, which may be given in doses of 2 to 6 grammes daily (30 to 90 grains), according to the severity of the case.

Iodide of potassium should be prescribed, not only in the tertiary period of syphilis as is usually done, but at all periods in cases of severe syphilis. It is indicated by the nature of the lesion rather than by the period in which this lesion occurs. All severe lesions require iodide of potassium as an adjuvant to mercury, especially the following: (1) phagedenic chancres; (2) recurrent or hypertrophic mucous patches; (3) ulcerative lesions at all periods; (4) embryonic neoplasms, gummas, sclerosis, etc.

Iodide is a resolvent. It is, therefore, indicated in all specific neoplasms, cutaneous and visceral gummas; and in all sclerous neoplasms, arteritis, and lesions of the nervous system. But it is only an adjuvant to mercury and cannot replace it, for it has no action on the syphilitic virus. Syphilis can be cured by mercury alone, but not by iodide of potassium alone.

Iodide is contra-indicated in the following conditions: (1) in larnygeal syphilis, because it is a vaso-dilator and may cause œdema of the glottis; (2) in pulmonary syphilis, because it may cause hæmorrhage; (3) in acute renal lesions, because it may cause congestion of the kidney, with hæmaturia, albuminuria, etc.; (4) in cerebral hæmorrhage, because it increases congestion and may cause recurrent hæmorrhage; (5) in lesions of the fundus of the eye, because it may cause retinal hæmorrhage; (6) in cases of doubtful epithelioma, because it favours extension of the growth.

General Principles of Treatment.—The treatment of syphilis should be continued as long as there are any symptoms, except for temporary suspension when there are signs of intolerance; such as stomatitis, diarrhœa or albuminuria. Treatment should also be continued in the absence of symptoms, in order to destroy the virus, and prevent, as far as possible, later manifestations. "Syphilis," says Fournier, "is a chronic infection; it requires chronic treatment." This chronic treatment should be intermittent and successive, with intervals of rest.

As soon as the primary chancre is diagnosed, mercurial treatment should be prescribed for *four years*, in the following way:—

1*st year.*—First of all, two months' treatment by pills, or two weeks of injections and a month of pills, or two courses of inunction of three weeks each. After this, treatment for one month out of two during the rest of the year.

2*nd year.*—Five months' treatment, divided as follows: one month of treatment followed by a month of rest, with a rest of two months in the middle of the year.

3*rd year.*—Four months' treatment: one month of treatment followed by two months of rest.

4*th year.*—Two months' treatment: one month in the spring, the other in the autumn; or, four periods of two weeks, separated by periods of rest of two months and a half.

When treated in this way, the patient has a great chance of escaping later manifestations. It must be remembered, however, that the virulence of syphilis varies considerably in different individuals, without any known cause.

While some cases of syphilis become cured in spite of insufficient treatment, others may show symptoms after four years' treatment. In these cases, mercurial treatment must be resumed and continued, in the hope of finally arresting the evolution of the disease.

Marriage of Syphilitics.—In ordinary cases, after four years' treatment, the patient should be kept under observation for another year without treatment. If after the fifth year the patient has shown no signs of the disease for two years, marriage may be allowed in the sixth year; but it is advisable for him to take mercury for one or two months immediately before marriage, in order to avoid procreating a syphilitic child.

Lastly, when his wife becomes pregnant, it is wise to give her mercurial treatment, at any rate during her first pregnancies.

YAWS, OR FRAMBŒSIA.

THIS is "a tropical, specific, infectious disease caused by a treponema; characterised by a peculiar granulomatous eruption, and presenting a course somewhat similar to that of syphilis" (Castellani [1]).

The disease is known by different names in different countries. In the West Indies it is called *yaws*, in Ceylon *parangi*. The French call it *pian*. It was described by Copland as *syphilis æthiopica*. The name *frambœsia* was given to it by Sauvage in 1759, on account of its raspberry-like eruption. It has also been known as *sibbens* or *sivvens*, etc.

SYMPTOMATOLOGY.—There are three periods in the evolution of the disease, as in syphilis; a primary lesion, a secondary period, and tertiary ulcerations and gummas.

The Primary Lesion.—The incubation is from two to four weeks, during which there are often feverish symptoms and a rise of temperature. The primary lesion is a papule, which becomes covered with a crust in about a week. There are often several papules, which coalesce. When the crust is removed, an ulcer is seen, with clean edges and a granulating base. This may heal and leave a scar, or it may form a granulomatous nodule like those of the secondary eruption. According to Castellani, the primary sore is never indurated. The lymphatic glands may be enlarged, but never suppurate.

The primary lesion is usually, but not always, extra-genital. It may develop at the site of an abrasion or insect-bite, etc. It may heal before the secondary eruption appears.

The Secondary Period.—This begins from one to three months after the primary lesion, and is often accompanied by pains in the joints and bones, sometimes by remittent or intermittent fever.

The eruption begins in the form of small papules, the size of a pin's head, which become covered with yellow crusts. Some of these disappear, while others develop into the characteristic frambœsial or

[1] *System of Syphilis*, vol. iii.

granulomatous nodules, which are covered with yellow or brown crusts. These lesions are of various sizes and occur on any part of the body, but especially on the limbs and face. They may persist for months and then become hard and hyperkeratosic. In most cases (after three to six months in children and six to twelve months in adults) the lesions dry up and disappear, leaving white or pigmented spots. Each granuloma goes through its evolution in two to four months.

This framboesial granulomatous eruption is typical of the disease, but papular, squamous and ulcerative eruptions may also occur. The granulomatous eruption is common on the palms and soles, and leaves patches of desquamation similar to those of syphilis.

The hair and nails are not affected.

The mucous membranes are not often affected, but small granulomas are sometimes found at the base of the tongue; also patches of leucoplakia.

The cervical and inguinal lymphatic glands are enlarged, hard and painless.

The bones and joints are sometimes affected with periostitis and arthritis, and multiple dactylitis is said to be common.

According to Castellani, iritis sometimes occurs. The cerebro-spinal fluid is normal.

The Tertiary Period.—Tertiary lesions do not always occur, for the disease may terminate with the secondary stage. The interval between the secondary and tertiary periods varies considerably, and may be several years. The tertiary lesions consist in gummatous nodules and deep ulcerations. The ulcers may be punched-out, serpiginous, deep and irregular, or fungating. The bones may be affected by chronic diffuse periostitis, or circumscribed nodes. Tertiary lesions of the internal organs and the nervous system have not been described, but probably exist.

Histopathology.—The epidermis of the papules is thickened, and there are epithelial downgrowths. The epithelial cells show vacuolation and degeneration. The dermis is œdematous and infiltrated with polynuclear leucocytes, large and small mononuclear cells, eosinophile cells, plasma cells, mast-cells, connective-tissue cells, and extravasated erythrocytes. In older papules the plasma cells predominate. The lesions differ from syphilitic lesions in the absence of perivascular infiltration and endothelial proliferation of the vessels, and in the absence of giant cells. In the papules of yaws, the epidermis is more affected than the dermis.

Microbiology.—Castellani has discovered a spirochæte, which he names *Treponema pertenue*, and regards as the specific cause of yaws. This micro-organism is found in the primary lesion, in the unbroken papules of the secondary eruption, in the spleen, lymphatic glands,

and bone-marrow. The open sores contain other spirochætes and bacteria. The *Treponema pertenue* is a delicate spirochæte, morphologically similar to the *Treponema pallidum*, or spirochæte of syphilis. However, Castellani considers that these two microbes are distinct, on the ground that monkeys immunised against the *Treponema pertenue* are not immune against the *Treponema pallidum*, and inversely.

This brings us to the question whether yaws is a distinct disease, as Castellani maintains, or only a variety of syphilis, as was formerly believed, and as Sir Jonathan Hutchinson still holds.

Castellani brings forward the following points in favour of yaws being distinct from syphilis:—

(1) A patient with syphilis may contract yaws, and inversely;

(2) Monkeys inoculated with syphilis are not immune to yaws, and inversely;

(3) Syphilis is of world-wide distribution, while yaws is limited to the tropics;

(4) Yaws is present in countries where syphilis is unknown; for instance, it existed in the Fiji Islands before syphilis was introduced into those islands;

(5) The primary sore is nearly always extra-genital;

(6) The histological structure of the lesions of yaws differs from that of syphilitic lesions, in the absence of endo-periarteritis and giant cells, and in the presence of epidermic proliferation.

Sir Jonathan Hutchinson[1] takes a broader view of the question, and regards yaws as a *variant* of syphilis—"frambœsial syphilis." His arguments may be summed up as follows:—

(1) The treponema or spirochæte of yaws is hardly, if at all, distinguishable from that of European syphilis. Sir J. Hutchinson remarks: "We are prepared to anticipate that there may be not one uniform type of spirochætal disease to be known as syphilis, but several variants. The parasite itself may assume, without losing its identity, features as widely different as those between the different races of mankind, or the different breeds of dogs. We must not hastily impose limits to the possible variations of organisms about which, as yet, our knowledge is imperfect."

(2) Yaws goes through three stages like syphilis, and is curable by the same remedies.

(3) The fact that a syphilitic subject can contract yaws is not conclusive, for syphilis may be contracted more than once, and the interval between the two infections may be very short. On the other hand, there is strong evidence to show that yaws does, as a rule, prevent syphilis.

(4) Yaws is not confined to the tropics. Sporadic cases and

[1] *Syphilis*, New Edition, 1909.

local epidemics have occurred in Europe; for instance, the epidemic which occurred among Cromwell's soldiers, and was known as "sibbens," or "frambœsia Cromwelliana." These sporadic cases afterwards revert to the normal type of syphilis.

TREATMENT.—Most authorities agree that iodide of potassium is beneficial in yaws, but opinions differ as to the efficacy of mercury. Castellani recommends large doses of iodide (up to a drachm daily) which should be continued after the lesions have disappeared; but he states that mercury has no effect. He claims to have obtained good results with atoxyl.

Local treatment consists in the application of mercurial lotions and iodoform, etc.

According to Castellani, yaws is probably hereditary, but the evidence on this point is inconclusive. At any rate, no lesions comparable to the well-known signs of congenital syphilis have been described.

INDEX

NOTE.—*Figures in heavy type indicate principal references.*

PRINTED BY OLIVER AND BOYD, EDINBURGH

Works on

Medicine and Science

FROM MR. MURRAY'S LIST

CHILDREN IN HEALTH AND DISEASE. A Study of Child-Life. By DAVID FORSYTH, M.D., D.Sc., Physician to the Evelina Hospital for Sick Children, Assistant Physician (late Physician in Charge of the Children's Department) and Joint Pathologist, Charing Cross Hospital. Demy 8vo. 10*s*. 6*d*. net.

"The physiology and psychology of the child at every stage of its existence is fully dealt with, together with the bearing thereon of the work of health visitors, the Notification of Births Act, and other points connected with the functions of public health authorities. The situation of the school buildings, drainage, closet accommodation, design of class-rooms, arrangement of work, treatment of infectious diseases, medical inspection, class-room hygiene, and the training of defective children of every type, are among the many subjects which receive attention. The essence of the book, however, is to be found in the author's view of the evil results of treating the different aspects of child-life as entirely distinct from one another, and of the great importance of close co-operation between the public health and education departments."—*Official Circular of the County Councils Association.*

MOSQUITO OR MAN? The Conquest of the Tropical World. By Sir RUBERT W. BOYCE, M.B., F.R.S. With Illustrations. Medium 8vo. 10*s*. 6*d*. net.

"A book which gives us for the first time a thoroughly complete, up-to-date history of a branch of parasitology, and dealing with the most wonderful of all the manifestations connected with the development of modern medicinal science. In 'Mosquito or Man?' the Dean of the Liverpool School of Tropical Medicine has given to the world a masterpiece of lucid exposition of the nature of the problem which Manson, Ross, and others have gone so far to solve."—*Liverpool Journal of Commerce.*

"This very interesting volume cannot fail to bring home to the reader the vast importance, economic as well as scientific, of this new conquest of the Tropical world. The text is elucidated by numerous plates from photographs which illustrate not only several of the insects concerned in the propagation of disease, but views of 'Anti-Mosquito' brigades, Screened Wards in Hospitals, Houses in course of formation, and many other scenes and objects bearing upon the contents of the work."—*Lancet.*

A HANDBOOK OF PHYSIOLOGY. By W. D. HALLIBURTON, M.D., F.R.S., Professor of Physiology, King's College, London. Ninth Edition, being the Twenty-second of Kirkes'. With nearly Seven Hundred Illustrations, including some Coloured Plates. Large Crown 8vo. 15*s*. net.

". . . One of the best manuals for the student which we possess . . . the book is an eminently trustworthy one, and will prove a valuable foundation for, and introduction to, the large treatises on physiology."—*The Lancet.*

"The popularity of this excellent student's text-book is shown by the fact that a new edition is called for every year."—*British Medical Journal.*

CANCER OF THE BREAST AND ITS OPERATIVE TREATMENT.

By W. Sampson Handley, M.S. (Lond.), F.R.C.S., Assistant Surgeon to Middlesex Hospital, Hunterian Professor, R.C.S. Medium 8vo. 12*s*. 6*d*. net.

"His views are somewhat novel, but are well-supported by the evidence he brings forward, and are of vital importance to all surgeons."—*The Medical Annual, 1906.*

A PRIMER OF PHYSIOLOGY.

By Prof. E. H. Starling, F.R.S. Illustrated. F'cap 8vo. 1*s*.

SYNOPSIS OF CONTENTS.—Introductory—Structure—The Food—Digestion—The Circulation of the Blood—Breathing—Excretion: The Functions of the Kidneys—The Skin and its Uses—The History of the Food in the Body—The Chemical Factories of the Body—The Defence of the Body against Micro-Organisms—The Physiology of Movement; The Muscles; The Central Nervous System—Feelings.

THE TREATMENT OF SOME ACUTE VISCERAL INFLAMMATIONS, AND OTHER PAPERS.

By David B. Lees, M.A., M.D. Cantab., F.R.C.P. Lond., formerly Scholar of Trinity College, Cambridge; Senior Physician to the Hospital for Sick Children, Great Ormond Street; Physician to St. Mary's Hospital. Crown 8vo. 6*s*. net.

"Dr. Lees' 'Treatment of Some Acute Visceral Inflammations' will prove a welcome addition to the literature dealing with the treatment of visceral inflammations. His experience is very wide, and the success which has followed the various methods advocated for the relief of dilatation of the heart, pericarditis and other local inflammatory processes, proves the correctness of the principles upon which his treatment is based. . . . We warmly commend the perusal of this volume, not only to practitioners, but to medical students."—*British Medical Journal.*

A MANUAL OF PATHOLOGY.

Handbook for Students. By Sidney Martin, M.D., F.R.S., F.R.C.P., Professor of Pathology, University College. With numerous Woodcuts from Micro-Photographs. Medium 8vo. 15*s*. net.

"This work is of such high excellence and the difficult task of giving a complete survey of such a rapidly-growing subject as general pathology has been so well accomplished that, in spite of the criticism we have offered, we can heartily recommend the work to students of pathology. In general terms it maintains the high standard of the school from which it emanates."—*The Lancet.*

"Is excellent and superior, we think, to any other work of the kind in the English language."—*Hospital.*

A TREATISE ON MEDICAL JURISPRUDENCE.

Based on Lectures delivered at University College, London. By G. Vivian Poore, M.D. With Illustrations. 8vo. 12*s*. net.

"Rarely have we found a text-book more charming to read and at the same time displaying such a thorough grasp of knowledge of the subject. By means of the free employment of illustrative cases, drawn from the records of the law courts and the author's personal experience, the dry details are endowed with lively interest, which is likely to imprint them on the student's memory. . . . We thoroughly endorse this plan. . . . We offer our warm congratulations to Dr. Poore on having written one of the most readable and instructive manuals in medical jurisprudence yet published, and we feel sure that it will meet with the success which it deserves."
—*The Lancet.*

THE NERVOUS SYSTEM OF VERTEBRATES

By J. B. JOHNSTON, Ph.D., Professor of Zoology in West Virginia University. With 180 Illustrations. 15*s*. net.

"The book is thoroughly scientific in its treatment of the numerous parts of the nervous system dealt with. It is written by one who is evidently a master of his subject, and every chapter bears the impress of knowledge obtained at first hand."—*Medical Times.*

A SERIES OF LECTURES DELIVERED AND PUBLISHED UNDER THE AUTHORITY OF THE UNIVERSITY OF LONDON

INTRACELLULAR ENZYMES. A Course of Lectures given in the Physiological Laboratory, University of London. By H. M. VERNON, M.D. Medium 8vo. 7*s*. 6*d*. net.

SIGNS OF LIFE FROM THEIR ELECTRICAL ASPECT. Lectures on Physiology. By AUGUSTUS D. WALLER, M.D., F.R.S. With numerous Illustrations. Square Demy 8vo. 7*s*. 6*d*. net.

"The book, like all the volumes which Dr. Waller has produced for the benefit of the student, is of great educational value, and is none the less so in this case because the reader is designedly never allowed to escape from the personal magnetism of his teacher."—*The Lancet.*

BIOCHEMISTRY OF MUSCLE AND NERVE

By W. D. HALLIBURTON, M.D., F.R.S., Professor of Physiology, King's College, London; Editor of Kirkes' "Handbook of Physiology." With Illustrations. Demy 8vo. 7*s*. 6*d*. net.

"Every medical man and others who, although not having special medical training, have scientific training, should read this book. The author knows how to bring out the salient parts of his subject with incisive clearness."—*Knowledge.*

CHEMISTRY OF THE ALBUMENS. By S. B. SCHRYVER, D.Sc. Lond., Ph.D. Leipzig, Lecturer in Physiological Chemistry to University College, London. With Diagrams. Demy 8vo. 7*s*. 6*d*. net.

"The book is unquestionably of value and interest. The work is thoroughly done. . . . Those who can appreciate it will certainly find much profit in this excellent work."—*Hospital.*

THE MORPHOLOGY OF NORMAL AND PATHOLOGICAL BLOOD. By GEORGE A. BUCKMASTER, D.M. (Oxford), University College, London, formerly Radcliffe Fellow Magdalen College, Oxford. With Illustrations. Demy 8vo. 10*s*. 6*d*. net.

"One of the most important contributions to the literature of this subject that has appeared within recent years. It is full of new and suggestive facts which no scientific practitioner of medicine can afford to neglect."—*The Practitioner.*

THERAPEUTICS OF THE CIRCULATION.

By Sir T. LAUDER BRUNTON, Bart., M.D., D.Sc., LL.D., F.R.S., Consulting Physician to St. Bartholomew's Hospital. With numerous Illustrations. 7*s*. 6*d*. net.

THE BACTERIOLOGY OF MILK.

By HAROLD SWITHINBANK, of the Bacteriological Research Laboratory, Durham, and GEORGE NEWMAN, M.D., F.R.S.E., D.P.H., Medical Officer of Health of the Metropolitan Borough of Finsbury, and formerly Demonstrator of Bacteriology in King's College, London. With Special Chapters also by Dr. NEWMAN on Spread of Disease by Milk and the Control of the Milk Supply. With Chromo-Lithographs, Woodbury type reproductions of Photographs of Cultures, and other Illustrations of Bacteria and of Apparatus, and also Charts illustrating Epidemics. Royal 8vo. 25*s*. net.

"Ought to find a place in the library of every medical officer of health and of every milk-producer. Scientific in method and lucid in exposition, the authors have given us a really invaluable text-book. . . . We are glad to commend this book as one of the most valuable of recent English contributions to the science of public health."
—*Spectator.*

BACTERIOLOGY AND THE PUBLIC HEALTH.

By GEORGE NEWMAN, M.D., F.R.S. Edin., D.P.H., Chief Medical Officer, Board of Education. With numerous Illustrations. Medium 8vo. 21*s*. net.

"The present work, though nominally a third edition of 'Bacteria in Relation to the economy of Nature, Industrial Processes, and the Public Health,' is virtually a new book, written with the object of supplying all that is necessary for the student of hygiene and the officer of health to know, so far as every day problems of sanitation and preventive medicine demand. . . . Dr. Newman has done a good work in producing a treatise which places at the service of the community what is known about all these topics."—*Daily Telegraph.*

THE RECENT DEVELOPMENT OF PHYSICAL SCIENCE.

By W. C. D. WHETHAM, M.A., F.R.S. Illustrated. Large Crown 8vo. 5*s*. net.

INTRODUCTION—THE PHILOSOPHICAL BASIS OF PHYSICAL SCIENCE—THE LIQUEFACTION OF GASES AND THE ABSOLUTE ZERO OF TEMPERATURE—FUSION AND SOLIDIFICATION—THE PROBLEMS OF SOLUTION—THE CONDUCTION OF ELECTRICITY THROUGH GASES—RADIO-ACTIVITY—ATOMS AND ÆTHER—ASTRO-PHYSICS—INDEX.

NOTEWORTHY FAMILIES—(SCIENCE).

An Index to Kinships in near degrees between persons whose achievements are Honourable, and have been publicly recorded. By FRANCIS GALTON, D.C.L., Hon. D.Sc. Camb., F.R.S., and EDGAR SCHUSTER, Galton Research Fellow in Natural Eugenics. Crown 8vo. 6*s*. net.

THE HEREDITY OF ACQUIRED CHARACTERS IN PLANTS.

An Aspect of the true Darwinism based on Personal Observations and Experiments. By the Rev. Prof. GEORGE HENSLOW. With Illustrations. Demy 8vo. 6*s*. net.

RECENT ADVANCES IN THE STUDY OF VARIATION, HEREDITY AND EVOLUTION.

By ROBERT H. LOCK, M.A., Fellow of Gonville and Caius College, Cambridge. With Portraits and other Illustrations. Crown 8vo, 5*s*. net.

The past few years have been marked by advances in Biology scarcely less extensive than those made by Physical Science within the same period; and amongst all the branches of the study of living things none have shown greater progress than that of the investigation of heredity and of the kindred problems which bear upon the great question of Organic Evolution. Coming at a most opportune moment, the rediscovery of the work of Mendel has supplied the key to problems which were being approached by several converging lines of inquiry, and is leading to a remarkable simplification of ideas as to the method of hereditary transmission of definite features in human beings and in other living things. To designate the science which deals with the problems of heredity and variation by the new methods now available the term Genetics has been proposed. The object of the present book is to describe the connection between the new science of Genetics and those ideas which have long been summed up under the expression Darwinism, as well as to point out the practical importance and theoretical interest of these subjects.

MICROSCOPY.

The Construction, Theory, and Use of the Microscope. By EDMUND J. SPITTA, F.R.A.S., F.R.M.S., etc. With numerous Diagrams and Illustrations. Second Edition. 12*s*. 6*d*. net.

"Let us hasten to urge every student of the microscope who wishes to gain a thorough understanding of its principles and possibilities and its defects, and every user of the instrument who desires a work of reference to which he may turn for an explanation of some unexplained optical phenomenon, or for particulars of up-to-date apparatus, to procure a copy of Mr. Spitta's book without delay."—*Nature*.

SCIENCE PROGRESS IN THE TWENTIETH CENTURY.

A Quarterly Journal of Scientific Work and Thought. Edited by H. E. ARMSTRONG, Ph.D., LL.D., F.R.S., J. BRETLAND FARMER, M.A., D.Sc., F.R.S., and W. G. FREEMAN, B.Sc., F.L.S. About 192 pp. Illustrated. 5*s*. net. Subscription £1 per annum, post free throughout the World.

ADVISORY COMMITTEE: HORACE T. BROWN, LLD., F.R.S.; R. GLAZEBROOK, M.A., Sc.D., F.R.S.; H. J. MACKINDER, M.A.; J. E. MARR, M.A., Sc.D., F.R.S.; H. A. MIERS, M.A., D.Sc., F.R.S.; E. A. MINCHIN, M.A.; P. CHALMERS MITCHELL, M.A., F.R.S.; SIR W. RAMSAY, K.C.B., Ph.D., Sc.D., F.R.S.; SIR A. RÜCKER, M.A., D.Sc., F.R.S.; E. H. STARLING, M.D., F.R.C.P., F.R.S.; A. D. WALLER, M.D., LL.D., F.R.S.

"Science Progress" contains original papers and summaries of the present state of knowledge in IN ALL BRANCHES OF SCIENCE of an authoritative character, written by those actually engaged in advancing knowledge on the subjects with which they deal. That very tangible success has been attained in realizing these ideals is demonstrated by the papers which have appeared in the seven numbers already issued.

WRITE FOR SPECIMEN COPY, POST FREE.

MR. MURRAY'S

Progressive Science Series

Large 8vo. Cloth extra, 6s. net per volume.

THE INTERPRETATION of RADIUM. By FREDERICK SODDY, Lecturer in Physical Chemistry in the University of Glasgow. With Diagrams.

THE PROBLEM OF AGE, GROWTH AND DEATH. A Study of Cytomorphosis. By CHARLES S MINOT, LL.D., Professor of Comparative Anatomy in the Harvard Medical School. Illustrated.

THE SOLAR SYSTEM. A Study of Recent Observations. By CHARLES LANE POOR, Professor of Astronomy in Columbia University.

CLIMATE, considered especially in Relation to Man. By ROBERT DE COURCY WARD, Assistant Professor of Climatology in Harvard University.

HYGIENE OF NERVES AND MIND IN HEALTH AND DISEASE. By AUGUST FOREL, M.D. Translated from the German by AUSTIN AIKINS, Ph.D. Large Crown 8vo.

EARTHQUAKES. In the Light of the New Seismology. By CLARENCE EDWARD-DUTTON, Major in the United States Army. Illustrated

INFECTION and IMMUNITY By GEORGE S. STERNBERG, M.D., Surgeon-General to the U.S. Army, retired.

THE STARS. A Study of the Universe. Illustrated. By Professor NEWCOMB.

RIVER DEVELOPMENT. As Illustrated by the Rivers of North America. By Professor I, C. RUSSELL. Illustrated.

THE COMPARATIVE PHYSIOLOGY OF THE BRAIN AND COMPARATIVE PSYCHOLOGY. By Professor JACQUES LOEB, M.D., Professor of Physiology in the University of Chicago. Illustrated.

THE STUDY OF MAN. An Introduction to Ethnology. By Professor A. C. HADDON, D.Sc., M,A. Illustrated.

EARTH SCULPTURE. By Professor GEIKIE, LL.D., F.R.S. Second Edition. Illustrated.

VOLCANOES. By Professor BONNEY, D.Sc., F.R.S. Illustrated.

THE GROUNDWORK OF SCIENCE. By ST. GEORGE MIVART, M.D., Ph.D., F.R.S.

A BOOK OF WHALES. By F. E. BEDDARD, M.A. With 40 Illustrations by SIDNEY BERRIDGE.

HEREDITY. By J. ARTHUR THOMSON, Regius Professor of Natural History in the University of Aberdeen. With Coloured and other Illustrations. Large Crown 8vo. 9s. net.

THE CHEAPER EDITION OF

DARWIN'S LIFE AND WORKS. Green cloth. Large Crown 8vo. 2s. 6d. net each.

A complete list of the Volumes will be sent post free on application

---

JOHN MURRAY, ALBEMARLE STREET, W.