Pathology and treatment of diseases of the skin : for practitioners and students / by Moriz Kaposi ; translation of the last German edition, under the supervision of, James C. Johnston.

Contributors

Johnston, James Chew.
Kaposi, Moriz, 1837-1902.
Royal College of Physicians of Edinburgh

Publication/Creation

London : Bailliere, Tindall and Cox, 1895.

Persistent URL

https://wellcomecollection.org/works/mre43mdx

Provider

Royal College of Physicians Edinburgh

License and attribution

This material has been provided by This material has been provided by the Royal College of Physicians of Edinburgh. The original may be consulted at the Royal College of Physicians of Edinburgh. where the originals may be consulted.
This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.

Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
https://wellcomecollection.org

R.C.P. EDINBURGH LIBRARY
R21480N0236

25

* Yale 10. 22

PATHOLOGY AND TREATMENT

OF

DISEASES OF THE SKIN

FOR

PRACTITIONERS AND STUDENTS

BY

DR. MORIZ KAPOSI

PROFESSOR OF DERMATOLOGY AND SYPHILIS, AND CHIEF OF THE CLINIC AND DIVISION FOR SKIN DISEASES IN THE VIENNA UNIVERSITY

ROYAL COLLEGE OF PHYSICIANS EDINR. LIBRARY.

WITH EIGHTY-FOUR ILLUSTRATIONS

TRANSLATION OF THE LAST GERMAN EDITION

UNDER THE SUPERVISION OF

JAMES C. JOHNSTON, M.D.

LONDON

BAILLIÈRE, TINDALL AND COX

20 & 21, KING WILLIAM STREET, STRAND

1895

Digitized by the Internet Archive
in 2015

https://archive.org/details/b21959821

PREFACE.

In spite of the fact that a number of excellent text books on skin diseases have been written by English and American authors, a familiarity with the views of our dermatological colleagues in France and Germany is so desirable that their published works will always meet with a cordial welcome in this country. To those specially interested in dermatology these works are already well known and thoroughly appreciated; but to many of our profession who are more or less unacquainted with the language in which they are written these admirable and authoritative works are known only by name. When such works are made accessible to all by means of careful and accurate translation, medical literature is enriched far more than by the multiplication of inferior text books.

A generation has almost passed since a revolution was wrought in the study of skin diseases by the zealous labors of Ferdinand Hebra. Through his marvellous acumen order was brought out of confusion, and many vague and erroneous ideas that had long prevailed were dissipated by his iconoclastic method of instruction. His brilliant power as a teacher enabled him to mould the views of his many pupils and to leave the impress of his thought upon the schools of every land where dermatology is now taught. In the person of his worthy successor, Moriz Kaposi, ability and attainments of the highest order are clearly manifested. Though handicapped by a severe but inevitable comparison, he has compelled admiration for his success in carrying on the indefatigable research and the valuable instruction of his famous predecessor; and through his keen faculty of observation and his exceptional opportunities for clinical study he has thus far been able to maintain the prestige of the Vienna school.

For many years Prof. Kaposi has occupied a conspicuous place in

the foremost rank of European dermatologists. Intimately associated as he was with Prof. Hebra, no man could have received a more thorough training in any special branch of medicine, and as a collaborator in the completion of an epoch-making work (Hebra's "Lehrbuch der Hautkrankheiten") he became imbued with the spirit and the methods of the great master whose mantle naturally fell upon his shoulders.

The classical work of Hebra and Kaposi (translated in English under the auspices of the Sydenham Society) has long been regarded as a most comprehensive and important publication in the department of dermatology. The lectures of Kaposi contain the views of Hebra, modified as far as has become necessary by the advancement of modern science, amplified by the original observations of the author, and presented to the reader in a most concise and attractive form.

As a descriptive writer Prof. Kaposi is unsurpassed, and in his lectures not only the common but many of the rarest forms of skin disease are depicted with a most astonishing fidelity to nature. To all English-speaking physicians, and especially to those who read only their own language, this excellent translation of a most able and practical work cannot fail to be of the greatest value, and to undergraduate students it will certainly recommend itself as a convenient and a trustworthy book of reference.

The translation of the work has been carefully supervised by Dr. James C. Johnston, to whom the reader will give due credit for smoothness and elegance of diction.

GEORGE HENRY FOX, M.D.

CONTENTS.

GENERAL DIVISION.

LECTURE I.

PAGE

Relation of dermatology to general pathology—Its scientific and practical importance—History of its development from ancient to the most recent times..... 1

LECTURE II.

General character of the pathological processes affecting the skin—Their essential conformity to those occurring in other organs and tissues—Their appearance marked by special characteristics produced by the peculiar anatomy of the skin—The symptoms and causes peculiar to skin diseases—Anatomy of the skin and its appendages.... 16

LECTURE III.

Anatomy of the skin (continued)—Sweat glands, sebaceous glands, hair, nails—Physiology of the skin—Its threefold function as a protective, heat regulating and secretory, and specific sense organ..... 34

LECTURE IV.

General symptomatology, subjective and objective—Primary and secondary lesions—Classification of eruptions..... 48

LECTURE V.

General etiology—Idiopathic and symptomatic lesions—Clinical conception of what constitutes a skin disease—General diagnosis..... 61

LECTURE VI.

Course, significance, effects, and prognosis of skin diseases—Therapy—Systematic classification of skin lesions..... 7

SPECIAL CONSIDERATIONS.

CLASS I.

HYPERÆMIÆ CUTANEÆ.

CUTANEOUS DISEASE INDUCED BY EXCESS OF BLOOD IN THE SUPERFICIAL CUTANEOUS LAYERS.

LECTURE VII.

PAGE

Hyperæmia of the skin, active and passive—Idiopathic and symptomatic hyperæmia—Roseola—Erythema—Anæmia of the skin..... 90

CLASS II.

CUTANEOUS ANÆMIAS.

Morbid phenomena of the skin caused by diminished blood supply of its finest vessels..... 100

CLASS III.

ANOMALIES OF THE SECRETION

OF THE SKIN AND OF THE CUTANEOUS GLANDS—DISEASES OF THE SKIN DUE TO ABNORMALITIES OF THE CUTANEOUS SECRETION AND THE CUTANEOUS GLANDS.

LECTURE VIII.

ANOMALIES OF PERSPIRATION AND OF THE SECRETION OF SWEAT.

Physiology of the secretion of sweat—Chemical constitution of sweat and its morbid secretion—Quantitative disorders—General and local hyperidrosis—Local and general sequelæ and complications—Treatment—Anidrosis—Qualitative anomalies of the secretion of sweat—Anatomical changes 104

LECTURE IX.

ANOMALIES OF THE SECRETION OF FAT.

Physiology of the secretion of fat—Pathology—Excessive secretion—Local and general seborrhœa—Diagnosis—Prognosis—Treatment Diminished secretion—Xerosis—Disordered excretion: its results as forms of proliferation, degeneration, and retention—Comedo—Milium—Molluscum verrucosum seu contagiosum—Atheroma..... 121

CLASS IV.

INFLAMMATORY DERMATOSES.

Skin Diseases due to Exudation and Inflammation.

LECTURE X.

General Remarks on Exudation and Inflammation.

PAGE

Exudation and inflammation in general—Cell fission : its relations to the former and to the fixed and wandering formed elements—Symptoms of cutaneous exudation and inflammation: their course and termination—Resolution, suppuration, hypertrophy, atrophy, degeneration..... 141

ACUTE EXUDATIVE DERMATOSES.

Acute, Contagious, Inflammatory Dermatoses.

LECTURE XI.

Acute Exanthemata.

The characteristics common to the acute exanthemata—Measles..... 152

LECTURE XII.

Scarlet fever—Scarlatina 163

LECTURE XIII.

Small-pox, Variola.

History—Variolation and vaccination—Variolosis—Varicella—Typical variola—Variola vera—Atypical variola with favorable course..... 175

LECTURE XIV.

Small-pox (*continued*).

Unfavorable atypical forms—Variola hæmorrhagica—Variola confluens —Complications and sequelæ—Anatomy..... 184

LECTURE XV.

Small-pox (*concluded*).

Diagnosis—Prognosis—Influence of vaccination on the severity of the disease—Etiology—Treatment—Prophylaxis—Vaccination: original and humanized lymph—Variola vaccina—Normal and abnormal course 198

LECTURE XVI.

Acute, Non-contagious, Inflammatory Dermatoses.

The anatomical changes in the erythemata identical but varying in degree —Erythema multiforme and herpes iris et circinatus—Erythema nodosum—Purpura rheumatica..... 210

LECTURE XVII.

URTICARIA, NETTLE-RASH.

PAGE
Forms and significance of urticaria, idiopathic and symptomatic, acute and chronic..... 228

LECTURE XVIII.

Vesicular eruptions..... 236

LECTURE XIX.

Herpes labialis—Herpes progenitalis—Herpes iris et circinatus—Miliaria rubra, alba et crystallina—Pemphigus acutus..... 251

LECTURE XX.

FORMS OF DERMATITIS (DERMATITIS ESSENTIALIS).

True Inflammations of the Skin.

Identity of anatomical changes—Clinical differences due to degree and cause of the inflammation—Idiopathic and symptomatic—Infectious—Dermatitis and gangrene—Dermatitis traumatica, mechanica, neuropathica, toxica, a venenatis, diabetica, et dynamica—Caloric form: burns and congelations 260

LECTURE XXI.

SYMPTOMATIC OR INFECTIOUS INFLAMMATIONS OF THE SKIN.

Diffuse erythematous inflammation—Causes: toxic, bacterial infection—Erysipelas: phlegmonous form—Pseudo-erysipelas—Circumscribed forms: furuncle, anthrax (idiopathic and symptomatic); endemic forms: Aleppo boil, zoonoses: maliasmus, cadaveric infection pustule, malignant pustule..... 281

LECTURE XXII.

CHRONIC INFLAMMATORY DERMATOSES.

Chronic Dermatoses presenting the Character of Inflammation (Exudative Processes, Hebra).

Anatomical importance and clinical subdivision of the chronic dermatoses characterized by inflammatory processes—Squamous dermatoses—Psoriasis 300

LECTURE XXIII.

Pityriasis rubra—Lichen scrofulosorum—Lichen ruber..... 318

LECTURE XXIV.

PRURIGINOUS DERMATOSES—ITCHING ERUPTIONS.

Eczema.

PAGE

Definition—Polymorphism and variability of the symptoms—Typical course of acute eczema—Chronic eczema—Anatomical lesions—Special forms of localization—Impetigo faciei—Eczema marginatum—Diagnosis 339

LECTURE XXV.

ECZEMA (*continued*).

Causes—Prognosis—Treatment 352

LECTURE XXVI.

PRURIGO.

Characteristics—Prurigo agria and prurigo mitis 363

LECTURE XXVII.

Acne disseminata—Acne vulgaris—Acne artificialis—Tar, iodine, and bromine acne—Acne rosacea 370

LECTURE XXVIII.

Sycosis—Significance, pathology, and treatment—Sycosis parasitaria—Impetigo—Ecthyma—Impetigo herpetiformis 381

LECTURE XXIX.

VESICULAR ERUPTIONS.

Pemphigus.

Definition of pemphigus—General division into pemphigus vulgaris and foliaceus—General symptomatology—Special forms of pemphigus and their pathology, anatomy, diagnosis, prognosis, treatment 390

CLASS V.

CUTANEOUS HÆMORRHAGES.

DISEASES OF THE SKIN DUE TO EXTRAVASATION OF BLOOD.

LECTURE XXX.

Significance and anatomical conditions—Clinical forms of cutaneous hæmorrhages—Involution—Idiopathic and symptomatic forms—Contusion—Injury—Purpura senilis, variolosa, rheumatica, simplex, hæmorrhagica—Scorbutus—Hæmophilia—Hæmatidrosis 403

CLASS VI.

HYPERTROPHIES.

CUTANEOUS DISEASES CHARACTERIZED BY INCREASE OF VOLUME.

LECTURE XXXI.

PAGE

General remarks on hypertrophy—Anatomical and clinical division according to the implication of the pigment, of the epidermis and the papillæ, and of the cutis as a whole—Pigment hypertrophy—Anatomical seat—Nævus—Lentigo—Ephelis—Chloasma—Addison's disease—Melasma—Appendix : icterus, argyria, tattooing..... 410

LECTURE XXXII.

Hypertrophy of the epidermis and papillæ..... 426

LECTURE XXXIII.

Ichthyosis—Keratosis follicularis and psorospermosis—Hypertrophy of the hair—Plica polonica—Hypertrophy of the nails..... 436

LECTURE XXXIV.

Connective-tissue hypertrophies. Diffuse: Scleroderma (ainhum—appendix: sclerema neonatorum) and elephantiasis arabum—Elephantiasis telangiectodes et neuroticum—Myxœdema—Circumscribed: papilloma (framboesia)..... 454

CLASS VII.

ATROPHIES.

DISEASES OF SKIN CHARACTERIZED BY TISSUE ATROPHY.

LECTURE XXXV.

General remarks on atrophy—Pigment atrophy of the epidermis: congenital (albinism), acquired (vitiligo)—Pigment defect of the hair, congenital and acquired—Canities præmatura, senilis..... 479

LECTURE XXXVI.

Atrophy of the hairs—Alopecia adnata, acquisita, idiopathica, and symptomatica—Special forms: alopecia senilis, alopecia præmatura—Alopecia areata—Alopecia neurotica—Alopecia præmatura symptomatica—Alopecia furfuracea—Alteration in the hair—Atrophia pilorum propria—Trichorrhexis nodosa—Atrophy of the nails..... 488

LECTURE XXXVII.

Atrophia cutis propria, idiopathica (xeroderma, striæ atrophicæ, atrophia senilis) et symptomatica (scars of pregnancy)—Quantitative and degenerative atrophy—Lupus erythematosus..... 499

CLASSES VIII. and IX.

NEOPLASMATA.

LECTURE XXXVIII.

PAGE

New growths—General remarks—Classification—Benign neoplasms—Connective-tissue neoplasms—Keloid—Scars—Molluscum fibrosum—Xanthoma — Fibroma — Lipoma—Neuroma — Dermatomyoma—Osteoma..... 511

LECTURE XXXIX.

Molluscum fibrosum..... 520

LECTURE XL.

ANGIOMATA.

Neoplasms of the blood vessels and lymphatics 526

LECTURE XLI.

Rhinoscleroma..... 533

LECTURE XLII.

Lupus..... 539

LECTURE XLIII.

LUPUS (*continued*).

Anatomy—Treatment—Scrofulosis—Tuberculosis of the skin..... 549

CLASS X.

MALIGNANT NEOPLASMS.

LECTURE XLIV.

Leprosy..... 561

LECTURE XLV.

General character of the syphilides—Classification according to morphological appearances—Special forms—Symptomatology—Diagnosis—Relations to constitutional syphilis—General and local treatment... 575

LECTURE XLVI.

CUTANEOUS SYPHILIS (*continued*).

Treatment..... 584

LECTURE XLVII.

PAGE

Mycosis fungoides (framboesia)—Lymphodermia perniciosa—Sarcomatosis cutis. 593

LECTURE XLVIII.

Carcinoma—The theory of cancer—Its forms—Epithelioma—Connective-tissue cancer—Pigment cancer..... 604

CLASS XI.

CUTANEOUS ULCERS.

LECTURE XLIX.

Definition of ulcer—General symptomatology—Classification—Idiopathic inflammatory, simple, and contagious ulcers—Ulcer of the legs—Chancre—Secondary inflammatory ulcers and those due to neoplasia 614

CLASS XII.

CUTANEOUS NEUROSES.

LECTURE L.

Neuroses of the skin—Theory—Motor, trophic, and sensory neuroses—Pruritus cutaneus, universalis et localis—Pruritus senilis..... 621

CLASS XIII.

PARASITIC DERMATOSES.

LECTURE LI.

Vegetable and animal parasites—General remarks on fungi and their botanical position, action on the skin, classification of dermatomycoses—Special : favus, pathology, treatment 626

LECTURE LII.

Herpes tonsurans—Forms : herpes tonsurans capillitii, vesiculosus, squamosus, maculosus—Onychomycosis—Sycosis parasitaria—Eczema marginatum—Pityriasis versicolor—Erythrasma 637

LECTURE LIII.

Skin Diseases due to Animal Parasites.

Animal parasites—Parasites proper and epizoa—Their mode of action on the skin—Dermatozoonoses—Scabies—Natural history of the acarus—Cuniculus 650

LECTURE LIV.

SCABIES (*continued*).

PAGE
Symptoms—Pathology—Etiology—Treatment 655

LECTURE LV.

DERMATOZOONOSES (*continued*)—EPIZOONOSES.

Acarus folliculorum—Pulex penetrans—Filaria medinensis—Leptus autumnalis—Ixodes ricinus—Cysticercus cellulosæ—Epizoonoses: pediculi capitis, corporis, pubis, et pediculosis s. phthiriasis—Pulex irritans, cimex lectularius, culex pipiens, œstrus 663

LIST OF ILLUSTRATIONS.

FIG.		PAGE
1.	Vertical section through the skin of the finger tip parallel to the ridges	19
2.	Fat cells	21
3.	Elastic fibrous network	22
4.	Skin papillæ with their epidermis removed and blood vessels injected	23
5.	Vascular and nerve papillæ with epidermic covering	23
6.	Prickle cells with nucleus and nucleolus	24
7.	(Chromo-lithograph.) Section of skin showing injected vessels	26
8. I.	Human touch corpuscles according to Wolff	30
8. II.	Schematic horizontal section of a tactile corpuscle, made through the ending of a nerve	30
9.	A Vater's or Pacinian corpuscle	31
10.	Sweat glands	35
11.	Section through a hair of the beard	36
12.	Cross-section of a hair, below the neck of the hair follicle	38
13.	Sebaceous gland with a lanugo hair	39
14.	Half of a transverse section of a nail through the nail bed proper	40
15.	The lines of cleavage of the skin according to Langer	59
16.	Section of a comedo	133
17.	"Molluscum corpuscles," partly or wholly contained in their epithelial coverings, some entirely denuded	137
18.	Microscopical section of a nodule of molluscum contagiosum	139
19.	Section through a pock in the stage of pus formation (after Rindfleisch)	195
20.	Section through an encapsulated pustule (one-half) (after Auspitz and Basch)	196
21.	Section through a papule of erythema papulo-vesiculosum	215
22.	Longitudinal section through the third intervertebral spinal ganglion of the right side in an attack of zoster lumbo-inguinalis	238
23.	Hæmorrhagic focus in ganglion	239
24.	Red blood corpuscles in a ganglion cell	239
25.	Primary nodule of psoriasis, excised intra vitam (vertical section)	304
26.	Section of a nodule of lichen scrofulosorum	323
27.	Section of an efflorescence of lichen scrofulosorum	324
28.	Lichen ruber planus. Primary nodule (vertical section)	328
29.	Chronic eczema. Vertical section through skin of upper arm	341
30.	Vertical section through an acne pustule	371

FIG. PAGE
31. Hebra's puncture needle..... 379
32. Dermatitis papillaris capillitii (longitudinal section)..... 382
33. From the infiltrated corium in dermatitis papillaris capillitii..... 383
34. Section of a portion of skin at the border between a decolorized and darkly pigmented leucoderma spot (Riehl)..... 421
35. Terminal stage of the mother star with distinct longitudinal division of the fillets..... 429
36. First stage of the daughter stars..... 429
37. Second stage of the daughter stars..... 429
38. Daughter coil after completed division of the cell body..... 429
39. Daughter coil in transition to the resting stage..... 429
40. Vertical section of a villus of condyloma acuminatum..... 434
41. Ichthyosis hystrix (vertical section)..... 439
42. Section of the skin of a negro..... 480
43. Transverse section of the root of the hair near the point of the papilla..... 484
44. Longitudinal section of the root and papilla of a hair..... 485
45. Section through a follicle in process of shedding of the hair..... 494
46. Section of a scar keloid..... 514
47, I. Section through a nodule of lymphangioma tuberosum..... 531
47, II. A portion of the same section (of the corium) greatly magnified, 531
48. Section of rhinoscleroma in the early stage (from the left ala nasi). 535
49. Rhinoscleroma. Nodule from the upper lip..... 536
50. Rhinoscleroma bacilli (encapsulated)..... 537
51. Lupus of the nose (vertical section)..... 549
52. Section of a microscopic lupus nodule under high power..... 550
53. Section of skin attacked by lupus hypertrophicus papillaris et verrucosus..... 551
54. Atypical outgrowth of epithelium (section from nates)..... 552
55. Lupus of the mucous membrane (epiglottis)..... 553
56. Section of a lepra nodule from forearm..... 567
57. Section of a lepra nodule..... 568
58. Bacillus lepræ..... 570
59. Section of a lepra nodule excised during life..... 571
60. Epithelioma (vertical section)..... 609
61. Penicillium crustaceum Fries..... 627
62. Fungi..... 628
63. Favus..... 631
64. Fungous growth from lower portion of favus body..... 633
65. Hair and root sheath in herpes tonsurans capillitii..... 639
66. Section of epidermis in herpes tonsurans maculosus..... 640
67. Onychomycosis trichophytina..... 644
68. Hair from a nodule of sycosis parasitaria (one-half shown) with mycelia..... 645
69. Microsporon furfur, the fungus of pityriasis versicolor..... 648
70. Acarus, sexually mature female, 0.35 millimetre long, 0.50 millimetre broad..... 651
71. Pregnant female itch mite..... 652
72. Itch mite, male ; view of abdomen..... 653

FIG. PAGE
73. Cuniculus, seen under low power 653
74. Larva of itch mite with six legs ; view of abdomen 654
75. Same, second moulting 654
76. Schematic section of the epidermis, cuniculus, and papillary layer of cutis 655
77*a*. Schematic section of a pustule in whose epidermic covering an itch burrow appears 656
77*b*. The same 656
78. Scabies crustosa 658
79. Acarus folliculorum (after Küchenmeister) 663
80. Leptus autumnalis (Küchenmeister) 665
81. Male head louse, with system of air tubes and respiration stygmen (Küchenmeister) 668
82. Hair with nits of the head louse 668
83. Clothes louse, female (Küchenmeister) 670
84. The crab louse (Schmarda) 672

GENERAL DIVISION.

LECTURE I.

RELATION OF DERMATOLOGY TO GENERAL PATHOLOGY—ITS SCIENTIFIC AND PRACTICAL IMPORTANCE—HISTORY OF ITS DEVELOPMENT FROM ANCIENT TO THE MOST RECENT TIMES.

GENTLEMEN :—The subject of skin diseases (dermatology, more properly dermato-pathology) presents to us an important branch of special pathology. It is a broad and rich department, which, although to a certain extent complete in itself, is intimately associated with the other departments of medicine and especially with general pathology.

It is most useful, at the very outset of the study of skin diseases, to realize fully the importance of this fact, and to abandon the notion which may have been previously conceived that we have to do merely with a certain clinical and practical routine which can be turned to profitable account in a physician's calling. The study of skin diseases will, on the contrary, yield more profit from a practical standpoint, and more satisfaction from a scientific one, if we carefully investigate and thoroughly understand the relations and analogies between the diseases of the skin and the physiological and pathological conditions of other organs, the conditions of the vascular, nervous, and lymphatic systems, and the various conditions of the body as a whole.

It will further appear that the pathological processes taking place upon the skin furnish a very instructive means for comparing and investigating the analogous pathological processes of the internal organs, inasmuch as the former are appreciable on the living individual and are accessible to sensory perception and observation, while the latter can be first observed only upon the dead subject, in whom the processes are either completed or are interrupted midway in their course.

So the importance of dermato-pathology is demonstrated in three relations :

First, it teaches us to recognize, understand, and cure the diseases of the skin, an organ indispensable to life ;

Second, it greatly enlarges and extends our knowledge of the pathology of the human body in general by showing the connection between skin diseases and those of other organs and systems ; and

Third, it is able to increase the knowledge we gain from general and experimental pathology, because it deals with processes of disease immediately within the range of sensory perception.

The significance of dermatology is an achievement of modern medicine and is the result of the strictly scientific method which was used in the study of skin diseases at the end of the last century, in conjunction with the enlightenment which the microscopical and experimental studies of the last few decades have yielded, especially those bearing upon the anatomy, physiology, and pathological histology of the skin.

The dermato-pathology of to-day rests upon a well-wrought foundation.

The history of individual skin diseases, as well as many names of these maladies and of their symptoms, which are still properly retained, necessarily lead us back to the consideration of what was accomplished in an earlier age. Indeed, some of these attainments can be rightly estimated only when one gives due consideration to the ideas associated with them at that time.

It would, of course, be remarkable if such noticeable and obvious indications of disease as those which affect the general integument, and so easily attract the attention of the layman by their peculiar color and form, had been overlooked by the physicians, some of whom, like the Greeks, eagerly studied the most obscure processes of the human organism, carrying their researches as far as the development of science, and especially of anatomy, in their day made it possible.

The fact is, we find even in the Old Testament statements concerning different diseases of the skin, and also some contagious diseases of the hair, under the names of *nega*, *bahereth*, *schehin*, *misepahat*, and *zaraath*.

Yet we are not in the position to interpret these terms in a strictly medical sense. In the Septuagint and other versions nega and zaraath are rendered lepra and scabies, and so until recently the opinion has prevailed in literature that the zaraath of the Bible is really leprosy. This is, as I proved years ago, an error. Zaraath, in Leviticus, chapter xiii., signifies nothing more nor less than an angry, possibly contagious skin disease, incurable or nearly so. It is a term which may be applied to leprosy, but also to scabies and certainly to mere burns.

More clearly intelligible pathological ideas and terms for diseased

conditions of the skin are found, however, in the Greek medical writings, even as early as Hippocrates, the contemporary of Socrates and Plato (470–370 B.C.). Thus the names *ἐξανθήματα* (from *ἄνθος*, bloom; *ἐξανθεῖν*, to bloom, *efflorescere*—skin efflorescences) and *ἐκθύματα* and *ἐκθίλατα* for eruptions, spots, and skin diseases generally, were used in a general way, just as the modern expressions are used by the physicians and laity of to-day.

Further, *φύματα*, *φύγεθλα*, *τέρμινθοι*, *ἐπινυκτίς*, *ἄνθραξ* are used for prominent or inflammatory swellings of the skin; *λειχῆν*, *λόποι*, *λέπρα*, *πιτυρίασις*, *ψῶραι* for dry diseases of the skin characterized by exfoliation of the epidermis, sometimes accompanied by itching; while *κνησμός* and *κνίδωσις* are used for itching and burning of the skin; *ἱδρῶα* for sweat blisters (sudamina); *φλύκταιναι*, *φλυζάκια*, *ψυδράκια*, *ἄχωρες*, *κηρίον*, *πομφοί*, for vesicles, bullæ, and eruptions associated with moisture or crust formation; *ἕρπης*, *ἐσθιόμενος*, and *κεγχρίας* for peripherally spreading, so-called "serpiginous," superficial or more deeply penetrating skin affections; *ἀλφός*, *λεύκη μέλας*, *ἐφήλιδες* for color changes and pigment anomalies of the skin; *μαδίσιες*, *μαδάρωσις*, and *ἀλωπεκία* for the different forms of diseases accompanied by loss of hair; *ἀκροχορδών*, *ἀκροθύμιον*, *μυρμηκίαι*, *ἰόνθοι* for warts and pimples; *ἐρυσίπελας*, *φαγέδαινα*, *γάγγραινα*, *ἐρυθήματα*, *πετέχιαι* for processes called even to-day by similar names; *χοιράδες* for scrofulous swellings.

It is not to be doubted that while Hippocrates regarded certain more or less important diseases of the skin as independent or idiopathic affections, others, which he designates as "abscesses," he considered the result of certain inward maladies or expressions of failing general health or a febrile state. He speaks of the so-called critical eruptions with which febrile diseases end, and believes that eruptions disappear spontaneously or as a result of treatment of the inner organs. and by their disappearance may cause these to become affected; that, *vice versa*, certain excretions and depletions—a hæmorrhoidal flux, for instance—may free one from certain skin diseases. Finally, theories are not wanting in regard to the causes of skin diseases. the idea being that the accepted "humors" were a factor in producing some skin affections, and that the season of the year, the state of the weather, the direction of the wind, or the age and sex of the individual exerted an influence upon others.

After Hippocrates, whose writings furnished the basis of medical studies for a thousand years longer, Cornelius Celsus deserves prominent notice on account of the attention he paid to skin diseases. This indisputably most objective of the ancient medical writers, who lived in Rome from 53 B.C. to 7 A.D., and who about 18 B.C. published his medical work of eight books, still worth reading to-day, gives in the third, fifth, and sixth books a tolerably correct and sys-

tematic treatise on skin diseases, one which is free from fantastic theories and which in substance and form is hardly surpassed by the work of any writer up to the eighteenth century. We find not only the ideas of Hippocrates more concisely defined and classified, but his nomenclature improved or replaced by new Latin terms, most of which are in use to-day, and the pathology of skin diseases enriched by some exact descriptions. Celsus in the third book unmistakably describes Elephantiasis, in the fifth book dwells upon the treatment of wounds and ulcers (Vulnera, Ulcera), and in special chapters calls attention to a list of skin diseases which he designates as : Carbunculus, Carcinoma, Therioma (Phagedæna), Ignis sacer, Ulcera ex frigore (Frost-ulcers), Furunculus, Phyma, Phygethlon, Abscessus, Fistulæ, Kerion, Acrochordon, Thymion, Myrmekia, Clavus, Pustulæ, Scabies, Impetigo, Papulæ, Vitiligo. In the sixth book are chapters entitled de Capillis fluentibus, de Porrigine, de Sycosi, de Areis, de Varis, Lenticulis, et Ephelide ; in the seventh, de Condylomatibus, de Varicibus, de Gangræna. Most of these terms are still in use to-day, although with a somewhat different meaning. For instance, Celsus included under Pustulæ not only purulent efflorescences, but also urticaria and sudamina ; under Scabies, an itching affection of the skin associated with desquamation and with moisture and crust formations, or what we to-day call eczema. Other terms, such as Sycosis for an affection of the bearded part of the face, and Porrigo for scald-head, are still used to-day or have been until very recently ; not to mention the names previously given for warts, corns, etc.

About the same time Pliny announced the appearance in Rome of a contagious skin disease, Mentagra, and he also, almost simultaneously with Scribonius Largus, mentions shingles under the name of Zoster or Zona ; while other writers, especially Aretæus, discuss Elephantiasis, a disease which was then just beginning to be prevalent in Italy.

Galen, who flourished in the second century of our era, reproduced, with many comments, in his elaborate works the materials gathered by Hippocrates and Celsus, so that the writers of the next few centuries drew principally from him. Deserving of special prominence among his followers are Aëtius of Armida (543 A D.), who first used the term *ἐκζέματα* ; Paul of Egina, who treated more in detail of specific skin diseases ; Oribazius, Alexander Trallianus, and Actuarius, who greatly advanced the knowledge of the ancient Greeks by concise descriptions of disease.

The Greek systems of medicine, however, including dermatology, for the most part disappeared from their native home amid the political confusion and martial disturbance which characterized the last days of the Western Roman Empire and the beginning of the middle

ages, and began to reappear in Western Europe in the eighth century, some of them greatly enriched by new and important discoveries made during their roundabout journeys through India and Arabia. In the medical works of the Indian writers Charaka and Sushruta, which must be assigned to the period between the fifth and the ninth centuries, are described not only the skin diseases mentioned by the Greeks, but also small-pox—Másúriká—in its various forms and dangerous complications ; probably measles ; and further tubercular and anæsthetic leprosy—Kushtá and Bátārakta—and the disease, until then unknown in the West, but which was later introduced into literature as Elephantiasis Arabum (Pachyderma).

The Arabian writers Razes, Serapion, Ebn-Zor, Haly-Abbas won prominence and authority, however, not merely by preserving and elaborating the teachings of the ancient Greeks, but by their new and practical contributions to the world's knowledge of skin diseases. Above all, their description of the symptoms of leprosy—*djudzam*—continued to be a standard authority, as well as their division of it into four kinds which evidently correspond to the four humors of Galen —*i.e.*, Lepra elephantina, due to an excess of black bile ; Lepra leonina, traceable to the red bile ; Lepra alopecia, dependent upon the blood; Lepra tyria, upon mucus (*i.e.*, phlegm). Furthermore Albarras (black and white) and Morphæa, probably identical with the Vitiligo, Leuke, and Melas of Celsus, are given as forms of leprosy. *Dal-fil* is the Arabian term for Pachyderma, which was not known to the Greeks, but which corresponds to the Elephantiasis of the translators from the Arabic—that is, to what was afterward called Elephantiasis Arabum. Besides small-pox and measles, the Arabs described very minutely the diseases of the scalp. Avicenna calls them *Sahafati*, evidently etymologically identical with the Hebrew Sapahat; while Haly-Abbas, on the other hand, uses the term *Alvathim*, from which the best translator from Arabic, Stephen of Antioch, derived the word Tinea, still in use to-day for scald-head. Among the five kinds of tinea we recognize the description of what to-day is known as contagious favus or ring-worm. Avenzoar mentions the true scabies together with its causative, Acarus scabiei.

The truly admirable researches of the Arabs were transmitted to the Western medical world through translations into Latin, made between the tenth and fourteenth centuries by the founders and successors of the medical school at Salerno, Constantinus Africanus, Rogerius, Rolandus, and others. In this way the acquaintance with the work done by the Greeks was renewed. Leprosy, however, was the one form of skin disease which almost exclusively occupied the attention of writers from the eleventh to the end of the sixteenth century : the Italians, Vitalis de Furno, Wilhelm von Saliceto, Lanfrancus, Montagnana ; the Spaniards, Theodoricus, Villanova ; the

Englishmen, Glanville, Gilbert, Gaddesden ; in France, Gordon, Guy de Chauliac ; in Germany, Hans Gersdorf and others. For leprosy had at just that period, in the twelfth and thirteenth centuries, developed into a really universal plague which challenged the attention of governments and of all classes of society, as well as of the medical profession. But the physicians made no progress in their views as to the character and treatment of leprosy beyond those which had been taught by the Arabs and promulgated through the school of Salerno. In the course of the fifteenth century leprosy gradually disappeared from the inland countries of Europe On the other hand, toward the end of this century a new epidemic appeared —Lues venerea, later known as Syphilis. Then opportunity was offered to discuss the numerous skin affections which are peculiar to this disease. But although the number of authors who at the end of the fifteenth and in the first decades of the sixteenth century engaged in the study of syphilis is very considerable, and among them are well-known names like Marcellus Cumanus, Musa Brassavolus, Gabriel Fallopia, Fracastorius, yet the positive results of their investigations relative to syphilitic diseases are very insignificant.

Nevertheless the sixteenth century may be regarded as the period in which the treatment of skin diseases began to be more independent and gradually emancipated itself from the traditional formulas of the Arabs. Besides the syphilitic eruptions which were, to be sure, still held to have close relations theoretically and etiologically with leprosy, they learned to recognize as specific kinds of skin diseases scorbutus, the petechiæ of fever, and the acute contagious diseases. The efforts increased to treat of skin diseases as such in a purely pathological sense.

John Manardus furnished, under the title of Lactumen, a thorough description of moist sycosis or eczema capitis of infants. Gorræus, besides other works, prepared a dictionary of synonyms very useful for the understanding of dermatological terminology ; Blondus, a monograph on maculæ corporis ; Ambroise Parée, besides much else, one monograph on small-pox ; Forestus, Schenk von Grafenberg, Montagnana furnished more accurate accounts of pemphigus, contagious scabies, and the different kinds of tinea ; not to mention the consideration which the descriptions or etiological relations of most skin diseases previously known received from many authors who, like those referred to in the " Aphrodisiacus " of Aloysius Luisinus, had treated of syphilis toward the end of the fifteenth and in the first half of the sixteenth century.

After all this, it is not surprising that at this time a great work was produced which deals exclusively with the pathology of skin diseases. It was founded upon the lectures of the Venetian Hieronymus Mercurialis and published by his pupil, P. Aicardius, in 1572, " De Morbis

Cutaneis," and is therefore the first purely dermatological work. Mercurialis, to be sure, does not show much originality in this work. He divides skin diseases, as did Galen, into those of the head, embracing the different forms of baldness and of tinea, and those of the rest of the body. As for the rest, Mercurialis' work is, in its descriptions and theories, really only a compilation from the Greek, Roman, and Arabic medical writings.

From this time on the number of authors increases who devoted either single chapters in general medical works or monographs and longer treatises exclusively to the pathology of skin diseases. Let us mention among those writing at the end of the sixteenth or during the seventeenth century, besides Fernelius, Vidus Vidius, and Sennertus, who speaks more in detail of various well-described skin affections, but especially of the acute exanthemata, Döring, who first described scarlatina in unmistakable terms; John Dolæus, who even then defined lupus according to the modern idea; but, above all, the everywhere cited Hafenreffer, in whose work, published about 1660, the whole subject of skin diseases is considered, and who mentions the Acarus scabiei under its popular name of "itch mite," and the comedones as supposed "worms." In the medical works of John Dolæus, already mentioned, and of Sydenham, Van Swieten (Boerhave), and De Haën, which appeared about the last of the seventeenth and beginning of the eighteenth century, we find some very valuable discussions upon our subject. Yet dermatology as a systematic science, a special branch of medicine, has been developed between the year 1750 and the present time.

Daniel Turner introduced this period with a very valuable work on skin diseases; Astruc produced a great work on syphilis and the non-syphilitic skin affections; Sauvages, together with pathological details, important historical contributions; Hensler, historical studies on leprosy and syphilis, which still rank as models.

As embracing the whole territory of skin diseases, and alike remarkable for its thorough historical and pathological contents and for its classic diction, we should mention the great work of Lorry, the Parisian, published in 1777, "Tractatus de Morbis Cutaneis." Lorry's work not only contains the collected dermatological literary material of his predecessors, but we also find in it a rich abundance of clinical facts well digested and objectively described, and definitions of disease remarkably clear and logical—as, for example, in case of ulcers—and a comprehension of the general pathology of skin diseases extending far beyond the narrow horizon of simple description. Lorry, in addition to the visible clinical characteristics of skin eruptions, considered their relation to the anatomical and physiological peculiarities of the skin as well as of the whole organism.

He divides them into idiopathic and apostatical, distinguishing

them, according to extent and location, as general and local ; he also distinguishes those which involve special skin tissues, those which result from general or local, and mechanical or toxic causes, and exhaustively discusses skin diseases pathologically and therapeutically from every point of view open to the medical science of that age.

In spite of its rich contents and the classical spirit which characterizes it, Lorry's work advanced dermatological knowledge among the greater portion of the professional public but little, because the book was too learned and it cost too much labor to extract from it its meaning. The little volume of our fellow-countryman, Plenck, published at Vienna in 1776, had therefore infinitely more effect. In this all skin diseases are given according to the form and aspect which they present directly to the eye as finished products of Nature, as maculæ, pustulæ, vesiculæ, bullæ, papulæ, crustæ, and accordingly divided into fourteen classes. These, it is true, included one hundred and twenty subdivisions or species of disease, which apparently made the whole system very difficult ; but the characteristics of both classes and species were briefly and comprehensively defined. very much as in the botanical system of Linnæus, where the orders, genera, and species of plants were determined by the appearance of the flowers and the number of their stamens. And so Plenck's "Doctrina de Morbis Cutaneis," on account of its concise and convenient rules, was held to be a sort of catechism, a simple and trustworthy clue in the confusing labyrinth of disease. But as soon as men began to study skin diseases on the living individual they were at once convinced that these present no such fixed appearances as Plenck's definitions assume, but exceedingly changeable ones, and would thus require to be placed sometimes in one, sometimes in another of his classes ; that which one day appeared as a papule, after a few hours presented itself as a vesicle and soon afterward as a bulla or ulcer. There is the further objection that, dealing only with the outer form, not with the inner pathology, Plenck puts together in one class quite different pathological processes, as scabies and variola, acute exanthemata and lenticula, leprosy and cutis anserina, besides committing many special pathological enormities like the presentation of a scabies syphilitica and similar diseases. The presentation, however, of fixed notions of the primary forms of skin diseases was of permanent value to the future study of dermatology, and in a certain sense and to a certain degree it removed the capriciousness in terminology and advanced sound knowledge. This beneficial influence was immediately apparent in the fact that Robert Willan adopted in his epoch-making work on skin diseases the system of Plenck, although he reduced it to only nine classes: (1) papulæ, (2) squamæ, (3) exanthemata, (4) bullæ, (5) pustulæ, (6) vesiculæ, (7) tubercula, (8) maculæ, (9) excrescentiæ.

In his descriptive text book begun in the year 1798, and completed after his untimely death by his gifted pupil and friend, Bateman, and published in London in 1799 under the title "Description and Treatment of Cutaneous Diseases" (also in a German translation by Friese), and in the "Synopsis of Cutaneous Diseases according to the Arrangement of Dr. Willan," published in 1815, Willan furnished for all time intelligible and correct, clear-cut descriptions of skin diseases, both of those previously known and of certain ones which he had recently observed; he also, through his objective description of the course of the diseases and recommendation of more rational methods of treatment, promoted the pathology and therapy of skin diseases; and, finally, he furnished a broad and firm foundation for the further study of skin affections by simplifying and establishing medical terms and synonyms, being aided in this by his thorough acquaintance with ancient medical works. Although Willan and Bateman's works could not fail to exert a mighty reformatory influence upon their English contemporaries, and by means of many translations, like those of Haneman, Sprengel, Blasius, upon foreign physicians also, yet this only gradually took practical effect. Almost independently of this a sudden and vigorous development of dermatology took place; first of all in France, which, begun and stimulated by the earlier researches of Lorry, Sauvages, Roussel, and Poupart, and maintained by means of the rich material of the Hôpital St. Louis, Paris, is associated with the celebrated names of Alibert, Biett, and many others. Alibert, as teacher, writer, and physician, apparently dominated dermatological study in France during the first three decades of this century. The system of skin diseases presented by him, and promulgated in a large illustrated work first published in 1806, was a so-called natural one. The *teignes* and *dartres* play leading rôles in it. In his work appearing in 1832 for the first time, he made some undeniable concessions to Willan's ideas in his presentation of a new system.

On the other hand, Biett had at once adopted Willan's system, and, although at the time less famous than his colleague Alibert, he accomplished vastly more permanent results through his lectures published by his pupils Cazenave and Schedel in 1828.

Still more, perhaps, is this true of the work of Rayer, whose comprehensive, thorough, scholarly work, "Traité des Maladies de la Peau," published in Paris in 1835, contains many clinical facts that are instructive to the reader of the present day. With the researches of the authors named the influence of what we may call the French school in dermatology, extending far beyond the borders of France, reached its highest point. Still, France produced later a considerable number of writers on dermatology, among whom Hardy, Cazenave, and Bazin especially did original work in many directions.

The prominence which its independence lent to the French school brought with it in course of time the unavoidable disadvantage that it resisted for a long time moving to the general scientific territory which had been prepared for modern dermatology since Hebra's time by the help of pathological anatomy and general pathology and indirectly by the aid of collateral sciences. All the more rapidly and successfully has this transition taken place during the last few years, thanks to the skilful researches and energetic efforts of a considerable number of dermatologists and histologists which France now has to show, and whose acquisitions, similar and equal to those of other nations, are clearly reflected in the medical journal conducted by Besnier and Doyon, as well as in numerous recent treatises and monographs.

What had been accomplished in dermatology in Germany up to the forties was of incomparably small value. Both the earlier writers upon cutaneous diseases, Peter Frank (1792) and Struwe (1829), and the later Riecke (1841), Schönlein, and C. H. Fuchs, endeavored to carry out fully in dermatology the humoral pathological views governing the general pathology of their time. This effort found extreme expression in the statements of Fuchs, who, in his three-volume work of 1840 on skin diseases, referred to the labors of Willan and the French scholars and vindicated the important influence upon the origin and character of the different skin diseases of the rheumatic, catarrhal, chymotic, erysipelatous, and other dyscrasiæ and conditions of the organism. At the same time he sought, like Schönlein, to adapt to skin diseases the so-called natural systems which had been introduced into botany and zoölogy. Schönlein had already taught that skin diseases, like plants, have stages corresponding to germination, growth, blossom, maturity, fruit formation, and decay. The arrangements of skin diseases in families, genera, species, and varieties appeared to Fuchs only a logical claim. The effort to carry out the comparison in all possible directions complicated the system of Fuchs very much and made it difficult of comprehension—a fault which was even increased by his new nomenclature, which contained such terms as "chymoplanien," "dermapostasen," and the like. The Schönlein-Fuchs teaching, in its effort to be natural, became perhaps the most unnatural and artificial of all, and it never acquired any considerable influence. In the meantime much preparatory work had been done in medical natural science, which was destined on the one hand to overthrow the ontological and humoral notions in pathology, on the other hand to afford new information and a positive basis for dermatological pathology. The discovery of the Acarus scabiei had been made centuries before, but had only recently been verified and acknowledged; and in this, as in cases of muscardine in the silk-

worm and favus in man, people had learned to recognize causes of disease which are entirely unconnected with the state of the blood and of the "humors" of the individual, and accordingly irreconcilable with the dyscrasia theories. The path was broken for the comprehension of many appearances of the diseases of the general integument by the diffusion of authentic knowledge concerning the histological conditions and physiological functions of the skin.

Besides the sebaceous glands, whose existence had been known since Malpighi's day, and which were adduced by Morgagni, Boerhave, and Cotugno in explanation of the eruptions in skin diseases, the sweat glands had been made known by Brechet, Roussel de Vauzème (1834), and Gurlt. Wendt and Henle had thrown light upon the structure of the skin, Berres and Fohmann upon the structure and course of the lymph and blood vessels. The existence of organic muscle fibres surrounding the glands of the skin had been proven by Kölliker, and the peculiar nervous apparatus of the skin by Wagner and Meissner, while the researches of Favre, Schottin, E. H. Weber, and others furnished a clearer insight into the secretory and other functions of the skin.

A new spirit animated and a new direction was given to the study of medicine in the forties (1840–1850). The *a priori* theory was cast aside and the science consequently freed from the thankless task of adapting facts to it.

Pathological anatomy had become the basis of medical study. It represented the scientific knowledge of the facts with regard to the tissue changes resulting from the progress of disease. Its comprehension logically demanded the observation of the course of disease undisturbed by therapeutic agencies, and the physical examination of the diseased appearances. The former was begun by Rokitansky, the latter by Skoda, and these men thereby became the founders of the new school of medicine, and, to speak more definitely, of the Vienna school.

What these two original minds did for pathology in general, Hebra, their pupil and our master, too early taken from us but never to be forgotten, accomplished for dermatology in particular. Hebra became the creator of the new dermatological school which is called by his name.

Disregarding traditional classifications of disease, he kept to the conditions established by Rokitansky and evident upon the skin, which appear as hyperæmia, inflammation, new tissue formation, effusion, etc. Following Skoda's example, Hebra diligently studied the physiological course of disease upon the skin; he made use of experiments for producing and carefully regulating artificial disturbances of the skin ; and he took note of the variations from the nor-mal course of disease caused by different agencies, including

therapeutical ones. By this means Hebra succeeded in proving the idiopathic nature of many diseases, and hence in showing that it was absurd to regard the psoric, herpetic, scrofulous, arthritic, and other dyscrasiæ as causes of all skin diseases; and thus he initiated a treatment of cutaneous disorders free from the influence of preconceived ideas.

Fortified by a thorough knowledge of the literature, he sifted the enormous amount of material which had been transmitted upon the subject, threw out the useless, preserved the valuable, clearly classified and described distinctly for all time the forms of disease, grouped together many scattered disorders which had natural connections with one another, confirmed descriptions of newly recognized forms of disease, and thoroughly reformed and reconstructed the pathology and diagnosis of skin diseases. Hebra developed the science of skin diseases so methodically and so completely upon this newly created positive basis as to place it on a par in many ways with the exact sciences.

In consequence of Hebra's scholarly treatment of skin diseases a result followed which had not been anticipated. The therapy, formerly founded upon all kinds of prejudices, superstitions, and uncertain caprices, or based upon the ignorance of a haphazard science, was, for the larger number of skin diseases, converted into a definite and successful course of treatment. It was reinforced by an exact knowledge of the course of disease, and to a certain extent by a knowledge of its causes, no less than by the experimentally determined physiological action of medicines.

The great personal influence which Hebra exerted in the course of his teaching must not be overlooked. He had great power as a teacher and writer because he never became wearisome, his lectures and treatises being clearly and logically expressed; and as a practical physician and clinician because of his highly objective and therefore unfailing exactness and readiness in diagnosis, and because of his successful method of treatment. So it will be readily understood that the new science in a short time won over to its side the majority of practitioners and of the older students.

Proving itself scientifically suggestive, satisfactory, and practical to the highest degree, the new science was destined to be the standard and, in its main principles, the ruling one.

This was brought about through Hebra's works, the first of which, his celebrated "Treatment of Scabies," appeared in 1844. This not only deals with the factors causing this disease, but exposes clearly the chief ideas which Hebra so pronouncedly held relative to skin diseases in general. In spite of the storm of indignation, doubt, and opposition which this work aroused in the lair of inherited doctrines, Hebra, struggling against many attacks, courageously main-

tained the theory of his new teaching, "objective diagnosis." In 1845 Hebra presented his system of skin diseases founded upon a pathological and anatomical basis, according to which they are arranged in twelve classes, dependent upon the cardinal pathological tissue changes which had been determined by Rokitansky. They are :

Class I. Hyperæmiæ cutaneæ.
II. Anæmiæ cutaneæ.
III. Anomaliæ secretionis glandularum cutanearum.
IV. Exudationes.
V. Hæmorrhagiæ cutaneæ.
VI. Hypertrophiæ.
VII. Atrophiæ.
VIII. Neoplasmata.
IX. Pseudoplasmata.
X. Ulcerationes.
XI. Neuroses.
XII. Parasitæ (skin parasites).

By means of this very simple system, marked by undoubted scientific characteristics and capable of further modification—a system which refers each pathological change in the skin at once to its precise place—its author is enabled to assign and arrange the numerous and different processes of diseases of the skin into natural groups.

Wherever the knowledge of pathological and anatomical changes is held to form the weightiest and most positive basis of clinical study, there has Hebra's classification of dermatology as a whole, or but slightly modified, at least in its chief characteristics, been accepted, even when in other respects important deviations from the Hebra school are found.

Aside from this intellectual reformation Hebra advanced in a most marked manner the material development of dermatology, thanks to the unusually rich clinical material which his name and the institution bearing it attracted, and thanks to the successful way in which Hebra therapeutically and didactically turned it to account.

Besides the numerous large and small treatises, in conjunction with Elfinger and Heitzmann in their great atlas of skin diseases—unsurpassed in beauty and exactness—his complete "Handbook of Skin Diseases" (to the second part of which I was so fortunate as to contribute) furnishes great assistance, in the study of dermatology, to the medical profession of to-day. Hebra's influence as a teacher was no less than that exerted through his literary productions. In his lectures for more than three decades to physicians from all parts of the world, thousands have received, besides the rich treasures of an enormous unrestricted and freely given clinical experience, the

privilege of becoming acquainted with the more important methods of study peculiar to the Vienna school, the presentation of processes of disease of the skin, their objective diagnosis, and their effective therapy ; partly to be used by themselves for the practical relief of suffering humanity, and partly to be taught by them. as devoted pupils of Hebra, to the youth of other lands.

Mention must be made that Hebra, since he considered his monumental work in no way complete, and since he therefore labored most vigorously to elaborate and complete it, was always ready to assist, by sympathy, advice, and material aid, each one of his many students who were co-workers and co-thinkers in his efforts. So our master Hebra during his life was the honored head of a scientific body which here, as on the other side of the ocean, in the Vienna spirit of the Hebra school (bearing for all time the name of its founder), struggled for the successful development of dermatology. We should not do justice to the traditions of our school if we should overlook the fact that since the forties, contemporaneously with Hebra, much that was advantageous to the development of the science of skin diseases was accomplished by others outside of the Vienna school. Among the Germans who made original contributions are Bärensprung and G. Simon, who at their clinic made the pathological anatomy of skin diseases an object of valuable study ; and the twin Scandinavian investigators, Boeck and Danielssen, who threw the first ray of light over the obscure subject of leprosy and who have found worthy followers in a number of younger workers. In England it is chiefly the clinical and practical side of dermatology that found eager promoters in Plumbe, Anthony Todd Thomson, Jonathan Green, Erasmus Wilson, Tilbury Fox, and many others, largely more recent authors; while, as already mentioned on page 9, dermatology in its most fruitful manner was promoted in France, as it was also in America and Italy, by a great number of skilful individual workers, for the greater part graduates of the Vienna school.

The importance of dermatology practically and pathologically has in the last few years been generally conceded. Hence in many countries associations for studying this subject have been founded, and many individual workers have arisen who have by their great works and achievements advanced this branch of science. Among these are to be found well-known celebrated names, and especially some successful and practical dermatologists who have gone directly from Hebra's school to settle here and in Germany, and whose names it would be impossible to enumerate exhaustively here. We will have occasion to become acquainted with them in their special lines when we study the pathology of special skin diseases.

The line of development which general pathology has taken since

the fifties has established the fact that the knowledge of the nature of skin affections has been advanced not only by those who have chosen it pre-eminently as their special study, but by others also. The most distinguished workers in other fields of medicine—namely, in pathological anatomy, histology, surgery, mycology, and bacteriology—have directly and indirectly contributed to the advance of dermatology. The facts with regard to the parasitic nature of certain skin diseases awaken the greatest interest in the botanist, and especially the mycologist and zoölogist, to whose exact methods of investigation dermatology has theoretically and practically much to be thankful for. The efforts to clearly understand the significance of inflammation and suppuration and the accompanying phenomena in the circulatory organs and the tissues have for a long time been aided by a very fitting object lesson upon the skin, as was made evident almost four decades ago in Henle's paper on "Mucus and Pus and their Relation to the Skin." Surgeons study upon the skin the processes most interesting to them, the healing of wounds, granulation, and the formation of cicatrices and epidermis.

The study of the inflammations and neoplasms of the skin is satisfactorily accomplished in proportion as the pathological anatomy of recent times, which is really pathological histology, is studied and the explanation sought in it for the tissue changes in inflammation and for an insight into the character and origin of new growths. Physiological histology and the study of development—embryology—which is the basis of pathological histology, can no longer be excluded from the list of general objective studies, since in these branches is to be found the explanation of many new growths and ulcers. Many functional disturbances of the skin, of which the neurotic are the most interesting, direct us to the study of the physiological function of the skin and the distribution and function of the cutaneous nerves, including the sensory, vaso-motor, secretory, and much-disputed trophic nerves. So the pathological territory of dermatology always widens, even to the border of general pathology, into which dermatology sends its offshoots, each of these two branches of science being mutually dependent. Viewed from such a standpoint dermatology is no longer to be considered a special isolated study nor simply as a subject for routine practice, but in the widest sense is to be recognized, at the very outset, as a desirable and necessary complement to the science of pathology and as an indispensable and beneficial contribution to the medical profession.

LECTURE II.

GENERAL CHARACTER OF THE PATHOLOGICAL PROCESSES AFFECTING THE SKIN—THEIR ESSENTIAL CONFORMITY TO THOSE OCCURRING IN OTHER ORGANS AND TISSUES—THEIR APPEARANCE MARKED BY SPECIAL CHARACTERISTICS PRODUCED BY THE PECULIAR ANATOMY OF THE SKIN—THE SYMPTOMS AND CAUSES PECULIAR TO SKIN DISEASES—ANATOMY OF THE SKIN AND ITS APPENDAGES.

THE processes of disease which are observed upon the human skin by no means differ from those of the other organs of the human body. They are, in the widest sense, the results of quantitative or qualitative alterations of nutrition or function. Let us not forget that the general integument does not, as the laity think, form a simple *involucrum corporis humani*. Indeed, it is a very complex organization, attached by its basement tissue to the fascia below, while through its ramifying blood and lymph vessels and its radiating nerve branches it is vitally associated with the centres of nutrition and innervation of the general organism; it is subject to the same productive and functional laws as are all the other organs and tissues of the human body. Therefore it is not to be expected that an alteration in the nutrition and function of the skin—that is, a disease of the skin—develops differently from the diseases of other organs. Indeed, the skin is subject to the same pathological changes as are all other organs—viz., hyperæmia, hyperplasia, and inflammation with its recognized sequelæ, resolution, suppuration, gangrene; atrophy, degeneration, new tissue formation, neuroses, etc.; and the more familiar we are with general pathology and pathological anatomy, the better are we able to recognize changes in the pathological processes of the general integument. And yet the diseases of the skin have undeniably their peculiarities and differences, which render their analysis and recognition difficult and require special study.

The skin is very peculiar in its anatomical arrangement, both as regards its glands and its epidermal covering, and also in its specific functions, the most important of which are the regulation of heat, respiration, secretion, and sensation. On account of these peculiar anatomical relations, special forms of disease are possible which

cannot occur in other organs, because the latter do not possess certain organic and tissue elements, and consequently do not exhibit the specific functional disturbances which correspond to such elements.

The skin being an exposed organ, its diseases are peculiar, in that their symptoms are directly perceptible to the sense of sight and touch and can be directly observed, as changes in color, arrangement, consistence, general aspect, and external relations—in fact, in all the physical attributes of the skin—phenomena which in diseases of other organs are largely unknown and which therefore must here be studied *de novo* or at least receive special attention.

Finally, the peculiar nature of skin diseases will suggest special etiological factors; for the general covering of the body, being exposed, is subjected to a number of external influences, as high and low temperature, mechanical and chemical irritants, parasitic animals and plants, etc., and hence becomes affected in a way which is less common in the more inaccessible organs.

From these observations it follows that it is necessary, for the general comprehension of skin diseases, to present these three considerations:

1. The anatomy and physiology of the general integument;
2. The general symptomatology; and
3. The general etiology of skin diseases.

ANATOMY.

The general integument—integumentum commune—as its name signifies, forms a membranous covering for the surface of the body, investing its different parts. At the great orifices of the body it is continuous with the mucous membrane of the cavities. Its surface is not everywhere of the same appearance and feel; aside from the varied color of special parts, it looks uneven and has a woolly or velvety feel. This arises from certain *inequalities* of its surface, which are produced by furrows, ridges, pores, and hairs. On the so-called hairy portion of the body the skin is covered with long hairs, at other points with fine, thin, so-called wool hairs—lanugo hairs. Only the palms of the hands and the soles of the feet, the dorsal surface of the terminal phalanges of the fingers and toes, the glans penis, inner surface of the foreskin, and the borders of the lips are lacking in hair. The *furrows* on the surface of the skin are long and deep, dividing it into large areas; and superficial and short, subdividing the former into smaller, generally oblong spaces. The former correspond chiefly to the flexures of the joints, as in the palm of the hand, or to certain fixation fibres of the cutis which unite it firmly to the underlying structures. The smaller furrows, running chiefly in the spaces between the papillæ and mouths of the

hair follicles, are, as the studies of O. Simon have revealed, chiefly dependent for their direction upon the tension of the skin, and correspond with the tension lines of Langer, which will be spoken of later. Lewinski has sought to attribute the longitudinal and transverse furrows of the surface of the skin to the repeated folding of the skin, the cutis becoming altered by the movements and the traction exerted by the muscles. This theory is opposed by the fact that the degree of tension possible for the skin of the palm of the hand and of the sole of the foot is disproportionate to the folds found there, and that the folds are to be recognized on the fetus at a time when muscle action cannot be considered. The furrows and lines which form the small subdivisions of the skin surface are doubtless the plastic expression of the direction of growth, arrangement, and grouping which the different parts of the skin—the papillæ, glands, hair, and connective-tissue fibres—have assumed, as Blaschko in his researches seems to have conclusively proved. The epidermis shows on its under surface, next to the corium, prominent longitudinal processes which correspond to the ridges of the surface; at their summit open the sweat glands, and Blaschko calls them, therefore, *gland columns*. Between two such is another less pronounced projection which corresponds to the furrow of the surface, and it is owing to this that the skin in all its layers appears to be folded inward. This second process, according to Blaschko, constitutes the fold. Gland projections and folds are united at regular distances by transverse lines, whereby the wedge-shaped epidermal depressions are formed which contain the individual papillæ, causing the under surface of the epidermis to have a loosely woven, honeycomb structure. The furrows and subdivisions are more fully developed upon the extensor surface of the extremities and joints, and also on the lumbar region, than on the flexor surface of the extremities and anterior portion of the trunk; in many skin diseases this difference is neutralized or reversed. Later similar researches by Philippson, Unna, and James Loewy have led to analogous conclusions concerning the significance of the furrows and lines of the skin surface.

Aside from the pronounced furrows and lines on the surface of the skin, there are to be seen fine circular depressions or *pores*, which for the greater part correspond to the mouths of the hair follicles and sebaceous glands, as upon the nose; or to the openings of the sweat glands, as upon the ridges in the hollow of the hand. These ridges, which correspond to the regularly arranged skin papillæ, are to be seen as delicate curved lines on the volar surface of the last digital phalanx. Finally, the *colors* seen upon the skin vary greatly; appearing sometimes as a diffuse, mottled, and ramifying redness, which results from the overfilling of the small blood vessels and

capillaries; sometimes as various shades of brown, proceeding from pigment in the epidermal strata—this pigment being found, in most of the Caucasian race, in the areola of the nipples and in the scrotum and labia, and being most pronounced in individuals of the dark races, to whom it gives their general dark color. The universal white color of the skin, as Max Joseph has rightly proven, is an optical impression due to the reflection of light through the transparent cutis and epidermis from the opaque subcutaneous structures, fat layers, and fascia.

The skin is, with the exception of the hairy scalp and the skin over the chin, sternum, linea alba, and glans, more or less easily movable and capable of being pinched into a fold; as a rule this is more decidedly so on the extensor than on the flexor surface.

As the skin shows great differences in the above detailed external characteristics, so does it also in its anatomical arrangement, which

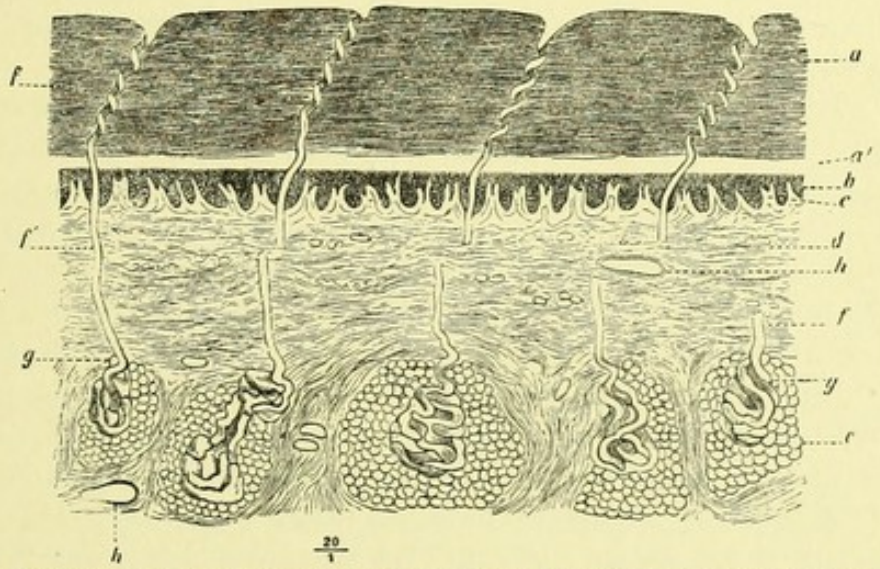

FIG. 1.—VERTICAL SECTION THROUGH THE SKIN OF THE FINGER TIP PARALLEL TO THE RIDGES

a b, epidermis; *a*, stratum corneum; *a'*, stratum lucidum; *b*, mucous layer; *c*, papillæ; *d*, cutis; *e*, fatty layer; *f*, excretory duct of the sweat gland extending into the cutis and running spirally in the epidermis; *g*, sweat gland coil; *h*, section of blood vessel.

depends upon its local and functional office, since in certain localities some constituents of the skin are more or less quantitatively and intensely developed or are utterly lacking. Aside from these local differences, the skin in general has essentially the same typical structure.

On a good section made perpendicularly through the cutis, as illustrated in Fig. 1 (after Henle), which represents a thick section through the skin of the finger tip, one can readily distinguish three *luyers*. The upper (*a b*), the epidermis, is sharply defined, and its prolongations fit closely the corresponding ones of the second, or middle layer of the skin. The latter is of uniform and dense appearance; it is called the cutis, derma, or corium. At regular intervals on

its upper sharply defined border are large or small, conical, pointed, club-shaped prominences—the papillæ of the skin; by these the corium is united to the corresponding depressions and elevations of the upper layers. At its lower portion the corium merges gradually, without any line of demarcation, into the loose layers of the subcutaneous connective tissue, which is known as the tela cellulosa, adiposa, or subcutanea.

This *subcutaneous connective tissue* consists of a coarse network of connective-tissue bundles which, starting from the underlying fascia or periosteum, pass in an oblique direction, forming anastomosing fasciculi or coarse bundles which are continuous with those of the cutis proper. Layers of fat lobules are found from place to place in the connective-tissue spaces; the subcutaneous tissue provided with these is called panniculus adiposus.

The fat lobules consist of fat cells, which, bound together by a common connective-tissue membrane, form masses or more lobulated clusters; the fat cells themselves are round or variously flattened, globular, homogeneous, and strongly refractive bodies. When heated with ether their fatty contents are extracted and the folded cell membrane, which sometimes contains a nucleus, remains. The moderate development of fat cells gives to the skin its tension and fulness, and to the body a desirable and beautifying plumpness and roundness. The excessive accumulation of fat, as in the cushions of fat on the buttocks and on the belly, forming the pendulous abdomen, betokens an excessive production of these fat cells. In wasting diseases and in starvation the fat cells are consumed—that is, burned—to maintain the warmth of the organism, and the skin becomes flaccid and wrinkled. Fat lobules are absent in the scrotum, penis, labia minora, eyelids, and pinna.

The coils of the sweat glands, wherever they exist, are found in the subcutaneous tissue (Fig. 1, *g*); on the hairy head the lower part of the hair sac also lies in this layer. Large branches of blood and lymph vessels and nerves are also present here; the first give off a fine plexus to the fat lobules and sweat coils and coarse ascending branches to the corium.

The *corium* (Fig. 1, *c* to *f*) is of firmer structure. It consists of a network of interlaced connective-tissue bundles that run parallel to the surface of the skin and are strengthened by anastomosis with obliquely ascending connective-tissue bundles from the subcutaneous connective tissue and by a rich network of elastic fibres. The anastomosis forms a close network, especially in the upper layer where it becomes very dense. The chief direction of these fasciculi and rhombic meshes formed by them is a definite one for most of the regions of the body, and accounts for the course of the blood vessels as well as for the arrangement and extension of certain skin diseases.

The fibres are here and there pushed aside by the hair follicles and sebaceous glands, by the vertically penetrating excretory ducts of the sweat glands, and the blood and lymph vessels and nerves which ascend in various directions. The fibrous tissue is disposed in thick bundles which directly surround and form the stroma of the hair follicles, the excretory ducts of the sweat glands, and the acini of the sebaceous glands. The most superficial fibres are pressed away from the general direction and are collected in a twisted manner to form the papillæ. Aside from the connective-tissue and elastic fibres which form the essential constituents of the corium, there are dispersed through it numerous simple and branched connective-tissue cells, as well as a varied quantity of lymph cells, which are the more numerous the younger the individual is.

The papillæ (Fig. 1, *c*, and Fig. 4) arise from the corium as prolongations of varied size, conical, club-shaped, or thread-like in form. They are single or are variously cleft into divisions having a common broad base, and consist of a variable amount of stroma, the inside being formed pre-eminently of elastic fibres. Some of them contain a loop of blood vessels, a distal artery, and a proximal vein, and are called vascular papillæ (Fig. 4, *a*); others contain a nerve corpuscle, a so-called Meissner's touch corpuscle or a Krause nerve-end bulb, and are called nerve or touch papillæ.

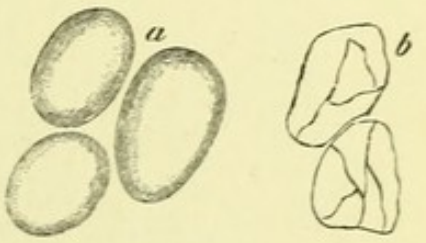

FIG. 2.—FAT CELLS.
a, cells with fat; *b*, cell membrane after extraction of its fatty contents.

The touch papillæ are found in the greatest number on the terminal phalanges of the fingers and toes, where, in alternation with the much more numerous vascular papillæ, they are arranged in regular curved rows; they exist to a considerable degree in other places also, as on the palm of the hand and the sole of the foot, the vermilion border of the lips and the nipples.

On the rest of the body the papillæ are situated more irregularly and further apart, the touch papillæ being less abundant than the vascular papillæ. We shall speak later concerning the blood vessels, lymph vessels, nerves, sebaceous glands, hair follicles, sweat glands, and muscles of the skin.

The *epidermis* (Fig. 1, *a*, *b*), in contrast to the other layers of the skin, completely lacks a fibrous structure and a vascular system. It is composed entirely of distinct cells, which are held together by a kind of cement substance. Julius Arnold considers this an albuminous product of lymph contained in the lymph channels which course between the epidermic cells and convey nutriment to them, and which are connected with the lymph canals of the papillæ. There are really two layers of the epidermis to be distinguished. The

deeper is the mucous or Malpighian layer ; it is remarkable for its horny appearance and dark coloring in contrast to the more clear, transparent, and lamellated superficial layer, the so-called horny layer or stratum corneum.

The Malpighian layer consists of distinctly nucleated cells, which are protoplasmic and consequently very quickly reproduced, arranged in parallel rows. These cells, and especially their nuclei, are easily stained by carmine and other coloring materials. This layer rests upon the corium, with the interposition of a structureless membrane

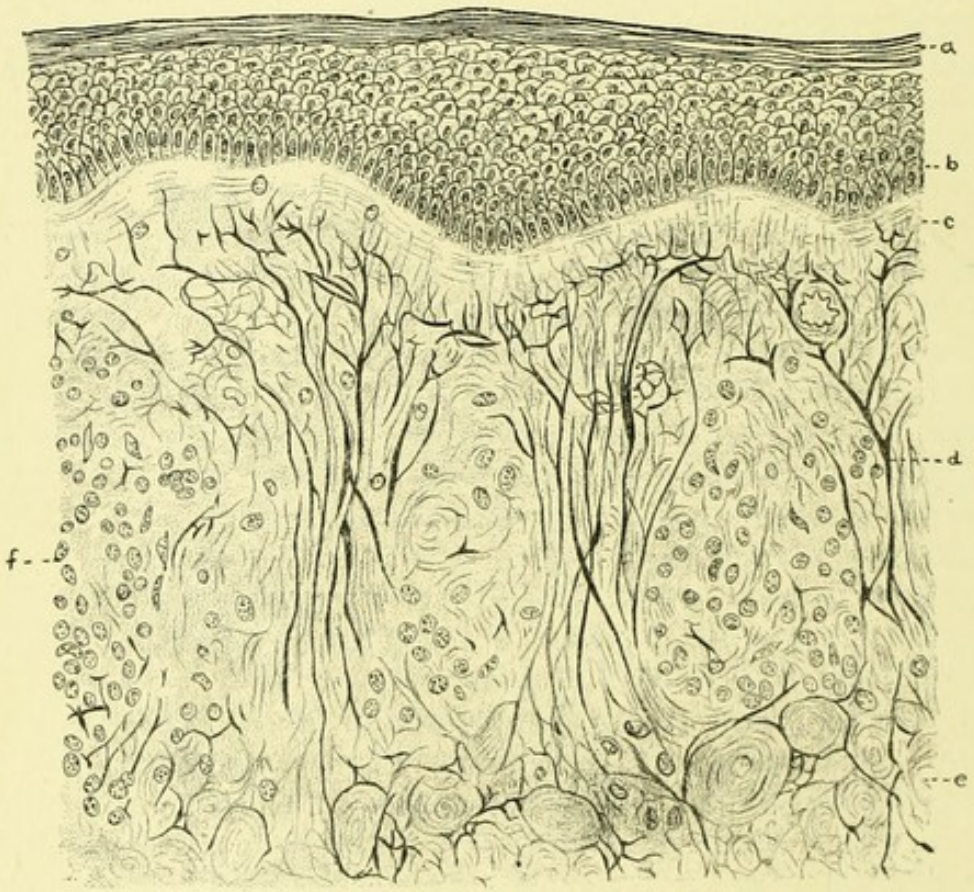

FIG. 3.—ELASTIC FIBROUS NETWORK, DEMONSTRATED WITH VICTORIA BLUE (LUSTGARTEN). *a*, epidermis ; *b*, rete Malpighii ; *c*, papillary layer ; *d*, elastic fibres ; *e*, fat lobules ; *f*, pathological cell infiltration of the corium.

which fits into the clefts between the papillæ and sends prolongations between the rete cells.

The cells of the deepest rete layer are granular, having their oval nucleus enclosed in a thin layer of protoplasm, and are arranged palisade-like with their long axis perpendicular to the surface of the corium, and terminate in a pointed extremity which penetrates the papillary tissue.

The next two or three layers nearer the surface have cells with an oblong nucleus. In men of light-skinned races these cells show but

little granular brown pigment, whereas in negroes much is found. The cells of the next higher layers are much larger, polyhedral, with round nucleus and a distinct cell membrane. They show numerous radiating filaments or prickles, which appear to interlock with those of the neighboring cells (Max Schultze's prickle or filament cells).

The significance of these filaments is not yet definitely determined. Schroen considers them to be the contours of lymph channels. From the most recent researches they are defined as protoplasmic processes which, of various shape and length, form a prickly sheathing to the body of the cell; proceeding from some cells they come into contact with the prickles of the neighboring cells (M. Schultze, Lott) or are merged with these (Ranvier, Unna). These cells gradually cease as the horny layer is reached. In the most superficial layers

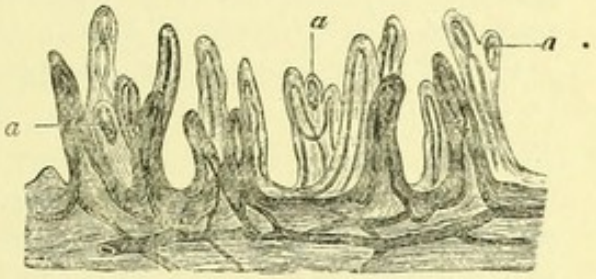

FIG. 4.—SKIN PAPILLÆ WITH THEIR EPIDERMIS REMOVED AND BLOOD VESSELS INJECTED.

a, touch papillæ containing a Meissner's corpuscle; the others, vascular papillæ.

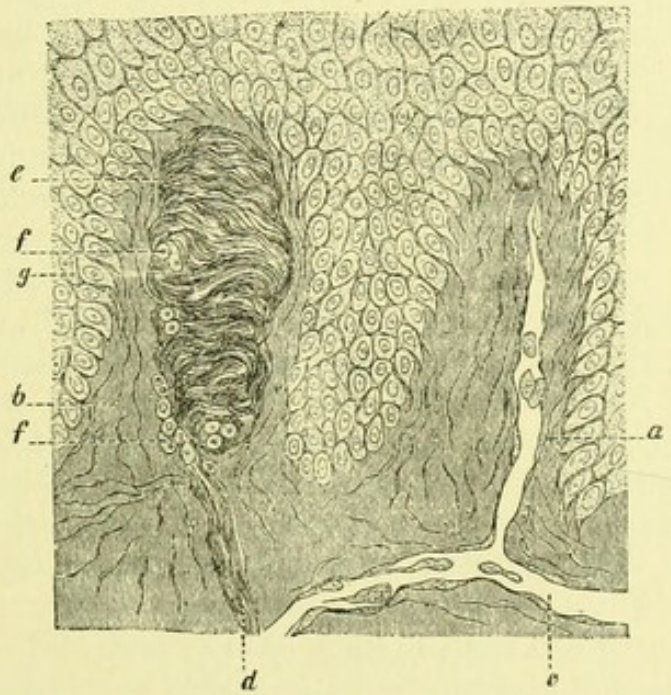

FIG. 5.

a, vascular papilla; *b*, nerve papilla; *c*, blood vessel; *d*, nerve fibre, which bears (*e*) a tactile corpuscle; *f*, transversely cut nerve fibre; *g*, cells of the mucous layer.

the cells become constantly more stiff and flattened, their nucleus becomes smaller, and they lie in layers more parallel to the surface. According to Auffhammer's and Langerhans' fine histological researches, the upper rows of the Malpighian cells are to be distin-

guished as granular cell layers from the granular appearance of their protoplasm.

Ranvier regarded these granules as drops of a fluid, oily substance and called it "eleidin"; while Waldeyer, upon the ground of their special chemical behavior, considered them to be related to the "hyalin" of Von Recklinghausen and applied the characteristic name of "keratohyalin" to the substance concerned in the cornification of the epidermis. Since his time many authors (among them Pavloff, Pollitzer, Zander, Unna, Blaschko, Lazansky, Mertsching, Winkler, and Herr von Schrötter, the last of whom considers eleidin and keratohyalin to be chitin) have endeavored through histological and chemical researches to throw light upon the relation of the so-called granules to one another and to the production of the horny cells of the epidermis. As yet, however, the process of cornification has not been clearly explained. The granules disappear again in the subbasal and not yet cornified cell layers; and, according to Unna's experiments, in the cells of the real horny layer the transformation into keratin affects only the outermost layer, while the central part still appears as an active substance which can be dissolved and eliminated. Liebreich therefore considered it probable that the granules in the cells of the stratum granulosum are not real albuminous bodies, but a mixture of albumin and cholesterin, which this inquirer has proven to exist as a regular ingredient of the keratin cells from many sources, as sheep's wool, birds' feathers and beaks, horses' hoofs, and human hair, and which he has introduced into therapy as lanolin. The explanation of Kromeyer, made upon the basis of his researches upon psoriasis, seems to me very acceptable. This author believes that cornification is quite independent of keratohyalin. He regards it as a quite gradual and uniform process, extending through the whole thickness of the epidermis to the beginning of the regular horny layer; a process which consists in a thickening and hardening of the cell membrane, so that the horny cell itself in its innermost part still contains a portion of its original protoplasm. Biesiadecki and Pagenstecher have seen existing between the rete-cells cells which bear an epithelial character, also some branched formative elements of the character of the so-called migratory cells. I also have verified their presence.

FIG. 6.—PRICKLE CELLS WITH NUCLEUS AND NUCLEOLUS.

According to K. Herxheimer there exist in the rete some very minute filaments which ascend and form a network with one another. Ehrmann considers them outlying projections from the pigmented migratory cells of the corium; Eddowes, fibrin coagulum; Kromeyer, epidermal fibres.

The horny layer of the skin—stratum corneum, or cuticula—appears upon section to be composed of layers of wavy fibres running parallel to the surface of the skin. On a closer examination this is found to be simply the result of the impression afforded by layers of flat cells superimposed one upon another. The nearer the Malpighian layer the clearer the cell character, although the cells are flatter and drier than the rete cells and seldom show a nucleus; the nearer the surface the more the cells appear as flat scales—corneous or epidermic scales.

The cells of the horny layer show only the very slightest amount of granular protoplasm, and that only in the deepest layers—basal and superbasal horny layers of Unna. They have therefore on the whole little, and in the most superficial layers scarcely any, evidence of life, and are but slightly colored by carmine.

The corneous scales, as you know, continually desquamate and are replaced by the pushing forward of the deeper layers. This supports the theory that the rete cells, by gradual advance from the deeper parts, become corneous scales. This supposition has not received the support of some authors, since they point to the fact that optically the transition from the rete cells to the horny cells is not a gradual one. There is a bright layer, the stratum lucidum of Oehl, between the mucous layer and the stratum corneum; some consider this to be only the result of a chemico-biological change which, as referred to above, the rete cells must undergo in order to become corneous plates. Schroen has taken a peculiar view of this; he thinks the stratum lucidum is a layer of flattened rete cells freed from the mucous layer, and that the stratum corneum which lies upon this cannot be a derivative of the rete cells, but a mere extension over the mucous layer of the cells which line the free opening of the sweat glands. The untenability of this view has already been proven by many authors.

The epidermis, as a whole, varies in thickness in different parts of the body. For example, it is thickest on the palms of the hands and soles of the feet, and under pathological conditions may become enormously developed on different parts of the body. On the other hand, under normal conditions it is very thin on the vermilion border of the lip, and as a rule is thinner on the flexor side than on the extensor side of the body. As a covering for the corium it adapts itself to the undulations caused by the papillary elevations, as on the vola of the finger; it fills in the interpapillary space and therefore appears quite even on the surface.

As a whole the epidermis dips downward into the hair follicle to a certain depth, the rete cells continuing to the base of the latter, clothing its inner wall and forming the hair root-sheath. Certain relations also exist between the epidermis and the cells covering the

sebaceous and sweat glands; these explain certain pathological conditions which will be mentioned later.

Growth and function, the two attributes peculiar to the living organ, are both possible to the skin through its vascular and nervous system; also the alteration of both these characteristics—that is, disease. It is therefore necessary to fix in one's mind the facts with regard to circulation and innervation.

As already described, only the corium and subcutaneous connective tissue possess *blood vessels*. They are arranged in two layers parallel to the surface, a deeper one lying in the subcutaneous connective tissue, and one, more superficial, which extends beneath the papillæ. Their relations are shown in the illustration (Fig. 7, chromo-lithographic plate), which represents a section from an injected piece of skin (Tomsa).

In the subcutaneous connective tissue there are coarse arterial branches running horizontally. They give off small circular branches and capillaries to the fat lobules and sweat glands, and, according to Spalteholz, by means of a free anastomosis form a characteristic cutaneous network in the lowest layer of the cutis, close to the layer of fat. The larger branches ascend—some perpendicularly, following the excretory ducts of the sweat glands, some obliquely—and penetrate the corium. From these vessels branches are given off to the papillæ of the hair sacs and the sebaceous glands, as well as to the connective-tissue and muscular bundles. The chief branches pass into the upper layers of the corium, and, after anastomosing, form a vascular network running parallel to the surface directly beneath the papillæ (stratum vasculosum or subpapillare); from this again terminal vessels ascend to the papillæ, where they become capillaries.

The venous distribution is arranged on an analogous plan, but of course in a reverse order to the arterial, with which it topographically coincides. The veins take their origin from the papillary capillaries and form their first great network in the subpapillary layer. From this the venous blood is collected by several greater trunks which, running parallel to the excretory ducts of the sweat glands or following the direction of the larger connective-tissue bundles, pass into the layer of subcutaneous connective-tissue, receiving on their way the venous branches which take their origin in the vascular network surrounding the hair follicles and the sebaceous glands. In the subcutaneous tissue the veins receive the branches coming from the sweat glands and fat lobules, and add to the vascular stratum running parallel to the surface a great venous trunk situated similarly to the arterial.

We have, then, as striking characteristics of the vascular system of the skin, a superficial or subpapillary, and a deep-lying or sub-

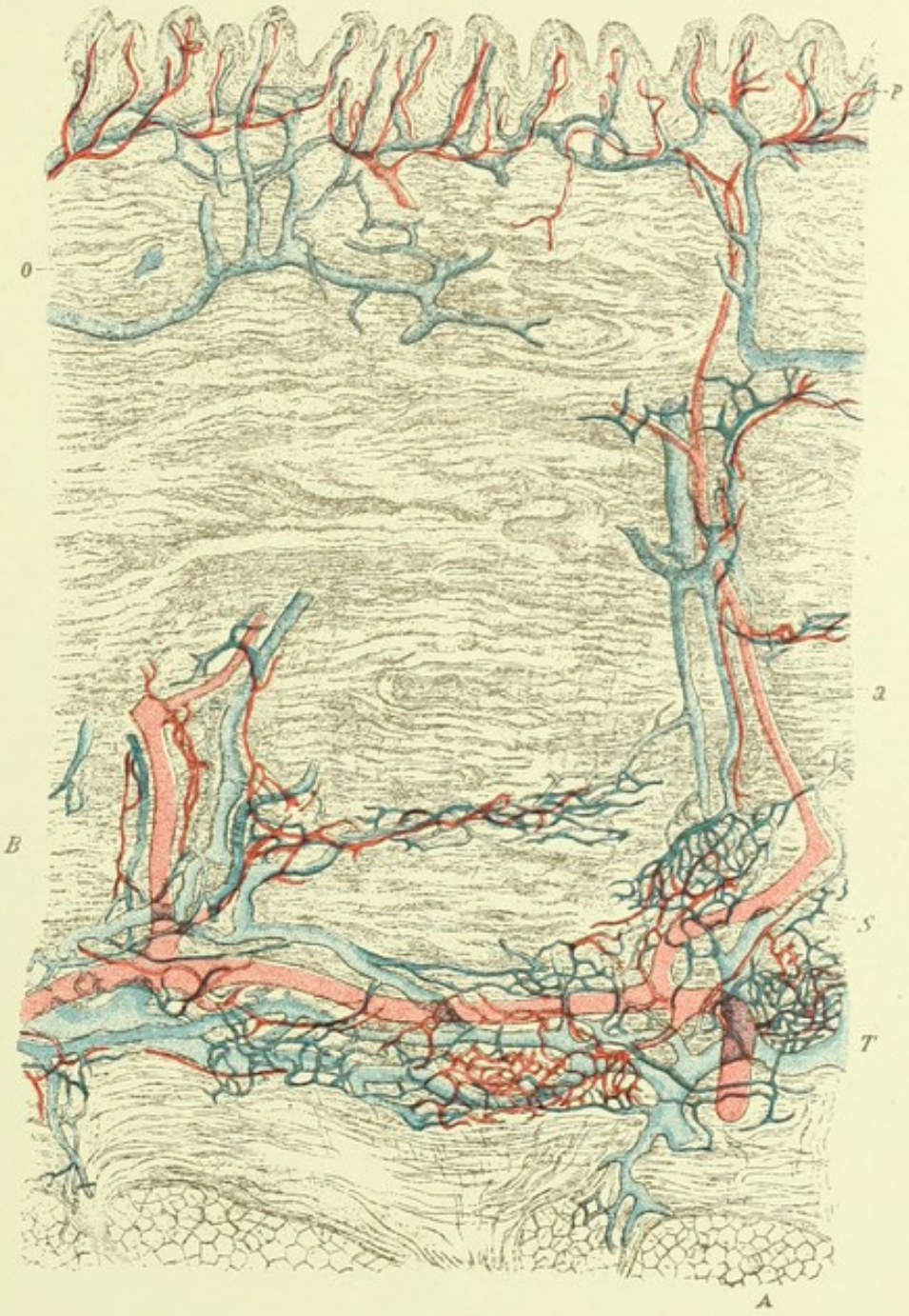

FIG. 7.—VERTICAL SECTION THROUGH AN INJECTED PORTION OF SKIN FROM THE PALM OF THE HAND (AFTER TOMSA).

The arteries are injected red, the veins blue. T, deep plexus of vessels corresponding to the layer of fat lobules and sweat glands; O, superficial (subpapillary) vascular plexus; at a, vessels accompanying the efferent duct of the sweat gland; P, loops of papillary vessels; S, plexuses surrounding the coil glands; B, ascending branches; A, layer of fat cells.

cutaneous, arterial and venous vascular stratum, both running parallel to the surface and communicating with each other through afferent and efferent, ascending and descending branches ; there are also vascular anastomoses in connection with the glandular organs of the skin ; and, finally, over the whole surface, the extensive capillary network of the papillæ.

The latter (Fig. 4), protected from the atmosphere by only a thin connective-tissue layer and the epidermal covering, afford a most complete analogy to the capillary network of the pulmonary vesicles. They maintain cutaneous respiration through the interchange of gases with the atmospheric air and the exhalation of moisture.

Moreover, since the vascular system alone furnishes nutriment to the skin, it also furnishes the material for the creation of its specific products, the secretion of the sweat and sebaceous glands. By its topographical relations it influences the locality of inflammatory processes and new growths and the direction in which they develop, so that, for example, these processes are particularly localized and extensive near the glands which possess the most pronounced vascular network, or along the horizontal vascular trunks beneath the papillæ. It is also easily understood that in the papillary and uppermost layers of the skin neoplasms and inflammatory growths are for a long time almost isolated, since their vascular system is to a certain degree independent of the deep-lying vascular layers. Moreover, the system of vessels distributed to the papillæ appears to be divided into several and to a certain extent independent territories ; at least one sees, as has been demonstrated many times, and especially by Renaut in regard to this subject, that, when the skin is artificially injected, distinct areas of injection are formed at first, the borders of which merge into the neighboring ones only by progressive filling of the papillary network from all sides. Every such area of injection on the surface of the skin forms the base of a wedge-shaped field of injection whose apex is the arterial trunk belonging to this area.

The anatomical explanation of the form and mode of extension of certain superficial diseases of the skin—hyperæmic spots, roseola, and exudation itself—will be found in these relations ; and the fact that these changes often affect only isolated and limited areas is due to the circumstance that a variation in the circulation in the area of injection may be occasioned by simple contraction or dilatation of a single afferent arteriole. Thus the vascular division of the skin affords much that is instructive in the study of the pathology of this organ, and its further investigation offers many opportunities to determine these conditions more thoroughly.

The *lymphatic system* of the skin is the necessary appendage of the vascular circulation which nourishes it. It takes its origin, as Teichmann's investigations first showed, from the papillæ, in a

manner not yet determined. It is probable, however, that the lymph channels originate chiefly in open, some perhaps in closed (Neumann), lymph spaces provided with stomata. These spaces gradually empty into a closed system of vessels. This forms a network which is superficial, being seated beneath the subpapillary vascular stratum.

A network of larger branches is found in the subcutaneous tissue; it is connected with the former through anastomosing branches. Besides these there are interfascicular spaces in the corium and papillæ, differing in size according to circumstances, and containing a varying amount of lymphoid cells or a more serous fluid. These, like the connective-tissue spaces containing the blood vessels, serve as lymph spaces, whose relation to the closed lymphatic system is not yet anatomically established.

Contrasted with the corium richly supplied with blood vessels and glands, the epidermis plays an apparently independent rôle in its developmental relations, since, with perhaps the exception of intercellular lymph spaces, it is quite lacking in vessels. Yet the development of the epidermis is very active. It is known that its uppermost layers are constantly thrown off and replaced by cells pushing forward from beneath; that the active and reproductive material for the epidermis can only come from the capillaries of the papillæ. Experimentation and clinical experience in healing of wounds have taught that in parts where the papillæ are destroyed the skin forms again only in a slight measure and has the character of the horny layers. The same holds true of the pigment, the formation of which is connected with the development of the epidermis; its probable source will be more particularly spoken of later. The source of the actual new formation and constant replacement of the epidermis is not yet decidedly determined. In pathological cases a new formation of epidermis results from the division of the old cells and their nuclei, which, according to the latest researches of Flemming, Strassburger, Rabl, etc., is accomplished by means of karyokinesis and will be minutely described later. This is undisputed. It may also take place like the physiological regeneration of the epidermis, and the basal prickle cells may therefore play the chief rôle. On the other hand, the processes taking place in the healing of wounds and in cicatrization uphold the view that new cells are formed from those on the edge, as in the case of the corneal epithelium experimented upon by Stricker. That the migratory cells proceeding from the corium could become epidermic cells, or, at least, that this could be the rule, seems improbable, since such migratory cells are seen under none but pathological conditions. The developmental independence of the epidermis, above described,

is of the greatest importance in determining the conditions presented by many dermato-pathological states.

The *nerves* of the skin have both medullated and non-medullated fibres. In the subcutaneous connective tissue and lower portion of the corium some fibres branch off from the nerve trunks and terminate in the Pacinian or Vater's corpuscles, or pass to the glands and capillaries. The main trunk of nerve fibres ascends through the corium to its upper layer and forms a subpapillary network accompanying the similarly situated vascular plexus. From this fibres arise to terminate in Meissner's corpuscles or in the end bulbs of Krause in the touch papillæ.

The capillary loops of the vascular papillæ have their special nerve plexus. According to Tomsa there is a network of nucleated nerve fibres in the periphery of the vascular papilla; from this fibrillæ pass toward the centre of the papilla and terminate on the capillary wall in bulbous extremities. Although the ultimate organic union between nerve endings and the capillaries is not yet definitely made out, yet the intimate connection that has been demonstrated to exist is of great importance, since it proves that the capillary vessels of the skin papillæ are under direct nervous influence. This connection is instructive as explaining how vascular contraction and dilatation, and even exudation, may, as in the case of urticaria, take place by direct irritation and yet be limited to individual papillæ.

Since Langerhans' investigations in 1868, it is decided that non-medullated fibres from the papillary stratum pass into the mucous layer of the epidermis and form plexuses between the rete cells, and then end at different depths in bulbous swellings or terminate in some unknown manner (Eberth, Biesiadecki, and others). According to Ranvier's view the nerves entering the epidermis are subject to a constant growth, while their ends undergo a gradual degeneration, being transformed into granules of nerve substance, which finally become quite free and penetrate the inactive layers of the epidermis. Ranvier denies the existence of Langerhans' interepidermal nerve plexus. Unna, by means of osmium preparations, believes that he has shown the relations of the interepithelial divisions of the nerves—namely, that two non-medullated fibres are given off from the terminal branch and enter two neighboring cells; that each cell is provided with a pair of terminal branches which proceed from two different nerve trunks and apply themselves to the cell nucleus in a manner similar to that which Pfitzner has already demonstrated. W. Wolff, however, has shown that Unna's views and his deductions drawn from the above-mentioned osmium experiment are not tenable. He denies utterly the existence of non-medullated fibres in the epithelium.

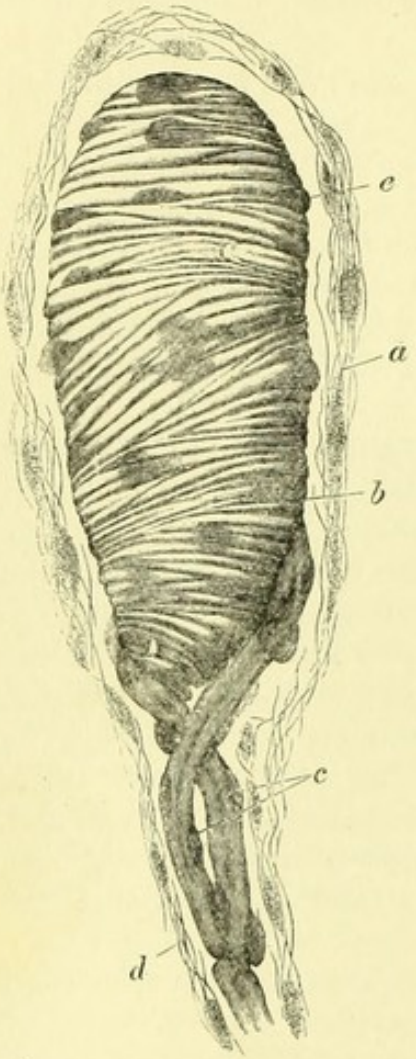

FIG. 8, I.—HUMAN TOUCH CORPUSCLES ACCORDING TO WOLFF.

a, pericapsular connective tissue; *b*, folded membrane; *c*, neurilemma with its nuclei; *d*, perineurium; *e*, nuclei of layers *a* and *b*.

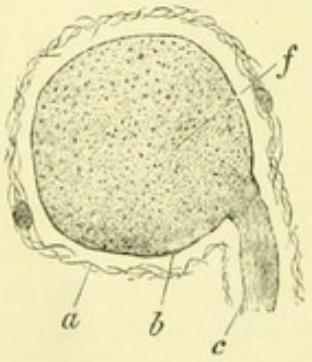

FIG. 8, II.—SCHEMATIC HORIZONTAL SECTION OF A TACTILE CORPUSCLE, MADE THROUGH THE ENDING OF A NERVE.

f, finely granular contents of a touch corpuscle.

Doubtless the end organs of the nerves of the skin are the already mentioned Meissner's or Wagner's corpuscles and Krause's end bulbs which occupy the tactile corpuscles, and the corpuscles of Pacini or Vater which are situated here and there in the corium.

The *corpuscles of Meissner* or *Wagner* (Fig. 8, I and II), which are oval bodies of 0.02–0.045 millimetre in diameter, occupy the greater part of the papilla in which they lie. On their outer surface are to be seen fine and broad transverse striations and bands, as well as oval nuclei; from researches made at different times the striations have been variously regarded as connective tissue, elastic tissue, or nerve fibres. The medullated nerve fibre coming from the corium approaches the corpuscle, sometimes at its base, sometimes midway or at its apex, and, after winding about the corpuscle and losing its medullary sheath, it terminates, according to Biesiadecki in four to six terminal fibres, according to Brücke in more, on the inner side of the tactile corpuscle. According to Thin the tactile corpuscles are simple, but many are separated by connective tissue and elastic transverse sheaths (continuations of the investing capsule) into two or three lobules superimposed one upon the other, each of which contains a nerve corpuscle; in or on these the nerve fibre now ends, after the medullated nerve as such has pierced the outer covering of the capsule.

Tomsa has demonstrated that the encapsulated tactile corpuscles are lamellated in structure. M. Kraus has shown that they consist of flat cells lying upon one another in layers and somewhat compressed by mutual contact; he has not been able to demonstrate anything peculiar in the union of the end of the

nerve with the cells, while Robinson thinks that the nerve of the tactile corpuscle pierces it spirally and then again enters the prickle layer, where it grows smaller by subdivision and ends in the lowest cell rows.

W. Krause, the discoverer of the nerve end-bulbs named after him, has recently demonstrated that essentially the same scheme of ending holds good for all nerves terminating in nodules. These (analogous to the touch discs of Ranvier) are flattened and form terminal nerve corpuscles, disposed partly in concentric lamellæ arranged around a central flat cell, partly in various other ways.

The latest researches of Wolff seem to have demonstrated decisively and clearly the real anatomical relations of the tactile corpuscles, which have been so variously conceived of and so differently explained. The outer cover, consisting of loose connective tissue, the pericapsular connective tissue, is a prolongation of the perineurium. It encloses the special capsule of the tactile corpuscle, which is described as a folded membrane, and in turn is the continuation of the neurilemma of its accompanying nerve branch; it also encloses an amorphous soft mass containing fine granules but no nerve substance. The axis cylinder, which loses a portion of its sheath as it enters the corpuscle, comes to an end soon afterward, so that this end organ seems to possess no peculiar, specific sensitiveness, but serves as a tactile instrument for the sensitive nerve, as, owing to the homogeneous contents of the capsule, it lessens the intensity of pressure and diffuses it equally.

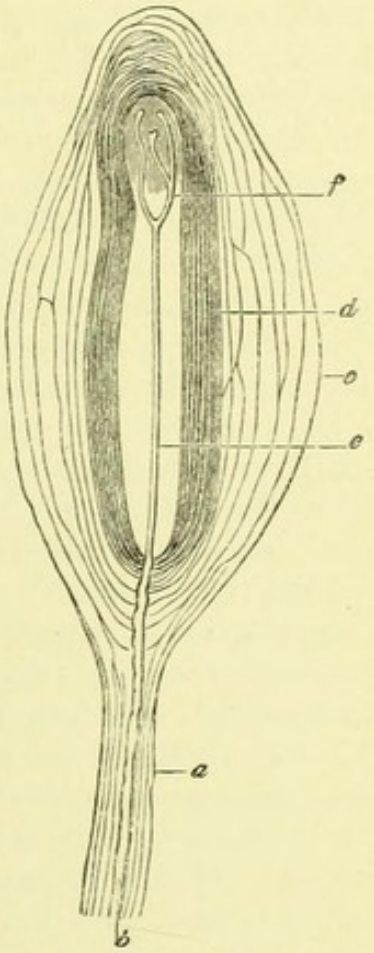

FIG. 9.—A VATER'S OR PACINIAN CORPUSCLE.

a, pedicle of corpuscle; *b*, nerve fibre entering it; *c*, *d*, external and internal surfaces of the envelope; *e*, axis cylinder with knob-like extremity (*f*).

The touch corpuscles are most numerous and regular on the terminal phalanx of the finger, less so on the hands and feet, the nipples and the lips. On the latter and on some other portions of the skin, as the glans penis and clitoris, the end bulbs of Krause exist, which are similar to the more simple Meissner's corpuscle described by Thin.

The terminal nerve organs, called now *Pacinian corpuscles*, first referred to by Langer and described later by Vater and known as *Vater's corpuscles*, are most numerous and typical in the mesen-

tery of the cat. They exist also, according to Genersich's researches, on the abdominal sympathetic plexus, where they are large; according to Geber, on the tongue; according to Rauber, in the fibrous covering of the spermatic cord and in the connective tissue of the mediastinum and tendons; according to Klein, in the corpus cavernosum of the penis. In the human skin they are most numerous in the subcutaneous tissue on the volar side of the hand and the plantar surface of the foot, and are so deeply placed as to be unfavorably situated for appreciating tactile sensations and so can scarcely be called organs of touch. They are oval bodies, 1.12 to 4.5 millimetres long, consisting of a number of concentrically lamellated connective-tissue capsules surrounding a cavity filled with serum. Axel Key, G. Retzius, and Genersich consider that the capsular covering is a thick membrane, which is clothed on its inner and outer sides with a nucleated tunic and contains serum in its centre, lodged in its interstitial connective-tissue spaces. A medullated fibre pierces the capsule wall, losing as it advances its medullary sheath, and under the form of a simple axis cylinder enters the cavity, in the upper part of which it ends, either without dividing or in two or three swollen extremities.

The nerve fibres ending in the skin, classified according to their physiological significance, are, first, *sensory*, acting as transmitters of the so-called cutaneous sensations, taking the latter in their widest sense; second, *motor*, some of which are distributed to the muscles of the skin and the arrectores pilorum, while others—the vaso-motor nerves—innervate the blood vessels and are further subdivided into vaso-constrictors and vaso-dilators. The latter class of nerves, which govern the local circulatory changes, are associated on the one hand with the nourishment and on the other hand with the secretory function of the skin; they are therefore called *trophic* nerves. It will be readily understood how some limited capillary circuits, under the influence of their governing vaso-motor nerves, at one time become dilated, at another contracted, so that we find overdistended blood vessels and congestion, or the reversed conditions, forming the forerunners of disease. Even this elementary explanation may suffice to show you the great significance of the vaso-motor nerves in the pathology of some diseases of the skin. Whether the nerves of the skin can be anatomically separated into secretory and trophic is a much-discussed but as yet undecided question. We shall consider the functional office of the nerves of the skin elsewhere.

Besides the transversely striated muscular bundles which in the region of the face pass from the deeper structure into the skin, other *muscles* enter into the structure of the latter. These muscles peculiar to the skin are non-striated or smooth. In addition to the

organic fibres which belong to the walls of the great vascular and lymph systems, and to the excretory ducts of the glands, muscles are found in the skin itself, as simple, or branched and anastomosing, fibres running parallel to the surface. They are very unequally distributed in the subcutaneous connective tissue and in the corium in different parts of the body. They are well developed on the scrotum, in the tunica dartis, on the prepuce and the perineum, and they occur as circular bundles in the areola of the nipple and in the skin of the nipple. In the uppermost layers of the corium, according to Neumann, they are also present to an extent varying in different parts of the body, being as a rule more prominent on the extensor surfaces. The muscles known as the arrectores pilorum have a characteristic direction ; they are attached by one or more root bundles to the papilla, and pass, either in one bundle or in two or more, obliquely downward past the base of the sebaceous gland to the hair sac, and are inserted into the inner sheath of the latter. Often the muscle sends a secondary bundle to the sebaceous gland. The contraction of the muscular bundle straightens hair sac and hair, which are normally oblique. The muscular bundles, which arise from opposite sides and form a loop surrounding the hair sac, serve by their contraction and approximation to raise the base of the hair follicle, as in the so-called goose flesh. Where hairs are coarse and congregated, as on the scalp, the muscular bundles of the arrectores pilorum form a network with one another, forming an extensive subpapillary muscular plexus. According to Unna's view the elastic tissue at the end of the oblique tensors of the corium is the real factor in producing movement, although by their mutual contraction both have an influence in regulating the tension of the skin and those conditions of internal pressure which affect the secretions, the circulation of the blood, and the transmission of lymph.

LECTURE III.

ANATOMY OF THE SKIN (CONTINUED)—SWEAT GLANDS, SEBACEOUS GLANDS, HAIR, NAILS—PHYSIOLOGY OF THE SKIN—ITS THREEFOLD FUNCTION AS A PROTECTIVE, HEAT REGULATING AND SECRETORY, AND SPECIFIC SENSE ORGAN.

THE sweat glands (glandulæ sudoriferæ), or convoluted glands, are situated in the subcutaneous connective tissue. They are simple, uniformly wide glands, coiled upon themselves and terminating in a cul-de-sac. An excretory duct ascends through the corium in a straight, and through the epidermis in a wavy, line, to open in a funnel-shaped orifice on its surface. The horny layer and the rete Malpighii enter this opening in the form of a hollow cone, which constitutes the wall of the wedge-shaped orifice.

From the border of the papilla outward the lining of the tube is formed of a simple layer of conical nucleated epithelial cells which leave free a narrow lumen; this is to be seen upon a transverse section. Outside of the cellular layer lies the membrane proper of the duct; it consists of a bright basement membrane with thick connective-tissue fibres lying outside of it. In the large glands of the axilla there are also non-striated muscular fibres running longitudinally. The arterioles supplying the convoluted gland arise from the deep-lying vessels, and, surrounding the coil, form, before they become capillaries and pass into veins, a plexus bearing a very remarkable resemblance to the plexus of the Malpighian corpuscles of the kidneys. A further analogy between the structure of the two consists, according to Ranvier, in the character of the epithelium of the gland coil, whose cells, like the epithelial cells of the urinary tubules, show a granular constitution, contain true fat globules, and enclose a system of fine canals which extend on one side to the membrana propria and open on the other into the gland lumen.

The great mass of sweat glands is found on the palm of the hand and the sole of the foot (2,685 to 2,736 upon a square inch, according to Krause). On portions of skin richly supplied with papillæ they open in the interpapillary furrows, on the ball of the finger at regularly disposed intervals, upon the palm of the hand and sole of the foot in longitudinal rows. They are not found on the border of the lip, the glans penis, or the foreskin.

The *hair* (pili), hair follicles, and sebaceous glands form an anatomically connected structure and will therefore be best considered together. The accompanying illustration (Fig. 11), taken from the work of Biesiadecki, represents a section of a hair of the beard and gives a good idea of the relations of these structures. A wedge-shaped depression is seen between two papillæ, which continues into the layer of fat cells, and at its blind end is seated upon a papilla which varies in size according to the depth from the surface. The

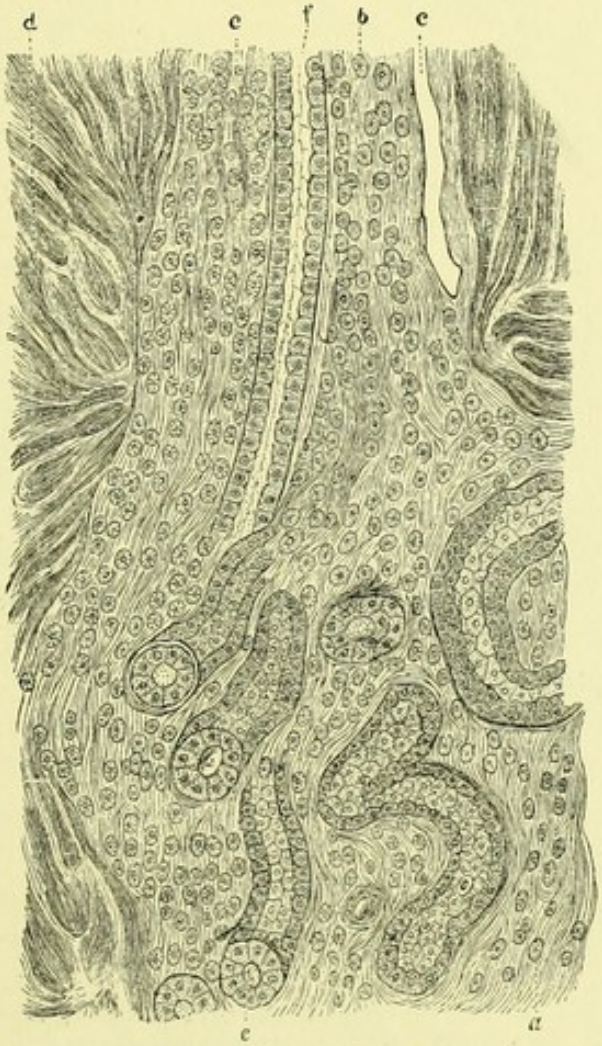

FIG. 10.—SWEAT GLANDS.

e, transverse section of a gland tube, longitudinal and transverse section, in vicinity of gland coil; *f*, lumen of excretory duct; *c*, *c*, blood vessels supplying gland; *d*, connective-tissue bundles; *a*, *b*, pathologically infiltrated cells.

pouch-like opening is the hair sac, the papilla at its base is the hair papilla; upon the latter is placed the hair, the shaft of which projects through the follicle to the mouth. At the side of the hair follicle lies the sebaceous gland with its acinus, which opens through its excretory duct into the hair sac. A muscular bundle, the musculus arrector pili, runs, in a direction oblique to the surface of the skin, past the base of the sebaceous gland and thence to the base of

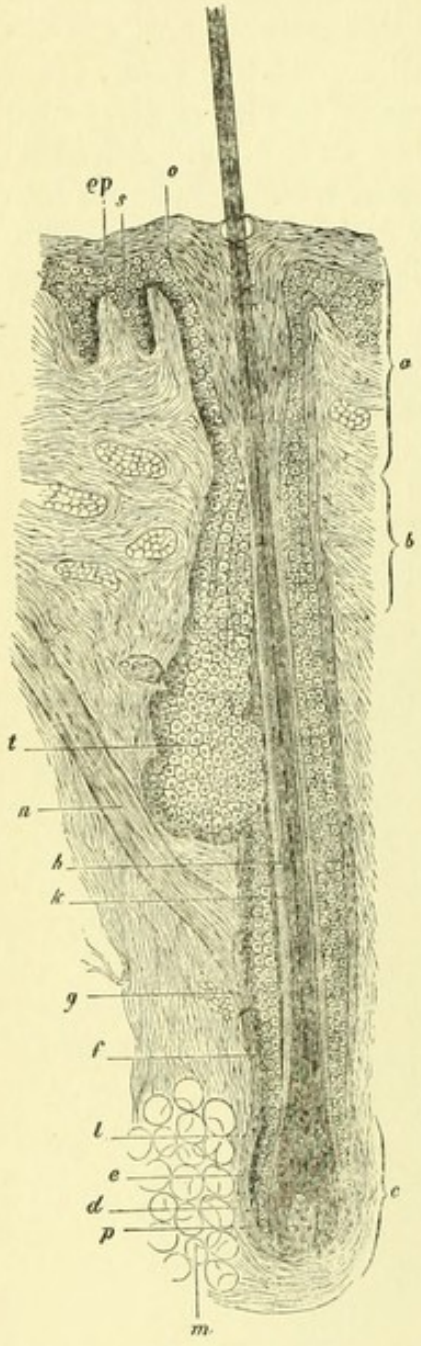

FIG. 11.—SECTION THROUGH A HAIR OF THE BEARD.

a, external dilated portion of the hair pouch; *b*, neck of same; *c*, dilated lower portion; *d*, external sheath of hair sac; *e*, internal sheath; *e*, *p*, epidermis; *f*, external sheath of hair root; *g*, internal sheath of hair root; *h*, cortical substance of hair shaft; *k*, medullary substance of hair shaft; *l*, hair bulb; *m*, fat cells; *n*, arrector pili muscle; *o*, papillæ; *p*, hair papillæ; *s*, mucous layer; *t*, sebaceous gland.

the hair follicle. This general arrangement, as well as the special and minute anatomical relations, holds true only for the thick and long hairs of the body.

The *hair follicle* shows an unequal lumen; its orifice (*a*) is funnel-shaped. At the small end of the funnel the sebaceous gland (*t*) opens; this is also the narrowest portion of the hair sac—the neck (*b*). From there downward it widens somewhat, especially toward the base where it forms the hair sac (*c*), into which the papilla (*p*) projects. The hair follicle proper includes the part below the opening of the sebaceous glands. It consists anatomically of three layers, the outer, known also as the outer sheath of the hair sac (*d*), the external fibrous coat of Kölliker, formed of connective-tissue fibres which, rising from the upper layers of the corium in compressed bundles, run parallel to the axis of the hair sac and surround its base. The fibres are thickest toward the under side; on the outer they either lose themselves in the surrounding connective tissue or form a sharply defined border. Between them lie the vessels and nerves of the hair sac. The second or middle layer of the hair sac is called the internal hair-sac sheath, or Kölliker's inner fibrous coat; it consists of transverse fibres, and betwee nthem a granular substance in which are embedded elongated nuclei resembling those of striated muscle cells. The third or innermost layer of the hair sac consists of a homogeneous, so-called vitreous membrane, which is better seen in cross-section. The hair papilla is formed from the stroma of the coats of the follicle, especially the middle one, being for the greater part also covered by the vitreous membrane. It is divided

into neck, body, and a wedge-shaped apex. In the papilla are non-medullated nerve fibres and a loop of blood vessels. The hair sacs do not lie vertical to the skin, but are implanted obliquely to its surface, as also are their hairs. This direction varies in different parts of the body and has been carefully studied by Voigt. The lines of direction of the hair differ according to the region of the body, running in peculiar lines and curves which in certain places form hair whorls. This arrangement depends, as has been definitely shown by Tomsa, upon the direction of the bundles of connective tissue in the corium.

The greatest interest, from a pathological standpoint, attaches to the anatomical contents of the hair sac, which consist of hair root sheaths, external and internal, and the hair. The outer root sheath of the hair is the most external of the structures in the hair sac, lying directly on the vitreous membrane of the hair follicle. It consists of rete cells which are continued directly from the papillary layer into the hair sac. Up to the point where the sebaceous gland empties the rete appears in all its layers, including the granular layer ; from there down the external root sheath proper retains only the deepest rows of cells and the prickle cells. The deeper it goes the fewer the rows become, until at the level of the bulb they are reduced to one. The internal root sheath (Fig. 11) is surrounded by the outer and in direct contact with it. In this two layers can be distinguished—the outer, the sheath of Henle ; the inner, the sheath of Huxley. Both layers of the inner root sheath consist of plates which are blended to form a lamellated, transparent covering for the hair, which stains slightly with carmine. Inside of all these structures lies the hair, protected by the sheath of Henle. This is divided into the cylindrical hair shaft, which in case of long hairs protrudes from the mouth of the sac, and the hair root or hair bulb, a nodular swelling by which the hair rests upon the papilla. Under the microscope there can be recognized on the outer side of the hair shaft an imbricated, spirally reticulated membrane, the cuticle (Fig. 11, *h*), in which two layers, an inner and outer, can be distinguished. Within this is situated the hair proper or cortical substance. It is composed of filaments running parallel to the long axis of the hair. These filaments owe their shape to the fact that the horny cells are pressed together and contain, besides numerous dark granules, much yellowish-brown pigment in the case of dark hair ; in gray hair this substance is destitute of pigment. In the centre of thick hairs is found a space which grows smaller and disappears toward the point of the hair. The root of the hair consists of cells similar to the rete cells, which they resemble in direction and configuration. Those which rest vertically upon the vitreous membrane of the papilla are cylindrical ; those of the higher layers are polyhedral. They are

loosely arranged, pressing tightly upon one another. In the upper half, where the hair bulb passes into the shaft, the cells of the bulb become oblong, spindle-shaped, and thicker, are arranged in long filaments, and pass imperceptibly into the cortical substance of the hair shaft. This holds true only for the cells of the outer layer of the hair bulbs. In the middle layer is found a zone of cells which are protoplasmic and stain with carmine. The other cells of the bulb contain and have situated between them much brown, even black, granular pigment. The mature hair grows by the formation from the papilla of new epidermic cells, which, as they are converted into the longitudinally placed horny cells of the hair substance that afterward constitute the hair shaft, push forward into the sheath of Huxley. We shall speak later of the special pathology of the hair and of the other important details in hair formation and regeneration, as well as concerning the pigment of the hair. Here I shall only remark that in view of the great diversity of opinion concerning the mutual relation of the inner and outer root sheaths, as well as that of each to the hair and to the external epidermis, we cannot consider that this important anatomical structure is as yet definitely understood. It will be of special interest to us in many processes—*e.g.*, lichen pilaris.

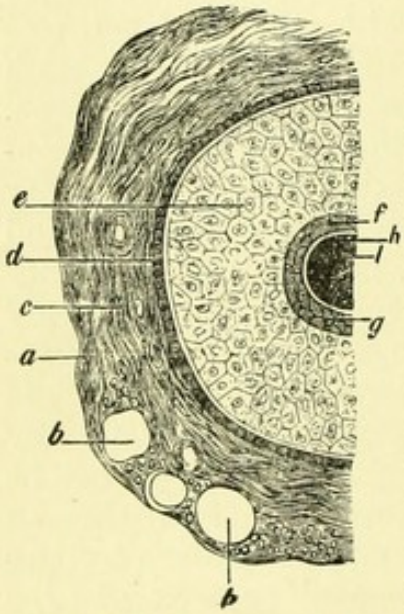

FIG. 12.—CROSS-SECTION OF A HAIR, BELOW THE NECK OF THE HAIR FOLLICLE.

a, external sheath of the hair follicle; *b*, cross-section of vessel; *c*, inner sheath of the follicle; *d*, vitreous layer of hair follicle; *e*, external root sheath; *f*, *g*, internal root sheath, comprising an external layer (Henle's layer, *f*) and an internal layer (Huxley's layer, *g*); *h*, cuticula; *l*, hair.

The above-described imbricated layers of the hair sac and its contents are represented in Fig. 11 in longitudinal section, in Fig. 12 in cross-section. I shall only briefly remark that most investigators agree that the corneous layer is not continued along with the rete cells into the hair sac. The horny layers extend only to the neck of the hair sac, filling in its mouth like an epidermic wedge. The rete cells form the outer root sheath as far as the fundus. Many—for example, Henle and Biesiadecki—now think that the rete cells, which as the external root sheath cover the hair sac, form horny cells on their internal aspect, and that these constitute the outer layers of the internal root sheath (that is, Henle's layer), but that the inner layers of the inner root sheath (that is, the sheath of Huxley) are derived from the original rete of the hair at the same time that the cuticle and hair are formed from the epidermal covering of the papillæ. Unna, however, very

convincingly demonstrates that those layers of cells of the rete distinguished as granular cells, which on the surface change into horny scales, do not reach beyond the neck of the sac; the prickle layer alone does so, and even this for the outer root sheath only; that this produces no horny cells and no Henle's sheath; that, on the contrary, Henle's and Huxley's sheath, cuticle, and hair are a unit and produced at the same time from the papilla as one original hair germ. The inner root sheath, the united sheaths of Henle and Huxley, proceed in their growth as far as the epidermal cone filling the mouth of the hair follicle, and there cease. The growing hair, with its cuticle now spirally pushed forward, breaks through Huxley's and Henle's sheath, pierces the epidermis lying in the mouth of the follicle, and appears on the surface. I am inclined to favor Unna's view; certain pathological appearances are made more comprehensible by it. In a hair sac is found, as a rule, one hair only, but sometimes there are two. The latter occurrence depends upon physiological changes in the hair, and will be discussed in connection with diseases of the hair.

The *sebaceous glands* are appendages to the hair follicles, as seen in Fig. 1 (*n*), but only in case of long, thick hairs. With lanugo hairs the relation is reversed, as shown in Fig. 13.

The sebaceous glands are acinous glands in which a gland proper and excretory duct are to be distinguished.

The former consists of round lobules, or acini, which are collected into clusters, these being united in a multilobulated gland. The wall of the gland lobule consists internally of a transparent membrane, and outwardly of a thick structure of connective and elastic tissue furnished with a rich vascular network. The inner part of the lobule itself is formed of epithelial cells; its outer layer, next to the transparent membrane, consists of clearly nucleated, cylindrical or cubical cells similar to those of the rete; nearer the centre of the gland the cells become larger, polyhedral, more like the corneous cells, and contain drops of fat which cover the nucleus. The cavities of the lobules empty into the large common gland ducts in which lie epidermal débris,

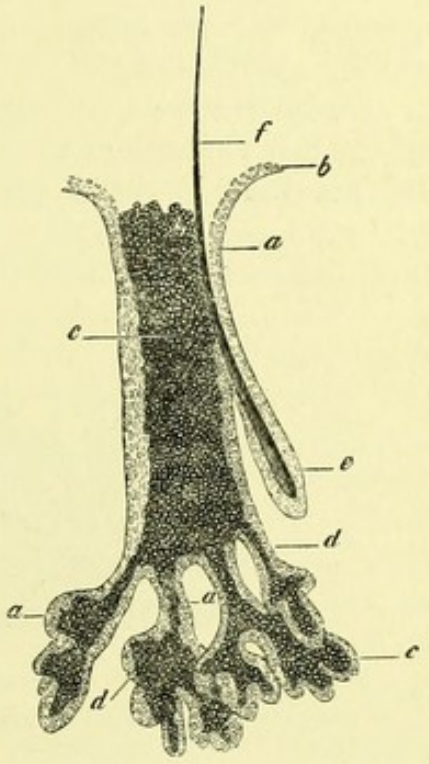

FIG. 13.—SEBACEOUS GLAND WITH A LANUGO HAIR.

a, gland epithelium; *b*, rete Malpighii, continued into the gland epithelium; *c*, gland contents, composed of free fat and cells containing fat; *d*, acini; *e*, root sheath with hair.

free fat, and fatty crystals. A common excretory duct, or two such, lined with epithelial cells and carrying fat, fat cells and their débris, open into the hair sac. The glands of the lanugo hairs open directly on the surface, forming large pores in the skin, which are often recognizable to the naked eye and bear one or more little hairs, or sometimes none. There are no sebaceous glands on the palm of the hand, the sole of the foot, the glans penis, and the dorsal surface of the third phalanx.

The *nails*—ungues—are long, four-sided, flat or shield-shaped bodies, curved from side to side, with the convexity above; they are elastic, though brittle in the anterior segment, resistant, and transparent, and are composed of an aggregation of horny epidermic cells.

On three sides they are embedded in a fold of skin upon the back of the last phalanx of the finger, and cover with their under concave surface the anterior portion of the latter, their anterior border pro-

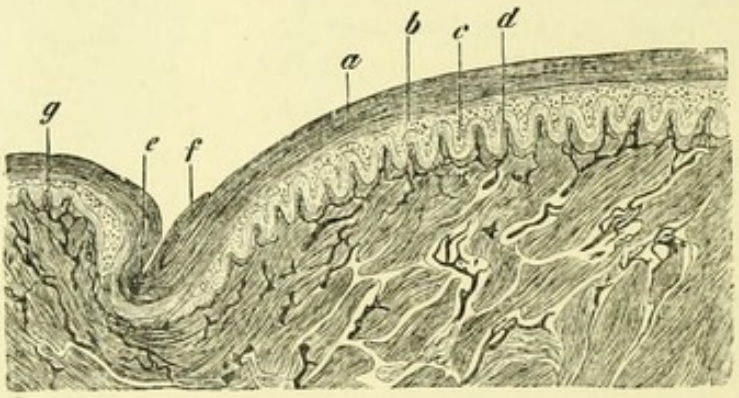

FIG. 14.—HALF OF A TRANSVERSE SECTION OF A NAIL THROUGH THE NAIL BED PROPER.

a, nail substance; *b*, loose horny layers underneath; *c*, mucous layers; *d*, transversely cut nail ridges; *e*, nail fold free from papillæ; *f*, the horny layers of the nail fold, which are pushed over the nail itself; *g*, papillæ of the skin of the back of the finger.

jecting somewhat beyond it. In connection with the nail must be mentioned the nail fold, the fold of the skin which covers in the posterior and two lateral borders; and the nail bed, the tissue on which the under surface of the nail is seated.

Besides the surfaces and borders mentioned there may be distinguished upon the nail the root, or part covered by the posterior fold, and the body, or part anterior to the latter; while the corresponding parts of the nail bed are called the matrix, lying under the nail root, and the nail bed proper, lying under the nail body. The *nail fold* in the upper half covering the nail is formed by a concave projection of the cutis and is called the nail wall; in the half turned toward the under surface of the nail it is formed by the posterior and lateral portion of the nail bed. The nail fold increases in depth from before backward, and is also made thicker by the fact that the epidermis of

the back of the finger is continued forward a short distance over the nail root. The inner anatomical relations are displayed in a transverse section, as is here represented (Fig 14) after Biesiadecki.

The *nail bed* is formed of rete, corium, and a subcutaneous tissue free from fat. Posteriorly, in the part corresponding to the root of the nail, and also in the vicinity of the matrix, broad papillæ, directed forward, rest upon mound-like eminences of the corium. At the curved border—the lunula—which, running parallel to the ball of the finger and seen through the nail, marks the separation of the matrix from the nail bed proper, these eminences become ridges —the nail ridges—which increase in height as they extend forward, and at the free border of the nail pass into long papillæ.

Contrary to the view heretofore generally accepted, that the entire portion of the nail bed bounded by the lunula is the matrix of the nail, Hans Hebra, as a result of his investigations, considers the matrix to be only that horseshoe-shaped portion formed by the posterior and lateral part, which possesses large papillæ, while the biconvex central space between this and the lunula is destitute of papillæ and has only ridges and is therefore not matrix.

The nail bed is nourished by a supply of blood vessels and nerves which penetrate the papillæ. The vessels of the matrix papillæ are of large calibre and often looped ; a rich vascular anastomosis from the pulp of the finger nourishes the nail bed, and, by means of ascending loops here and there, passes directly to the ridges, whose extremities have been shown to be abundantly supplied. The Malpighian layer covers the papillæ and ridges and fills in the spaces between. On the posterior angle of the fold it joins with the rete of the former, and forms a wedge pointing backward and extending into the corium. It is generally larger at the nail root, and here merges gradually into flat nucleated cells, which are easily stained by carmine, and finally into non-nucleated horny cells. The transition between the mucous layer of the nail bed and the flat epithelium is, on the contrary, sudden, as in other places in the skin, and the border between the epithelium and the nail substance is sharply defined. The under surface of the fold covering the nail root is lined by epidermis, which partly protrudes over the nail ; no nail substance is formed from this ; the former being derived exclusively from the papillæ of the matrix, which plays the same rôle for the nail that the papilla does for the hair. The horny cells are prevented by the lateral fold from extending toward the side, and are forced backward ; therefore the uppermost nail cells form an imbricated covering, the superficial being shoved over the underlying ones. Concerning the method of stratification to which the nail cells are in this way forced, Unna has expressed his opinion that the plane of stratification of the nail cells must run from behind and

above, forward and downward. The horny cells are very resistant to chemical influences.

The above-mentioned anatomical and histological relations call for a general presentation showing the great variety and at the same time the peculiar way in which the known pathological processes may affect the skin. One and the same process—*e.g.*, hyperæmia, inflammation, or hypertrophy—may affect only certain layers or tissues of this complicated organ, or may extend simultaneously to all the elements and systems entering into the structure of the skin. A decided hyperæmia, or even an exudation, can, as in pemphigus, affect the papillary layer, which has a special vascular network, while the deeper layers of the skin appear to be quite undisturbed. It may happen that the circulatory and nutritive changes exist just in the vicinity of the vascular anastomosis surrounding the glands, while all the interglandular tissue is free from disturbance ; or that the epidermis, in a manner possible only to itself, degenerates or becomes hyperplastic, as within the sebaceous glands or on the surface of the skin, without showing alteration of the parent tissue, either primarily or secondarily. As for the other processes which are treated of in this book, they affect other organs also, and are already known to general pathology and pathological histology. For it is not to be assumed that inflammation of the connective tissue of the skin, for example, differs from that of the liver. But the peculiar manner in which, on account of their special anatomical relations, certain parts are affected depends upon the special disease, its localization, its manner of spreading, and its characteristics —important facts which only special pathology can make us acquainted with. It will therefore be useful to discuss the latter and also the relations belonging to general pathology.

We must keep the *physiological significance* as well as the anatomical relations of the skin constantly in mind, in order to understand the course and progress of skin diseases.

The general integument functions in three ways—as an organ of protection for the body, as a secretory organ, and as a specific sense organ.

The *protection* which the skin, as an enveloping organ, gives to the body is mechanical. The three layers participate equally in this function, though in different ways. The fat of the subcutaneous connective tissue is especially concerned in protecting the underlying organs, muscles, nerves, and vessels from pressure, blows, and injuries which the daily contacts of life render unavoidable. The corium affords this protection through its firmness, great elasticity, and movability; while the epidermis, by means of the thickness and com-

plete insensibility of its corneous layer, as well as by its impermeability, resists injurious and poisonous agents.

The epidermis, moreover, aids materially in economizing the expenditure of heat and moisture of the body.

The subject of the *regulation of the body heat* and of the different factors concerned in the production of warmth will be discussed here only so far as it touches upon the maintenance of a definite physiological temperature in the body. Since the blood is of a higher temperature than the medium normally surrounding the body, the temperature of each would be made equal with the other were it not for the mechanism regulating and checking heat radiation. The mean loss of heat from a body of 82 kilos in twenty-four hours is 2,095 to 2,592 calories, or 1.36 to 1.60 a minute. The corneous cells are bad conductors of heat, and prevent excessive loss of body heat from the superficial papillary vessels—a loss which, by the removal of the epidermis, would, in view of the enormous surface space which the capillary system occupies, certainly cause such a degree of cooling as to effect the death of the individual. This has been experimentally proven. The horny layer also, through its coherence and firmness, exerts a pressure upon the underlying rete cells and the capillaries of the papillæ, and, thus supporting their walls, prevents their being overfilled by the heart impulse and consequently prevents excessive loss of heat from them. The degree to which the radiation of heat from the skin depends upon the condition of its superficial vascular system has been many times scientifically confirmed and definitely ascertained.

When the papillary vessels of the skin become distended, as under the influence of a high external temperature, or locally through mechanical and chemical irritants, or over the general surface by increased heart action (fever), or locally again as a result of the isolation of the blood currents in several blood vessels by ligation (Landois and Hauschild) or by paralysis of their walls, the loss of heat increases from a small amount to 100 per cent; while in portions of the skin which, through pressure of elastic bands, are almost deprived of their circulation, the loss of heat is materially lessened, being, for instance, 4.4° C., in contrast with a loss of 5.6° from the normal skin, as shown by an air calorimeter (Winternitz).

The epidermis is of especial value also in regulating the *fluid economy* of the body. As soon as the skin is broken in any place a great amount of serum oozes out from the rete cells, or really from the papillary vessels. This moisture continues until a new protecting horny layer is formed. When this exposure exists over a large surface of the skin the loss of fluid is important, and the individual is in a short time seriously affected by it—*e.g.*, in pemphigus foliaceus. Aside from the nervous irritation resulting from the exposure of the

papillary nerves when they are deprived of their protective epidermic covering and subjected to the irritation of the atmospheric air, other and more important injuries may be sustained. The result is also unfortunate when the horny layers of the epidermis are retained, but so reduced in thickness as to be of little use, as in certain skin diseases where the thinness of the layer favors too great heat radiation, which causes the invalid to experience a feeling of continued chilliness.

As a *secretory organ* the skin functions in a specific manner by means of its sweat and sebaceous glands and its papillary vascular system. Concerning the mechanism of the two we shall speak more in detail in the chapter pertaining to the diseases which affect them. Here we are concerned only with the imperceptible secretion which takes place through the papillary vessels and which is known as perspiration, a portion of which represents the *cutaneous respiration*. A well man, according to Séguin, loses through his skin in twenty-four hours one sixty-seventh of his whole body weight, which is as great as the amount given off by the lungs, and chiefly through loss of carbonic acid and water. Of this, according to Scharlinger, only 10 grammes (according to Aubert only 3.9) are carbonic acid, the remaining loss being water. Whether the skin also gives off nitrogen is uncertain. On the other hand, the skin takes up oxygen in about the same volume as the carbonic acid exhaled.

The gaseous interchange by means of the skin is so small—carbonic acid exhalation $\frac{1}{220}$, oxygen inhalation $\frac{1}{180}$—as compared with that of the lungs, that when it is artificially suppressed scarcely any perceptible effect upon the organism ensues; a fact which deserves to be especially emphasized, since many have attributed the results of certain skin affections to the suppression of the cutaneous respiration.

In what manner the exhaled gases and the fluid from the cutaneous vessels make their egress is not definitely known. No doubt the sweat glands offer the chief path of exit; in fact, this is generally admitted. It remains only to be decided whether, aside from the secretion by the glands of matter through the excretory ducts, there is not also a discharge of water and carbonic acid from the capillaries of the papilla directly through the stratum of epidermis. Against this view stands the apparently positive fact that the horny cells, as such, are under ordinary circumstances scarcely penetrable by fluids and gases, since upon this very property depends to a great extent the physiological function of the skin as a protective organ and as a regulator of heat and moisture. It can only be questioned whether the spaces between the epidermal cells, which are connected with the lymph spaces of the rete cells, may not offer a sufficient passage for the above-mentioned exhaled products. The same query,

too, comes up in considering the question of the so-called *absorptive power* of the skin. A certain power of absorption is to be ascribed to the skin by virtue of which it is able to take up certain dissolved or finely divided substances, to absorb them, and to take them into the circulation. This absorptive power is generally slight, more so than is often supposed—*e.g.*, by those who use medicated baths. The corneous layers of the epidermis are almost impervious to liquids and finely divided solid substances, as may be inferred from the researches of Fleischer. But no doubt, under certain circumstances, absorption does take place through the unbroken epidermis, as in the case of mercury when used by inunction, or of tar, iodine, naphthol, and fat, and also of substances dissolved in chloroform, ether, and alcohol when applied to the skin (Parisot, Röhrig, R. Winternitz). Indeed, according to Parisot, absorption occurs even from watery solutions through the palm of the hand, which is free from sebaceous glands and fat. Recently Paschkis and Obermayer have demonstrated that salts of the alkali metals, independently of the method of application, and also, as it would appear, independently of the nature of the acid, are absorbed by the skin; the same holds true of metallic arsenic suspended in oil or vaseline.

It must be granted that in some of these cases the absorption is accomplished by mechanical pressure—brushing or rubbing; in others it takes place through the hair follicles and the sweat glands, whose walls are covered only by a single layer of epithelial cells; or it occurs in portions of the skin which over a small area, not perceptible to the naked eye, have lost the horny layers of the epidermis, but are still covered by the cells of the deeper layers, which are more easily penetrated by substances finely divided or in solution than are the complete corneous upper layers of the epidermis; or, as Ritter states from his researches, absorption occurs as a result of the chemical effect of the penetrating substance, the epidermis being loosened and swollen or its vessels thrown into a state of inflammation. In the rabbit, at all events, absorption of fat in great amount takes place after a mere pouring of oil over the hairy parts (Lassar).

The skin performs its most important physiological function in being a *specific sense organ*, the organ of touch. As such it receives, by means of the sensitive nerve endings in the touch papillæ, impressions from without, which in general are designated collectively as sensations of touch or general sensations. These latter are very different in quality, since they reach our consciousness under the form of sensations of pressure, pain in all its varieties, burning, pricking, tickling, itching, temperature sensations, etc., thus affording us notions in regard to the physical condition of external bodies and placing us in proper relation with the outer world. The sense

of touch occurs also as the so-called *common sensation*, by means of which the condition and difference in situation of the portions of skin that are irritated are recognized and precisely determined—this constituting the ability to localize, or sense of locality. This specific sensory power, dependent upon the distribution of touch papillæ, is developed differently on various parts of the skin, most so upon the finger points, where the papillæ are abundant, and upon the lips. The most instructive results in this direction are due to the well-known researches of E H. Weber, who by means of the so-called æsthesiometer has tested the degree of sensitiveness in the different portions of the skin and of the resulting perception—*i.e., of the space sense.* This is the more marked when the touch nerves are more abundant, and is greater according to the mobility of the skin. So it has been ascertained that on the application of an æsthesiometer to the skin of a grown person the two points of the æsthesiometer can be distinguished on the volar surface of the third digital phalanx at a distance of 2 3 millimetres ; on the second phalanx, at 4 5 millimetres; on the dorsal surface of the third phalanx, at 6.8 millimetres; on the plantar surface of the big toe, at 11.3 millimetres. Yet circumstances, aside from the difference in locality, exert an influence upon the degree of variation in perception. Means and methods for these investigations have since then been perfected.

All the previously mentioned varieties of tactile sensation are appreciated by the sensory nerves. According to Landois' view two kinds of fibres which are functionally distinct are found in the latter. These are : 1. Those which give rise to impressions of *pain*, and are, in a narrow sense, sensitive nerves. 2. Those which serve for the tactile sensations, and are therefore known as touch or *tactile* fibres. These latter at the same time appreciate *temperature* and *pressure*. They derive these sensations, however, from mechanical and thermal impressions acting upon their terminal tactile apparatus only, and when they experience such stimuli along any other point in their course, as in knocking the ulnar nerve, painful and not tactile sensation is aroused. Goldscheider, from his own investigation upon this subject, has decided that the terminal mechanisms for the appreciation of temperature, at least, are anatomically distinct from those of touch. Goldscheider, moreover, following the theories of Müller and Helmholtz affirming the specific energy of the sensory nerves, has, after prolonged tests, decided that not only the points on the skin sensitive to pressure and temperature are distinct, but those for the appreciation of heat and cold do not lie together. The sensitive points are arranged, according to Goldscheider's last determination, in linear series radiating from certain points of the skin, which are points of radiation or centres of sensation. Each series is combined with the neighboring ones, forming a sort of figure. The

points of radiation for the temperature series coincide with the points of the pressure series and are generally localized near the hairs of the skin ; not at their point of exit, but at the site of their papillæ. The different sets of series for the different sense qualities strike sometimes the same direction, sometimes are divergent, and are variously combined. According to Goldscheider we must assume that the nerves conveying the sensation of cold, warmth, and pressure are perfectly distinct and have an entirely different distribution, although for the most part they appear to run united up to the points of radiation. It is highly probable also that the nerves of sensation and touch possess a separate terminal nerve apparatus and separate fibres, and that even in the brain they have distinct centres, although nothing positive is known concerning this. In favor of this view are :

First, the fact that all organs provided with sensory nerves do not convey sensations of touch, pressure, and temperature, as does the skin, but, as in the case of the intestines, are at the best capable only of conveying the sensation of pain.

Second, the fact that it is found in pathological conditions and as a result of experiment that the paths of the nerves of touch and of sensation run separate in the spinal cord. The course of the localized sensations of touch lies through the posterior roots into the ganglia of the posterior horn, and from there into the lateral column upward to the brain (C. Ludwig and Woroschiloff). The course of the sensations of pain lies through the posterior roots and the whole gray matter, the smallest fibre of which, after the destruction of the others, is capable of transmitting the sensation of pain.

LECTURE IV.

GENERAL SYMPTOMATOLOGY, SUBJECTIVE AND OBJECTIVE—PRIMARY AND SECONDARY LESIONS—CLASSIFICATION OF ERUPTIONS.

GENERAL SYMPTOMATOLOGY.

On account of its histological and physiological peculiarities the skin is affected by disease in a peculiar way, although the pathological processes and the tissue changes resulting from them really correspond with those of other organs. Now, these peculiarities must of necessity produce peculiar symptoms, a thorough knowledge of which is indispensable to the comprehension of the pathological processes. The symptoms through which the nutritive and functional changes in the skin manifest themselves may be divided in general into subjective and objective ones.

The *subjective symptoms* are limited to disorders of sensation. They occur under the form of a diminution of sensibility (anæsthesia, paræsthesia) ; or an increase of sensibility coupled with a change in its character, as in pain (neuralgia), itching, tingling, tickling, and the sensation of ants crawling over the skin (formication) ; or as an alteration of reflex irritability. Naturally most of the symptoms of this class can only be known through information derived from the patient ; some, as anæsthesia and itching, however, are revealed objectively, the subjective symptom of itching being recognized through the objectively perceptible signs of scratches and excoriations.

The *objective symptoms* of skin diseases are most numerous and varied. They form the necessary and most positive basis for the recognition and comprehension of the pathological processes occurring in the skin. Their thorough study cannot be too strongly recommended. They are, to use a not unfitting metaphor, the written characters which each disease has itself described upon the skin, and which mark the degree of its intensity, its localization, course, the manner in which it has spread, and the time during which it has lasted, so that we really need only to read off these written characters with judgment to know the disease, its origin, complete course, and character.

The objective symptoms of disease correspond to observable tissue changes, and consequently to those skin diseases which result in a

demonstrable alteration of nutrition—using this term in its most general sense. Since affections of this sort in the skin are essentially the same as those of other organs and systems, one would judge that a similar agreement existed in their symptoms and that the latter are in no way peculiar to the skin. This is actually the case.

Hyperæmia manifests itself in the skin by distention of the blood vessels and redness; anæmia by pallor; inflammation is characterized by redness and swelling, etc. The symptoms, however, are more obvious here than elsewhere, not only on account of the direct accessibility of the skin to observation by the senses, but also, and peculiarly so, because, first, we observe them on the living organ; second, because the special anatomical structure of the skin results in a correspondingly peculiar arrangement of pathological processes; and, finally, on account of the specific etiological factors, which can have effect only upon the skin and therefore produce only specific results.

These concurrent conditions, in conjunction with some others not yet considered, produce a certain *type*, to which a local skin disease must regularly correspond, without reference to the far-removed origin of the disease. When, for instance, through blood-poisoning, as in small-pox, inflammation and suppuration of a hair follicle ensue, this local disturbance will develop according to the same type and afford the same symptoms as the inflammation and suppuration of a skin follicle which has become affected by scratching with the finger nail or by irritation from sweat. The anatomical structure and the course of the blood vessels of the follicles are typical and therefore the forms of their inflammation are always similar.

Such types of symptoms are constituted by the so-called eruptions of the skin. The expression *eruption* dates from a time when the external appearances in skin diseases were regarded as the criterion by which the nature of the latter should be determined, while the real pathological processes which caused these appearances were either not known or were ignored. The name is retained to-day, although with a concrete pathological significance. By eruption is now meant a morbid change occurring in the skin, which generally is small, limited in circumference, and in its form (morphologically), development, course, and anatomical significance retains a certain type. The eruption receives a distinctive name according to the nature of this type, so that the term employed in any given case is always associated with the idea of a single definite form of eruption and no other. It does not follow, however, that we can take arbitrary liberties with the terminology of skin diseases. We must, on the contrary, adhere to names and concepts already firmly established and generally received, for the stability of which,

as we have already stated, most has been done by Plenck, Willan, and Hebra.

The pathological process which produces the eruption reaches its highest point locally in the typical development of the latter. The symptoms due to the eruption, therefore, are synonymous with the local and typical primary symptoms of the disease itself (efflorescentiæ cutaneæ primariæ). Secondarily the efflorescence changes by still further development, extension, transformation, or retrogression, partly through the arrest of the original local process of the disease, and partly, after cessation of the latter, through reparative changes by which the normal processes of nutrition compensate for the existing tissue disturbances. The lesions produced in this way, which necessarily result from the primary ones, are called the secondary lesions.

The *primary lesions* or eruptions appear in the following types:

1. Macula, macule, spot. 2 Papula, papule. 3. Tuberculum, tubercle. 4. Phyma, tumor. 5. Urtica, wheal. 6. Vesicula, vesicle. 7. Bulla, bleb. 8. Pustula, pustule.

Macula, or spot, is the name applied to any circumscribed area of skin of abnormal coloring; such areas occur in red, brown, and various shades of yellow and white. Their form and size are equally varied. They are changeable, and transient or permanent, hereditary or acquired. Those in shades varying from bright to dark red may arise from hyperæmia of the papillary layer and upper part of the corium, in which case they disappear upon pressure. If exudation is at the same time present the spots are somewhat raised, and pressure of the skin at their site reveals a yellow streak. Such spots, when from a lentil to a finger nail in size, are called *roseolæ*. When the redness is diffused over a great area it is known as *erythema*. Hyperæmic spots in which vessels run that are apparent to the naked eye are called *telangiectases;* when hereditary, *nævi vasculosi* (vascular moles). A hyperæmic spot in the middle of which is another eruption constitutes an *areola*. In other cases the red spot is created by a hæmorrhage into the papillary or upper layers and does not disappear under pressure of the finger. It is then called *purpura*. Hæmorrhagic spots when punctate are called *petechiæ;* if in streaks, *vibices;* when large and irregular, *ecchymoses*.

Bluish-red, greenish-yellow, and yellowish-brown spots are produced in the process of involution of hæmorrhages.

Yellowish-brown to dark-brown and black spots (*nigrities, melanosis*) result from the overabundant deposit of pigment in the rete cells of the deepest layers and also in the upper layers of the skin. They appear on the face as broad areas or striate markings, called *chloasma;* or there, and also on the hands and other portions of the

body, in spots of a size varying from the head of a pin to a lentil, being then known as freckles (*ephelides* or *lentigo*) and *nævus pigmentosus*, or *nævus spilus* (pigment moles).

White spots are due to a deficiency of pigment; they are hereditary and limited to spots (*achroma*) or general (*albinismus*). They are acquired in the course of life as a result of other pathological processes, or are idiopathic (*leukopathia, vitiligo*).

Spots on the eyelid and vicinity, varying from a straw to a citron yellow, are formed from tissue alteration in the corium and constitute *xanthoma* or *vitiligoidea*.

Besides the more typical anomalies of color already described, there are also discolorations (*dyschromasiæ*) of the skin which denote some alteration of the general nutrition of the body, as the waxy color in chloranæmia, the dark-yellow discoloration in cases of carcinoma, and the bronze shade in Addison's disease and leprosy. They are observed also when foreign substances are deposited in the cutis, as the yellow resulting from the bile in icterus, the red and blue from tattooing with cinnabar and carbon, the gray from deposits of silver in argyria.

Papula—papule—is the name applied to a solid, elevated, pathological formation varying in size from a poppy-seed to a lentil. Papules are round, conical, or flat, red or pale, firm to the touch or compressible, and, generally speaking, are varied in their special characteristics according to their site and their constituent elements and the pathological processes producing them. Sometimes superimposed layers of dry epidermis predominate in the formation of the papule, as in psoriasis; again it is formed by exudation and cell infiltration into the rete layers, as in eczema papulosum; or by hæmorrhage into the rete and the papillæ, as in lichen hæmorrhagicus; or from a collection of epidermic cells in the mouth of the hair sac, as in lichen pilaris; from the acini of the sebaceous glands becoming distended and filled with solid contents—milium; or from new tissue formation in the corium, as in lupus, syphilis, and sarcoma. According as the pathological processes are different, so must the duration, course, and, above all, the nosological significance of the papule be different. Some are stable, as milium; others very changeable, as for instance the papules produced by inflammation, which are capable of undergoing rapid transformation. Acute inflammatory papules which are surrounded by an areola are known as pimples.

Tubercula—tubercles—are circumscribed, firm, pathological formations of the skin, larger than the papule, and varying in size from a pea to a hazelnut. They may be quite embedded in the tissue of the skin, so as to be appreciated and recognized only by the examining finger; or they are somewhat elevated above the surface, forcing

the epidermis before them. Their pathological significance is similar to that of the papule.

Phyma—tumor—is a term for quite large tumor-like formations which are situated in the subcutaneous tissue, or both there and in the corium, and which, by pushing forward the tissue, form large elevated or pendulous tumors.

Urtica—a wheal—consists of a flat, prominent, solid elevation of the skin, slightly red or glistening white, with red periphery (urticaria porcellanea). It varies in size from a lentil to a thumb nail, and is round or irregular in shape. Each wheal develops suddenly, indeed almost in a moment, and runs a very short course. It originates from a circumscribed, chiefly serous, exudation into the papillary and rete layers. The wheal may spread peripherally while its central portion disappears, resulting in circular and gyrated forms of wheals. The sensation of burning and itching is always associated with their presence.

Vesicula—vesicle—is an elevation of the epidermis, varying from a millet seed to a lentil in size, filled with a transparent and watery or milky, rarely a bloody, fluid. The transparent watery vesicles represent the regular type; they contain a serous exudation which, after rupture or injury of the vesicle wall, exudes in clear watery drops. The normal vesicle is transparent; only after some duration do the contents become opaque through an admixture and metamorphosis of the constituent elements (cells, granules, molecular masses). Hæmorrhage sometimes occurs in the beginning, but generally later, and makes the contents a dark bluish red and cloudy. The color of the base of the vesicle contributes to the color of the vesicle itself, according as the former is white or red or blackish (hæmorrhagic). Many vesicles are hemispherical, others acuminated, still others present a shallow depression in the centre (umbilication). They vary also in their consistence. Many are very firm and bear considerable pressure without rupturing; such vesicles have a thick wall; such a vesicle, also, is said to be deeply situated. Others have a very thin wall which is easily ruptured and readily allows its contents to exude; these are superficially seated.

The vesicle is always a product of an acute serous exudation from the papillary vessels, and is produced by the fact that the exudation collects in certain spots, forming masses in the epidermic layers. In this manner the rete cells become swollen and pushed apart; a network results. The impermeable horny layer, on the other hand, is arched forward and forms the wall of the vesicle; hence the vesicle wall is thin and delicate or thick and firm, according as the exudation is near the surface or is deeper and nearer the papilla.

Vesicles similarly arise when a free serous exudation takes place between the epidermic strata surrounding the mouths of the follicles and glands, and extends into the latter.

The minute anatomical relations concerned in the formation of the vesicle are very instructive and have already occupied the attention of many investigators. We will speak more exhaustively upon this subject in another place.

Each vesicle, as such, has a short existence. It either disappears through the drying up of its contents or through purulent change, passing then into another kind of eruption—the pustule.

The same holds true of the efflorescence called *Bulla*—bleb. This resembles in all respects the vesicle, from which it is differentiated only by its greater size, from a bean or nut to a hen's egg. There are blebs in which the contents are chiefly serous, others in which they are opaque or bloody. Some are superficial and possess a very thin epidermic covering, as in pemphigus ; others are deeply situated and include the whole mucous layer, as many burns and blisters.

Pustula--a pustule—is a yellow, yellowish-green, or brownish-green elevation of the epidermis filled with pus. Its base is formed generally of reddened skin, since the occurrence of pus presupposes an intense local inflammation, of which it is the result. Frequently the pustule is so situated that its centre is occupied by a hair follicle, the duct of which appears to be filled with pus. Several kinds of pustules were formerly distinguished : *achor*, a pustule of the last-named variety, in whose centre a hair is situated, a kind of pustule chiefly found upon the head ; *psydracium*, a pustule of large circumference ; and *phlyzacium*, a large pustule, the contents of which are bloody. Yet these characteristics are in no way constant and are of little use in practical terminology ; much more frequently one meets the name *impetigo* for the small and superficial pustules, and *ecthyma* for the large and deeply seated ones.

Although the idea of a pustule implies the existence of pus simply between the layers of epidermis, yet generally this is true only for the first part of its course. Later it may affect the papillary tissue which forms its base and cause suppuration in it. When only the epidermis is destroyed in this process the injury will be repaired through the reproduction of epidermis—that is, the pustule will heal without leaving a scar ; but if the connective-tissue portion of the skin and the papillæ are destroyed by suppuration, it heals by the formation of new connective tissue—*i.e.*, by means of a scar.

It has already been repeatedly shown that the primary lesions heretofore described must pass in their regular course into other local forms, which, however, are also of a typical kind, and which, in

contrast to the former, are called secondary. We group them together as *secondary lesions.* They are: 1. Excoriationes, excoriations. 2. Ulcera cutanea, ulcers. 3. Rhagades, fissures. 4. Squamæ, scales. 5. Crustæ, crusts. 6. Crustæ lamellosæ, scaly crusts. 7. Cicatrix, scar. 8. Pigmentation.

Excoriationes—cutaneous or epidermal excoriations—are what their name signifies, solutions in the continuity of the epidermis, usually the horny layers. In spite of their anatomical insignificance they play a great rôle in dermatological diagnosis and pathology. Their form, number, localization, and the objective symptoms produced by their frequent repetition are important for the diagnosis of certain processes of disease. If one irritates the epidermis only to the depth of its horny layer by scratching with the finger nail or by means of a needle, there results a corresponding furrow with a border of fine epidermic scales. As a result of the mechanical irritation in this case, the papillary blood vessels contract, so that a white, anæmic line appears, which, according to the irritability of the site in question, remains for several or more seconds until the vessels, dilating and filling quickly, produce a red, hyperæmic streak which remains for several minutes. (Often the original anæmic white line appears at once, flanked by red, hyperæmic skin.) The redness and exfoliation along the scratch mark disappear, the former quickly, the latter gradually through replacement by new epidermis. But where a place becomes excoriated by continued scratching, then the hyperæmia recurring so often leads to the exudation of blood corpuscles and a deposition of blood pigment, so that brown streaks remain for a long time. If the excoriation extends so deep that the mucous layers are exposed, the excoriation appears grayish blue and moist. Serum is discharged from the rete cells; this dries into a yellowish-brown mass and is thrown off after a time by the epidermis underneath, which, as it becomes cornified, pushes it forward. If the excoriation reaches as far as the papillary bodies, then the blood vessels of the latter become partially injured and some blood exudes (bloody excoriations). All these heal finally without leaving any trace, since only epidermis has been lost. But these excoriations can be associated with more considerable injury to, and destruction of, the papillary structures, as when the mechanical power which produced them was intense—*e.g.*, from scratching in case of intense itching, as in prurigo and pruritus cutaneus—or still more readily when the epidermis and the papillary layers, having themselves become spongy and swollen from previous disease, are more susceptible to injury, as is the case when tubercles, blebs, and pustules are present over which the excoriations were made. They are then deepest in the places where these inflamed spots exist, and are differently shaped according to the form of the latter. In the case of

urticarial wheals, for example, where the epidermis over a great extent becomes moist and spongy, the excoriations are broad, deep furrows, whereas with the small tubercles of prurigo they appear as blood-colored epidermal defects of the size of a millet grain.

Ulcera cutanea—ulcers of the skin—are secondary lesions. They exist only in a previously inflamed or otherwise diseased portion of the skin, and mean a loss of substance involving the corium. Their surface discharges a secretion differing from normal pus, and they are incurable, or nearly so, because the granulating process, which should make good the loss of substance, is delayed or disturbed by local or general causes. Every ulcer has a base and a margin—that is, the inner edge of the border. The character of both will be studied later in the lectures upon special pathology—viz., whether the margin is smooth, serrated, eaten away, undermined, or elevated; further, whether the form is round, crater-like, dish-shaped, kidney-shaped, or serpiginous; whether the course is acute or chronic, and many other facts which relate to the causation, significance, localization, etc.

Rhagades, Rimæ cutis—skin fissures—are furrows or cleft-like tears and openings in the epidermis. They sometimes extend deeper, even into the corium, and are there bordered by steep margins and are bleeding or ulcerated at the base. They result from the stretching and tension exerted by the underlying muscles when from disease the skin has become less elastic and its epidermis more brittle.

Squamæ—scales—is the term applied to the corneous plates which separate from the surface of the skin. Under normal physiological conditions shedding of the epidermic cells always takes place, but is imperceptible, their place being regularly supplied by transformed rete cells. In pathological conditions this shedding is perceptible, and is called *desquamation* when it is observed as a result of a local pathological process. When it exists as an independent affection it goes by the name of *pityriasis*. The scales occur under the form of small and branny, or larger, thin and glistening or dirty white, and dry or fatty plates, or as thick, flat lamellæ, or, finally, in the form of large, coherent, parchment-like masses which, for example, appear in the form of a glove finger, corresponding to the finger of the hand. Accordingly desquamations are classed either as desquamatio furfuracea, membranacea, or siliquosa. In certain diseases (psoriasis) the scales are grouped in masses or in several large plates, which are loosely connected with the deeper cells, but, as a whole, cleave for a long time to the skin and exfoliate only in their uppermost layers.

Scales are formed also from the fat glands, an abnormal quantity of fatty epidermis being constantly secreted and collected upon the cutaneous surface (seborrhœa sicca).

Crustæ are masses which remain upon the skin through drying of outpoured serum, pus, or blood. The former in a fresh condition are the color of gum or honey; the latter shade from brown to black. In the beginning the mass is soft and elastic, but through age becomes hard and brittle, and, through internal metamorphoses and various admixtures, becomes discolored. Their size corresponds, as a rule, to the quantity of outpoured liquid, and they may become very thick through exudation, pus, and blood slowly and gradually added to and dried upon their under surface. The shape generally conforms to the configuration of the affected portion of skin from which serum, pus, and blood are secreted. Special forms result when the suppurative process which causes the crusting advances from the centre peripherally ; the crusts then appear massed together as concentric rings or discs, the central ones being the smallest and oldest. The crust also may be either umbilicated in the centre (concave) or conical (convex) : the former, when the process of exudation clears up in the centre ; the latter, when the exudation, notwithstanding its peripheral progress, still continues in the centre and contributes material for the thickening of the crusts. The last-named form of crusts gives its character to the so-called *rupia*.

Crustæ lamellosæ are a mixture of crusts and scales.

Cicatrix—scar—is a new formation of connective tissue in the skin which replaces a loss of substance of the connective-tissue portion (not the epidermis). It has a smooth, shining surface, devoid of the regular elevations, lines, and furrows of the normal skin, and without pores, hair, and papillæ. Fresh scars are red ; older ones are shining white, having sometimes a brown pigmentation on the periphery ; their consistence is firm but varies. Their surface lies either on a level with the plane of normal skin or somewhat below, although often projecting above it (hypertrophic cicatrices). The size and form of scar do not correspond exactly to the loss of substance involved, since during their formation and still later they shrink. A good scar is thin, soft, smooth, and movable ; a bad one is swollen, hard, uneven, raised, and crossed with bands.

The scar consists of an irregular network of new connective tissue ; in fresh scars this is more or less homogeneous and rich in connective-tissue cells, round cells, and blood vessels. With increasing age the intercellular substance becomes more clearly fibrous and contains fewer cells and vessels and less fluid.

Pigmentation occurs as a sequel of some past process which was associated with hyperæmia. It is, therefore, the result both of inflammatory and of neoplastic processes. The form and extent of the coloration are determined by the way in which the original process was localized and the course it followed. It is either permanent or transient. In the latter case it is most intense in the spot that

has been most recently affected, and shows the greatest diminution and disappears the soonest on the part that was the earliest attacked.

Connected with the morphological peculiarities of the eruptions described there is a series of appearances, very important for symptomatology, which arise from the peculiarities of their distribution, arrangement, and mode of extension, and for which there is no analogy in the pathology of other organs. Efflorescences are found isolated upon the skin (efflorescentiæ solitariæ), or separated into many groups (efflorescentiæ discretæ), or disseminated (efflorescentiæ dispersæ), or irregularly massed (efflorescentiæ aggregatæ, confertæ), or collected in regular masses (efflorescentiæ corymbosæ), or arranged in simple circles (annularis, circinatus); and it seems as though there were no local or general cause for, and indeed no regularity at all about, these conditions. Nevertheless there is a remarkable constancy, and in many cases a regularity bordering upon law, in the localization and arrangement of the efflorescences, and in the distribution of the various lesions, which in the aggregate are known as a skin eruption or exanthema. It finds its partial expression in the fact that the efflorescences in general appear *symmetrically* upon corresponding portions of either side of the body—upon the palms or backs of both hands and both knee or elbow joints. To account for this peculiarity of localization of eruptions, already noticed in a previous age, but especially particularized by Hebra, it was supposed that in many cases there existed an irritation of the vaso-motor centres lying in the gray axis of the spinal cord. At least Jarisch, in an autopsy upon a case of acute febrile vesicular eruption with symmetrical localization which came under observation in our clinic, demonstrated inflammatory changes in the central, lateral, and posterior portion of the anterior horns of the spinal cord at the same height as the skin eruption, and he states that he has seen the same in cases of syphilis and lupus erythematosus. Even granting that this state of things was not pathological, but accidental or artificial, occurring in the process of hardening, still the idea that a rôle is played by the vaso-motor centres in symmetrical localization seems warranted by the fact that after producing disease artificially upon a portion of the skin, as in the hollow of the knee, it is found after several hours that the knee hollow of the other side is affected, and often other portions of the skin still become diseased in a manner similar to that artificially produced. Further, this law or peculiarity is noticeable: that the eruption in many processes is pronounced upon the extensor surfaces, in others regularly appears by preference upon the flexor surfaces of the joints and extremities, or on the parts around the openings of the body. At present an explanation of this law is wanting. On the other hand, the anato-

mical and structural arrangements of the skin furnish an apparent explanation for a number of other laws governing the distribution and mode of extension of eruptions. It is certain that the latter, in their site of origin and method of extension, follow exactly the direction of the nerves of the skin. This is the case in herpes zoster, in many papillary moles, in pigment moles, and in exanthemata, though there is not necessarily a causal relation between them and the nerve itself. In the first-mentioned disease this is the case ; in the latter the relation is only apparent, and in reality is produced by the correspondence of the lines of direction of all the anatomical structures constituting the papillary layer. In the classical works of Türck, which were published by Wedl, and in those of Voigt, one can inform himself concerning the course and ramifications of the nerves of the skin and recognize this correspondence. Voigt has also proved that there is a system of lines and regularly placed whorls upon the skin which correspond to the arrangement of the hair sacs. Since often, and in many diseases—*e.g.*, lichen scrofulosorum—the separate efflorescences constituting the eruption regularly affect the follicles themselves, it is conceivable that in their arrangement they should reproduce the configuration of the follicular distribution and appear disposed in regular groups. One often sees the eruptions, as in psoriasis and herpes tonsurans maculosus, arranged in numerous long, parallel rows, which on different portions of the body follow different directions, but in regions that correspond to each other are always arranged in the same way. On the side of the thorax they are parallel to the ribs, on the shoulder and neck are in concentric rings which have in all cases the same point as a centre. There are certain regions which lie as neutral points between these separate wave-like systems of circles—a highly interesting consideration to which Hebra paid great attention.

These conditions are founded upon different facts, and first of all upon the *lines of cleavage* of the skin. It has been long known that in certain portions of the body the skin has these lines of cleavage. When the skin is pierced with a round shoemaker's awl the opening made is not round but is a longitudinal fissure, and this has a different direction on different parts of the body, but a constant one for each locality. C. Langer has experimented upon a large number of cadavers, and has produced a series of pricked lines by puncturing, of which he has made a drawing (Fig. 15). These lines of cleavage correspond to the chief direction of the fibres. The main trunks of the nerves and blood vessels follow them, indeed so do the follicles also ; and the chief direction of all is governed on the one hand by the site of certain points of fixation in the bones and fascia, and on the other by the direction of growth of the whole man —viz., of the extremities—as Voigt has demonstrated. The cuta-

neous vessels, nerves, and the connective-tissue fasciculi of the skin follow along lines laid down by the law of growth, so that those diverging from the spinal column on the side of the thorax run parallel to the ribs; those on the forearm go in spirals running from above outward, downward, and inward. The arrangement and distribution of the efflorescence are predetermined, sometimes by one,

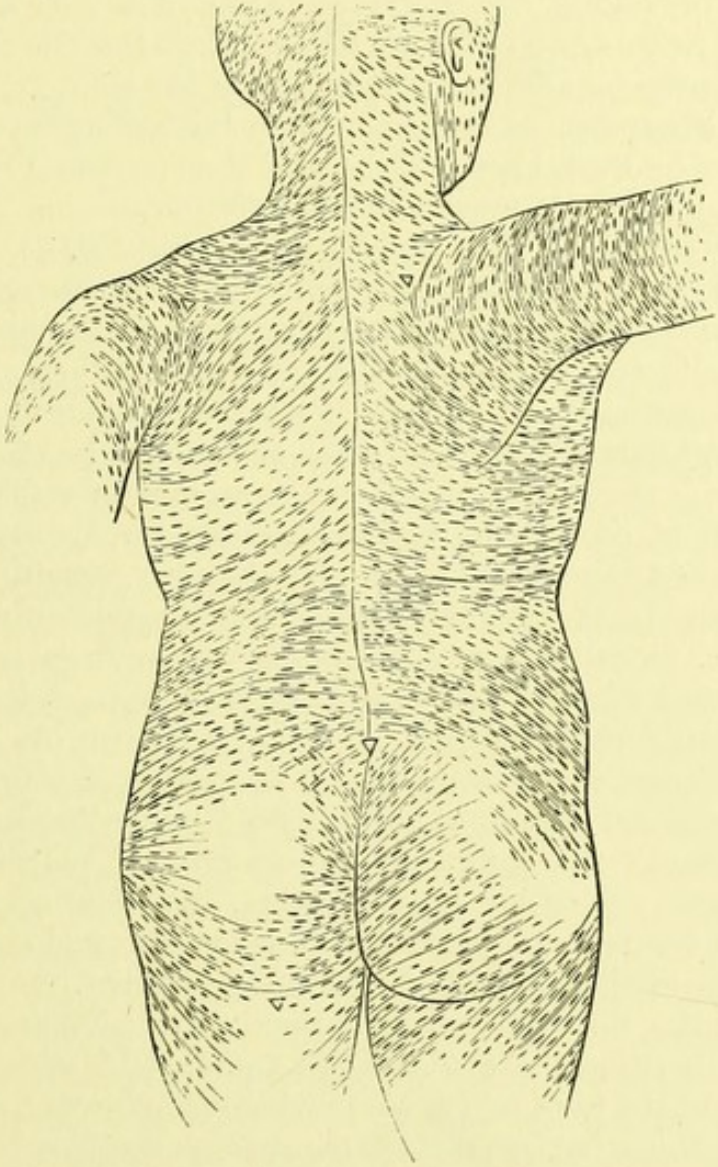

FIG. 15.—The lines of cleavage of the skin according to Langer.

sometimes by another of the above-mentioned causes, and sometimes in a measure by the union of all. O. Simon has demonstrated that the direction of ulcers changes at once when the tension upon the skin is relieved by cutting around or excising the portion of the skin involved. Wertheim also refers the direction assumed by ulcers to the same cause. All the peculiarities of arrangement are not, however, made clear by the conditions above stated. I believe

that the cause of many of them lies in the distribution of the small and the very small vessels, which has been but little studied. This belief is based upon the fact that a varying number of papillæ are supplied by each separate terminal vessel (see page 27).

The individual efflorescence appears to be limited in its further course also by the lines of cleavage. When the efflorescences, including even the deep penetrating ulcers, extend peripherally, they acquire the prevailing direction for the region of body affected. So the spots of roseola and of herpes tonsurans on the side of the thorax show oval forms parallel to the long axis of the rib.

Yet the efflorescences in their further development spread beyond the limits set by these laws. The forms thus produced must, in giving the symptoms, necessarily be noted for purposes of classification and characterization. We note them as *circinatus*, *annularis*, when an efflorescence presents the form of a circle through peripheral advance and central involution; *iris*, applied to the appearance of efflorescence in two or more concentric circles or in a circle with a primary efflorescence in the centre; *gyratus* applies to the form of winding markings which result when several circles of efflorescence in the course of their development come into contact with one another: at the points of contact the pathological process, redness, and infiltration disappear and only the peripheral arches remain.

The extension of the efflorescence, and consequently also of the skin disease, is called *per continuum* where from one point of origin it passes steadily on over the adjacent skin, and *per contiguum* where different portions of the skin which are in juxtaposition are affected in the same way. A number of other characteristics or incidental variations are described, according to their form, as discoid, scutiform, figuratus, circumscriptus, marginatus, confluens, diffusus; the color, as variegatus, intertinctus; and a great number of accompanying peculiarities, symptoms, and circumstances which relate to the age and the sex of the patient, the intensity of the disease, the time of year, geographical or historical data, etc. The terms denoting these characters simply add somewhat to the significance of the term which characterizes the disease, and in general imply nothing but what their etymology signifies—as, vernalis, æstivus, autumnalis, hiemalis, septentrionalis, tropicus, senilis, infantum, acutus, chronicus, febrilis, apyreticus, pruriginosus, agrius, mitis, etc.

LECTURE V.

GENERAL ETIOLOGY—IDIOPATHIC AND SYMPTOMATIC LESIONS—CLINICAL CONCEPTION OF WHAT CONSTITUTES A SKIN DISEASE—GENERAL DIAGNOSIS.

GENERAL ETIOLOGY.

A THIRD factor which gives an individual stamp to the pathological processes of the skin, in spite of their essential similarity with the diseases of other organs, lies in their *causes*.

These causes are partly the same that lie at the basis of diseases of other systems, but for the greater part are specific, since many cannot affect other organs as they do the skin. Moreover, a special significance is associated with the causative factors, in that frequently a perfectly specific form of skin disease corresponds to a specific cause; while, again, many other etiological factors may produce different kinds of disease, or, finally, the same form of skin disease may be occasioned by various factors.

The agreement between forms of disease and causes is hence by no means one that holds good generally. This circumstance, together with a second, that the etiological factors for many diseases are not known, makes it for the time impossible to systematize skin lesions upon the basis of their etiology.

In general skin diseases are divided, according to their causes, into two categories

1. Those which are caused by the conditions of the blood and fluids in the organism itself or by the state of the general system; or are caused by disease of individual organs and systems; or come from hereditary conditions. Such diseases are the essential or incidental symptoms of these states and conditions—*symptomatic skin lesions.*

2. Skin diseases which are produced by some agency affecting the skin directly—*idiopathic skin lesions.*

In earlier times pretty nearly all skin diseases were considered symptomatic, belonging to the first-mentioned category, since it was thought that a general psoric dyscrasia or a kind of "sharpness" of the blood—acrimonia sanguinis—or herpetic condition of the blood must exist in the individual when a skin disease was present. Even though a local agent, as the acarus in scabies or the fungus in favus,

was found later, this was not accepted as a cause by many physicians, not to speak of the laity, until a short time ago.

The psoric theory has lost its positive hold since the proof of a purely local cause for scabies has been established, which has also been proven clinically and experimentally for a number of other skin diseases, and since Hebra demonstrated in regard to scabies and eczema that they are producible in any individual.

Nevertheless we do recognize a great number of general and dyscrasic conditions and affections of the inner organs which directly or indirectly are the cause of skin diseases. Some of these causal factors are well known; others cannot at present be definitely defined. It is, in fact, a purely arbitrary assertion, again become prominent since Hebra's death, but resting upon no foundation, that we dermatologists disregard the constitutional and internal causes of skin diseases. On the contrary, our study is constantly directed toward recognizing and comprehending where possible the relationship which exists between diseases of the skin and other systems. But wherever this relationship is not proven or, according to the present state of our knowledge, is not intelligible, we prefer to consider concrete skin diseases as distinct pathological phenomena rather than to place hypothetical conditions in the foreground.

To the symptomatic forms of disease (*i.e.*, those occasioned by general causes) belong first the acute exanthemata produced by a *specific contagium*, as small-pox, measles, scarlatina; also rabies, syphilis, farcy, and pyæmia—processes in which, besides the specific blood-poisoning, a skin disease appears under the form of varying kinds of erythema, papules, vesicles, pustules, or erysipelatous and furuncular inflammations, these being necessary or essential symptoms of the disease. In other general diseases symptomatic skin affections are occasionally present: as in typhus are found macules, papules, and vesicles (typhous roseola, purpura, miliaria, and the typhoid exanthema of Dietel); in cholera, roseola and erythema (cholera exanthema); in uræmia and diabetes, pathological excretion from the skin (uridrosis), inflammation, gangrene, itching; in scorbutus, hæmorrhages; in scrofula and tuberculosis, inflammation and ulceration; in leprosy, macules, tubercles, anæsthesias, and ulcerations. Cancer dyscrasia, chlorosis, anæmia, leukæmia, and cholæmia have affections of the skin in connection with them—*e.g.*, abnormal coloring, itching, new tissue formation—or, as in chlorosis, create a disposition to certain skin diseases.

Among the *affections of single organs* which produce skin diseases may be enumerated chronic catarrh of the stomach and intestines; enlargement of the liver and spleen; all kinds of abnormalities and functional conditions, including even the physiological ones, of the female genital system; Bright's disease and albumin-

uria. All of these may occasion acne rosacea, pruritus cutaneus, urticaria acuta and chronica, anomalies of secretion and pigmentation of the skin, pemphigus, and impetigo herpetiformis. Heart diseases cause cyanosis and œdema of the skin. Tuberculosis of the lungs or other organs sometimes leads to like diseases of the general integument. Diseases of the peripheral nerves occasion herpes zoster and all kinds of atypical forms of inflammation, as well as disturbances of nutrition and sensation ; affections of the central nervous system—viz., of the spinal cord—cause zoster, pruritus cutaneus, and pityriasis rubra.

The vaso-motor nervous system is responsible for a number of skin diseases which have been known since Eulenberg's and Landois' time as angioneuroses—*e.g.*, urticaria, acne rosacea, and zoster. In this connection it is only to be mentioned that these affections are occasioned by an alteration in the vascular nerves, which have been experimentally shown to be vaso-dilators and vaso-constrictors for certain vascular areas, producing locally a dilatation or narrowing of the capillaries and finest vessels of the skin, and hence giving rise to local anæmia or to hyperæmia and exudation, and as a further result to essential nutritive disturbances of the tissues—trophoneuroses. The exciting causes of these conditions may, if affecting a limited area, be found in the ganglia situated along the vessels themselves, or in the spinal ganglia ; for extensive and universal affections they must be looked for in the medulla oblongata or in the vaso-motor centres in the cortex of the cerebrum ; and in either situation may be produced directly or by reflex action. To the first of the two last-named categories would belong hæmorrhages of the spinal ganglia and a zoster caused by them ; to the last a pemphigus occasioned by a reflex stimulus from a pregnant uterus. We shall return later to this subject, in the treatment of special pathology and in the chapter on "Nerves of the Skin."

Heredity is a factor in skin diseases, either directly as in syphilis, or by producing a predisposition as in ichthyosis, psoriasis, eczema, polytrichia, alopecia, cancer, nævus, and albinism.

Other conditions which must be considered as general factors in producing skin diseases are differences in age, sex, dwellings, conditions of nourishment and living, vocation, manner of living, and climatic and geographical conditions. For instance, eczema, seborrhœa, and urticaria occur most frequently in the nursing child and in the first year of life ; prurigo begins always toward the end of the first year ; psoriasis is present in vigorous middle life ; pruritus, epithelioma, and papilloma are met more frequently among persons more advanced in life. Lupus erythematosus is found more often among females than males. Lichen scrofulosus and prurigo prevail more frequently among children of the poor and poorly

nourished classes than among those more fortunately situated. Many diseases are more frequent in certain lands and localities than in others ; for example, prurigo is seen more often in our country than in England, while psoriasis is more common there than with us. Indeed, certain diseases exist only, or at least in a pronounced form, in certain zones and territories ; as, for instance, leprosy in particular districts in Norway, the Mediterranean coast, and the continents and islands of the South Sea. Certain articles of food and medicine produce in many persons reflex skin diseases either by acting upon the digestive tract or by irritation of the nerves of taste. Under this head belong urticaria from eating strawberries, oysters, lobsters, etc.; and the skin diseases which are caused by the internal use of copaiba balsam, iodine, bromine, quinine, opium, arsenic, and many other medicines, and which have become accurately known only during the last few years, and are grouped together under the name of *medicinal exanthemata.*

Although the general etiological factors that have been mentioned are so numerous and so positive, we are usually unable to demonstrate the direct relations between most of them and the skin diseases caused by them.

On the other hand, the etiological relations are exceptionally clear in the case of the *idiopathic diseases,* or those which are produced by the action of external injuries directly upon the skin. The change occasioned and the kind of disease which naturally results from these injuries correspond directly to the nature and mode of action of the latter. These agencies are divided, according to their nature, into mechanical, dynamical (caloric), chemical, or organisms (fungoid or parasitic) which directly infest the skin.

The action of the first of the three kinds of skin irritants, the *mechanical,* is easily understood. In every condition of practical and active life the skin experiences mechanical injuries which disturb the epidermis or the skin, even to its deepest layer, causing exudation of serum and blood, and inflammation and swelling. Frequent pressure from the constant handling of implements, or from bandages, girdles, and shoes, causes a thickening and swelling of the skin and organic changes of the corium and papillæ. Contusions cause a rupture of blood vessels and exudation of blood under the epidermis or into the cutis. Scratching with the finger nail is a most frequent cause of skin disease. We shall investigate later this method of cutaneous irritation.

Excessively high *temperature,* as that produced by the sun's rays and the radiation from the hearth and flames, as well as very low temperature, cause sometimes transient redness and desquamation, sometimes intense inflammation or charring.

Chemical agents which have an injurious influence are : all the

strong acids (muriatic, acetic, sulphuric, and nitric); caustics; a number of chemicals and dye stuffs, as aniline dyes; as well as plants and juices of plants which contain an active principle (mezereon, arnica, mustard seeds, croton oil); most of the ethereal oils and resins, as camphor; and all the so-called antiseptics (carbolic acid, corrosive sublimate, creolin, iodoform, etc.) which are brought into contact with the skin either accidentally or intentionally, as in the processes of manufacture or with a therapeutic view. They either destroy the epidermis or the deeper layers directly, or they act by exciting the skin to hyperæmia and inflammation.

These injurious agencies have a secondary effect, since they not only irritate the skin directly, but *weaken its power of resistance* as a whole against irritants, so that agents now irritate the skin which formerly were able to do no harm. For example, if any one, by means of an application of arnica to a wounded finger, produces an eczema there, then the skin of the face exposed to a a slight degree of heat from the fireplace, or the skin of the neck rubbed by a hard collar, will become eczematous, while formerly the rubbing of the latter and the heat of the former were well borne. Even a general nervous excitation may result from a local irritation. If an eczema results from the application of mercurial ointment to the pubic region, the vascular papillary system in the vicinity of the auricle and the face may undergo reflex irritation, so that it becomes hyperæmic, and exudation and eczema appear long before the eczema of the pubic region has spread beyond its boundaries. These relations are altogether too little known, for, if they were, physicians would not use cutaneous irritants so indiscriminately. There will be occasion later to further discuss this subject.

Many of the irritants mentioned are relative, in that one affects the skin quickly and intensely, another only after some time or not at all; in general the action being more quick and intense the more the skin has already been irritated or affected by disease.

Even water which is chemically inert produces skin diseases by its excessive application in the form of washes, packs, douches, and bandages, causing maceration of the epidermis, erythema, eczema, furuncles, etc.

On the other hand, uncleanliness and lack of care of the skin are the cause of many other skin diseases, which are produced by accumulation and decomposition of epidermis and cutaneous secretions, by the irritating effect of the latter upon the skin, by closure of the glandular excretory ducts, etc.

Vegetable and *animal organisms* produce skin diseases, as skilful research taught for the first time a few decades since. They sometimes live and vegetate in the skin, especially the epidermis—true parasites and epiphytes—and sometimes are only incidentally

found upon it—epizoa. They cause locally a loosening of the epidermis, hyperæmia, and exudation as a result of mechanical and chemcal irritation, or destruction of the papillæ, extravasation of blood, and accompanying inflammation, as in the case of many epizoa; they also act as irritating agents, producing in a reflex way many other skin diseases, as pruritus, urticaria, and eczema.

CLINICAL ASPECT OF SKIN DISEASES.

We have thus far discussed in a general way only the anatomical and physiological substratum of skin diseases, and, having regard to general pathology and pathological histology, have pointed out the changes which may affect the elements of the tissues and organs as well as the functions of the skin, have described the symptoms of the local changes that may take place in the skin and an outline sketch of their regular course, and have also presented the general causes of skin disease. But with all this we have not yet given a complete general presentation of a skin disease in a clinical sense—a thing which it is the particular object of dermatology to accomplish. For the complete conception of the clinical nature and form of a skin disease the character of its special course is most important—that is, its special origin, development, duration, mode of extension, local and general effects, and so-called sequelæ. These circumstances, combined with the others above mentioned—namely, the etiology and the local disturbances produced in the tissues and functions—give an idea and a picture of a skin disease, a skin lesion, as a complex of symptoms constituting a special form of disease.

A short example may illustrate this. Two children have each, on the extensor surface of the leg, an eruption of red, itching tubercles, marked with scratches and covered with crusts, upon a considerably thickened, somewhat œdematous, and darkly pigmented skin. The local change histologically and in its whole development and course is the same in both children, but the skin disease in a clinical sense is not the same. In one case this may be prurigo, a very obstinate affection, difficult or impossible to cure; in the other it may be a chronic but yet surely curable disease, an eczema. Many persons are affected with the form of disease known as urticaria wheals; in all the eruption is the same, involving anatomically the same changes and pursuing locally the same course, yet the clinical significance of the lesions may be different in all. In one the disease is an acute urticaria following the ingestion of strawberries, and will disappear in a few days; in a second, a child, it is the introduction to a life-long affection, prurigo; in a third, one symptom of a process endangering life, pemphigus pruriginosus; in a fourth, a sign of local irritation due to bugs; in a woman, a reflex phenomenon caused by a displacement of the uterus. The difference lies in the complex

formed by the sum total of the phenomena, among which those relating to the special course pursued are the distinctive ones. You will better comprehend the significance of what has been said the further you progress in special pathology; but you will have opportunities, in examining various patients, to distinguish—*i.e.*, to diagnose—similar forms of disease which clinically are quite different, and to determine the prognosis and therapy accordingly. This naturally leads me to give some general and profitable suggestions for practical work in regard to these last three points, and first in regard to the general diagnosis of skin diseases.

GENERAL DIAGNOSIS.

In illustration of what has been said I shall present a number of patients. Having in mind the practical purpose of our being together, I must point out the diagnoses of individual cases and arrange the corresponding therapy. With that we shall at once enter upon the territory of special pathology of skin diseases.

The different forms of disease make an unsatisfactory impression upon the mind of the inexperienced, in contrast with the precision and alertness with which the experienced teacher points out the individual case as being one of eczema, psoriasis, pemphigus, lupus, etc. So is it always with the young practitioner to whom such forms of disease are presented for the first time. They all appear to him confused, singular, kaleidoscopic, and in their multiplicity one and yet heterogeneous. It seems as if he should never succeed in unravelling the tangle of symptoms presented to his eyes. This cause for disquietude, which has at the same time something of discouragement for the beginner, will surely disappear in a short time when through practice you shall have learned to see and to differentiate. You will also be relieved immediately from this disquietude when you realize that the problem of diagnosis is quite different from what it is generally supposed to be, and that this problem is, as a rule, successfully solved by means of the proper method.

Many imagine that at the first glance at a case of skin disease they should be able to diagnosticate it immediately as an eczema, psoriasis, lupus, etc. This is an error; even the most experienced and skilful practitioner cannot always succeed in doing so. Even to a man like this, one or more cases occur yearly the like of which he has never seen before nor can find in literature, and which, therefore, are absolutely or comparatively unique; and yet such cases can be properly diagnosed, in the sense that the significance and essential nature of their separate symptoms can be properly estimated. The assignment of such a disease to its place in our classification follows naturally, being determined by the whole sum of diagnostic symptoms, and of the pathological changes, their location, course,

etc. The careful diagnostician attains this skill by following a certain method in diagnosis and passing over it step by step. He applies this rule even to the more usual cases, though in these practice enables him to accomplish his task more quickly, to a certain extent by leaps. The less experienced must pursue the same course slowly, and therefore it is necessary for him to know what method and line of thought he has to adopt for the purpose of diagnosis.

The first important step is to learn what the *history* of the affection may contribute. In private practice, where a certain respect must be shown to the personal wishes of the sick, it is impossible to cut short the remarks and complaints in which the patient indulges. These should, however, be directed into a channel bearing upon the subject—that is, one which will furnish information concerning the locality of the disease. Even then it will often be found that this information does not correspond with the facts. For example, a complaint is made and advice is sought on account of a disease of the face, no mention being made of the important and real affection because it is present in quite another portion of the body, where, in the judgment of the patient, it does not annoy him or is of no importance. Even greater are errors or misstatements of the history with reference to other important considerations connected with the disease, as its duration, origin, course, causes, etc., so that, as a rule, the history is disregarded in making the diagnosis. We have simply to accept it quietly and sympathetically, letting it pass without making any impression upon us, and never challenging it previous to the examination of the patient. This must begin with the examination of the actual objective appearance of the skin, and upon this objective condition alone should the diagnosis be founded. We stand, in relation to skin diseases, in the same position that we occupy in the study of natural sciences to the natural objects, plants, minerals, etc., whose nature we diagnose according to their objective, physical, and chemical peculiarities. The diagnosis of skin diseases must and can be objective only, and the verity of the history must be measured by it, not *vice versa*.

In order to attain a satisfactory result the *examination of the patient* must be carried out methodically and with an idea of what we intend to accomplish. With reference to the first requirement the following rule should be observed : The examination of the skin should be conducted by good daylight and in a moderately warm room. A large number of dermato-pathological symptoms will be expressed by difference in color, such as redness in all possible shades and pigmentation. Artificial light, as well as direct sunlight, detracts from our ability to perceive such appearances. The same holds true for too high or too low temperature, in which even normal spots on the skin may appear red, mottled, bluish red, or white.

Further, the examination must be, as a rule, continued over the whole surface, and not be limited to that portion which is designated as affected or which is offered for inspection. We have to be careful in doing this, especially in females, in whom we cover up again each portion of the body after it is examined, and look at the genitals only in urgent cases. With men, however, this caution is not required.

A general examination not only shows the general condition of the skin and the body of the patient, his state of nutrition, and special conditions, but it is advisable and indeed necessary for the clear conception of skin lesions, since important symptoms completing or giving a characteristic aspect to the whole picture are found out in this way and in contemplation can be grouped together as a whole.

First of all, the symptoms appreciable by the sense of *sight* must be considered. It is unskilful to touch immediately the affected spot with the finger; by so doing the redness of injection is displaced, the color and form of many eruptions are changed, a portion of the diseased territory is covered, the affected area is divided into many small fields, and the general view is interfered with.

The sense of *touch* comes next in order as an aid for the appreciation of temperature, of the consistence and the character of the surface of the skin, and of individual diseased portions; of the distinction of hyperæmic from hæmorrhagic and pigmented spots.

Some physicians say they can distinguish certain forms of disease, as measles, scarlatina, small-pox, etc., by the sense of *smell.* We do not place much reliance upon this sense as a means for the diagnosis of skin diseases.

We employ the *microscope* and *chemistry* as important, and sometimes decisive, scientific aids in diagnosis; the latter, in a rather limited way, for enlightenment upon the chemical relation of pathological forms and secretions, the former to obtain information regarding parasitic organisms and an insight into the histological relations of processes of disease.

The first purpose of the examination is not the systematic diagnosis of skin disease, but chiefly to gain an idea of the general condition of the skin and whether this is mainly normal or abnormal as regards color, injection, or pigmentation; the character of its pores, lines, and furrows; its tension, smoothness, and flexibility; its hairiness, oiliness, and thickness; the amount of adipose matter, etc. One notices whether these appearances, which within the normal vary more or less widely, correspond to or are foreign to the individual and his environment—his race, age, sex, business calling, and manner of living. Experience, as well as observations made upon the normal skin, must regulate the opinion in this respect.

After a general examination conducted in this way an abnormality of the skin may be discovered. Then it must be decided in what

form it exists: whether it is atypical, occurring as a diffuse, irregular redness, thickening, pigmentation, etc., or typical, occurring under the form of the so-called primary eruptions. Exact inspection and a clear perception of the obvious eruptions (as macules, papules, wheals, etc.) and of the secondary manifestations of disease (as scales, crusts, cicatrices, etc.) are of the greatest importance for diagnosis. Let no one think, however, that these morphological characters are alone sufficient for the diagnosis of the processes of disease, or that it is sufficient to have recognized correctly individual eruptions. It is, on the contrary, necessary to decide whether the efflorescence or the pathological change is situated in the epidermis or in the corium and subcutaneous tissues; whether it is associated with the appearances of an acute or chronic inflammation, or has more the character of new tissue formation; what course it takes—*i.e.*, whether it is attended or not with change of the skin, with exfoliation, and with ulcerating or cicatricial destruction of the skin; in what manner it spreads, whether in a typical way from a central point peripherally, or atypically; the relation of the eruptions, in their arrangement and extension, to one another or to certain regions of the skin, nerves, and blood vessels; and so on, proceeding always synthetically from the individual to the general. I shall not repeat here my remarks, under the head of general symptomatology, upon the characteristics of the forms of eruption, their location, their course, and the multiplicity of their anatomical significances. I must refer to what has been already said upon that subject. Here, in diagnosis, it is necessary to acquaint ourselves with the details of the conditions whose characters are there generally given.

Crusts of dried serum and blood or fat and epidermic scales lying upon the diseased area often render a research into the true condition difficult. The removal of these is necessary for a correct diagnosis. As it is not always possible to remove them, since the removal is attended with pain for the patient, it is better to defer the diagnosis until the removal shall have been effected by proper means (emollient fats or plasters and water), so that a free exposure is made of the actual primary symptoms of disease.

In this way one advances surely, step by step, toward a view of the whole course of the disease and to a systematic diagnosis. When all symptoms are grouped together a picture is formed, not of one individual efflorescence nor of several, but of a clinical whole. The shape, arrangement, mode of distribution, anatomical change, and, what cannot be sufficiently emphasized, the course of the eruptions—all these together give the picture of disease and a means of diagnosis, in the sense that we associate with the name of a special disease a certain group of symptoms combined in a manner which from experience we know to be typical of the eruptions. All these facts

are determined objectively—*i.e.*, without any contribution on the part of the patient. Even the subjective symptom of itching can be recognized objectively : " when one itches one scratches " is not only a saying but a scientific truth. The scratching finger shows lines upon the skin ; in the beginning these are white, but appear later as red streaks with slight exfoliation. In severe scratching there are excoriations which are bloody or covered with crusts. The longer the man scratches the more the older evidences of scratching are associated with the new, and the more intense the itching the deeper are the excoriations and the more these are complicated with signs of inflammation. From the mere appearance of the skin it can be diagnosed whether moderate or intense itching is present, whether it has existed for a long or a short time ; whether, on the other hand, a visible skin disease is an itching one, as eczema or scabies, or one that does not itch, as syphilis. Further diagnostic points are furnished by the locality of the scratches, since under certain diseased conditions only particular regions of the body, or, on the other hand, the entire skin, itch and are scratched.

These instructions are sufficient to serve as points for the general procedure in making a diagnosis. The special facts in the diagnosis of diseases will be treated of in their special pathology. It is hardly necessary to mention that a physician who understands his business, when called upon to diagnose a case of skin disease, gives attention to the condition and functions of the other organs and systems of the body—the heart, kidneys, etc., and, where pelvic disease exists, to the state of the genital system. He considers, that is, the fact, spoken of already under the head of general etiology, that many skin diseases are caused by or associated with diseases and conditions of the internal organs, not to mention all the possible complications of skin diseases and the pathological conditions of the whole organism or individual organs attendant upon the latter.

LECTURE VI.

COURSE, SIGNIFICANCE, EFFECTS, AND PROGNOSIS OF SKIN DISEASES —THERAPY—SYSTEMATIC CLASSIFICATION OF SKIN LESIONS.

DIRECTLY connected with the diagnosis of skin diseases is the scientific interest in their course, significance, and effect upon the portion of skin involved and the whole organism, as well as in their curability—facts which, as a whole, make up the *prognosis*. There is a very great multiplicity and diversity in these particulars. Many skin diseases run an acute *course* which is typical and definite; others, again, are chronic and last through life. Many may pursue either course. Some are constantly or occasionally, others never, accompanied by fever. There are some which can occur only once; others may appear repeatedly. Of some forms it can be said that they remain limited to one or to certain places on the skin, or that, on the contrary, they may be widespread generally over the skin. The duration and extent may be markedly limited by effective treatment. Several diseases, very different in their course, acute and chronic, may sometimes exist in the same individual—*i.e.*, syphilis, psoriasis, eczema, and variola.

Very different also are the *subjective* and *objective significance* of different skin lesions. Skin diseases are detrimental to the beauty of the person affected, and are therefore morally and practically very injurious, even when the lesion is pathologically quite innocent—*e.g.*, acne of the face. Moreover, the direct local and general *effects*, as well as their later results, are to be considered. Many skin diseases, even after lasting for years, produce no permanent change in the skin, and during their existence produce no injury beyond disfigurement, a sensation of tension, pain, associated febrile symptoms, and inconvenience in the performance of business and social functions. Other skin lesions are either occasionally or, from their nature, inevitably associated with contraction, degeneration, or purulent disintegration of the skin, and hence cause permanent changes or loss of tissue, producing in the region of the face destruction of the nose, the eyelids, the eyes themselves, and in the extremities, especially when complicated, lasting disfigurement or interference with the mobility of the joints, contractility, sensation, and many other functions.

Aside from the skin diseases which are etiologically connected with the pathological conditions of the vascular and lymphatic systems, the specific dyscrasiæ, or diseases of internal organs, and which, as referred to in the section upon general etiology, occur in indissoluble association with them, most skin diseases have no detrimental influence upon the general organism and its nutritive functions or upon the constitution. There are many which are borne through a whole lifetime without occasioning a remote or general disturbance; others again, however, exercise an evidently injurious effect upon the whole organism or upon individual organs and systems. In the latter case the most unfavorable general effect does not always bear a direct relation to the intensity of the pathological processes found on the skin. All the processes accompanied by extensive and intense exudation, purulent secretion, fever, or itching depress the organism by causing an appreciable loss of liquid, indigestion and loss of appetite, emaciation, sleeplessness, pain, or by causing the nervous irritation which gives rise to these symptoms. Some skin diseases—*e.g.*, lichen ruber and pityriasis—for whose existence, according to our present knowledge, no internal disease can be held responsible, appearing in an individual hitherto perfectly well, regularly lead in time to marasmus and tuberculosis, and others, like cancer and sarcoma, to specific cachexia which can frequently be traced to metastases. Finally, there are also skin affections, such as prurigo, chronic eczema, and lupus erythematosus, which can be borne by most men without any general bad effect, while with others they occasion Bright's disease, pneumonia with its possible sequelæ, or, through lymphangitis, erysipelas, caries, and complications of all kinds with a possible lethal outcome.

Besides understanding the conditions existing in the natural course and life history of skin diseases, it is most important to predict their relative *curability* or *incurability*. Many affections always heal according to the natural law of their course—some with, others without, leaving any local or general traces of their presence. No skin disease is absolutely incurable; and if we are not in a position to remove every kind of skin affection, still we accomplish it in many cases, and can in others, by removal or mitigation of some symptoms, either shorten their course (and in these, as in the comparatively incurable, make the condition more bearable for the patient) or retard the injurious and dangerous results of the lesions, thereby lengthening life. The prognosis accordingly depends, to a great extent, upon the treatment, the result of which in a large measure lies in the physician's hands. For this reason I must make some general remarks concerning methods and means in the therapy of skin diseases.

GENERAL THERAPY.

It is remarkable that the diseases of the skin, as regards the possibilities and indications for their treatment, are not yet classed, in the eyes of physicians and the laity, as are the diseases of other organs and systems. While in the case of the latter the indications for using the most speedily working remedies and methods are considered as self-evident, many physicians and laymen, even if they do not actually regard cutaneous affections as a *noli me tangere*, yet think that in the treatment of the latter a certain precaution must be observed so that no harm to the organism shall result from their cure. Physicians, especially the younger ones, must fight in practice against this opinion.

It is very suspicious that this opinion is expressed the loudest, as a rule, where the knowledge of and the ability to carry out the right treatment are wanting, and that in the course of time it has been silenced through weight of evidence where it before had been most asserted, as in the case of scabies. But the opinion is still prevalent enough to make it necessary for us to combat it.

This notion is associated with the ontological idea prevalent in former years, which regarded cutaneous diseases as due to the deposition of the material of disease or vicarious secretions and excretions of a physiological and pathological kind which have been discharged spontaneously on the surface under the form of skin eruptions, and that the internal organs must of necessity be affected when these disappear or "strike in." Such a revulsant effect was particularly feared from external remedies—salves, plasters, and tinctures.

Clearer insight has, however, deprived the ontological view of processes of disease of any basis in fact. We also know that a psoric or herpetic dyscrasia which can be defined in terms of some morbid material, an acrimonia sanguinis which can be expressed by a pathologico-chemical formula, does not exist. We know, again, that in secretions and products formed in cutaneous diseases no material of any sort foreign to the organism can be found, but only serum and formed elements, and indeed elements of every kind, but always having the same nature as those existing normally in the body; no "psoric" or "acrid" materials are found in the products or deposits of disease. Besides, it is physiologically untenable to assume that it would be possible for serum or tissue particles secreted and discharged on the skin surface to be driven back again into the organism. And, finally, it is known that both pathological histology and experiment have proved most cutaneous diseases to be purely local processes which develop in the tissues of the skin, and which, in a measure, can be at any time produced at will in one who is in perfect health.

It is hence believed that the admissibility of, and indication for, attempting the local treatment and the cure of cutaneous diseases is only a logical inference from the facts cited, and therefore, at least by physicians, cannot be doubted or opposed.

In fact, this opposition is made nowadays no longer on theoretical grounds, but because of certain actual facts. It is noticed that many cutaneous diseases—namely, those which consist materially of hyperæmic and acute or chronic inflammatory processes—and also many forms of neoplasms, and even scabies, when there is a concurrent acute febrile disease (pneumonia, typhus, erysipelas, and convulsions and diarrhœa in children, etc.), or during syncope, sudden collapse, and moribund conditions, tend to disappear, become fainter, or, according to their nature, vanish entirely; but, on the other hand, when the convalescence from these diseases takes place and the strength is again established they develop and increase anew.

These observations have given rise to the idea, even until now partially received, that under known circumstances a cutaneous disease, or a "psoric" agency contained in it, is actually driven back into the inner organs, brain, lungs, etc., and produces there the inflammation or the excitation leading to convulsions, and finally, through the *vis medicatrix naturæ*, is sent back again to the skin, by which is explained the disappearance of the febrile and other phenomena connected with the disease of the inner organs.

Sober observation has taught that the relation between these phenomena is entirely different; that in the cases mentioned the febrile pneumonia, the symptoms of typhus, etc., were already existing, and the skin disease disappeared during the course of these affections; that therefore the retrogression of the cutaneous disease made its appearance first in consequence of these other diseases, and did not precede them; that hence it could not appear as a cause of the series of symptoms, but rather as the result of the internal diseases.

This retrogression, moreover, can in some, if not in all, cases be explained as follows: It is conceivable that when the skin becomes suddenly anæmic, as in syncope and in collapse, the injection-redness and infiltration which belong to the psoriasis disappear; or that the itch mites in the skin of a febrile patient subjected to abnormal heat and abnormal circulatory and nutritive relations thrive less and even become entirely extinct; and that psoriasis appears again or the eggs of the itch mites are developed as soon as, after the termination of the anæmia or of the febrile disease, the turgescence and the nutrition of the skin again become normal and therefore favorable for the production of those processes and the life of these animals. To all these data, which have proved that the

idea of "retrogression," "repulsion," and "driving back" of skin diseases is scientifically inconceivable and inadmissible, may be added the fact that in a hundred thousand cases of cutaneous diseases of all sorts which have been treated and cured by the methods and means of the Vienna school, no harm has resulted to the constitution. We are therefore prepared to combat and overcome this reprehensible opinion, from whichever side it may attack our plan of therapeutics. After we have cured a child of seborrhœa of the head, or an old person of eczema, we cannot always prevent the possibility of some other disease appearing at the time of the decadence of the skin disease, such as convulsions in the child or œdema of the lungs in the old person. But, fortunately, such a combination occurs very seldom, and criticisms of such accidents should be ignored by a physician, just as he should the well-known imputation that an unfortunate termination of a pneumonia could have been averted had mistura oleosa been used instead of decoctum althææ. If the physician makes this line of thought his own he will never lose the "courage of his responsibilities," which in the successful treatment and therapy of cutaneous diseases, as well as in practical surgery, is absolutely necessary.

MEANS AND METHODS OF TREATMENT.

It will now be useful, in order to avoid repetition later, to become acquainted with the *means* and *methods* in general which in practice are applied in the therapeutics of cutaneous diseases.

The remedies here considered are especially those which are called "external"—*i.e.*, which are applied directly to the diseased places—only a few being efficacious internally.

We are indebted to the first—*i.e.*, external remedies and local treatment—for very reliable and brilliant cures. In order to make use of these remedies physicians must first be acquainted with the particular conditions upon which the success or failure of their application depends.

It is a daily experience that good and versatile physicians find their greatest difficulty with cutaneous diseases, and find themselves unable to cure a common skin disease—*e.g.*, an eczema—notwithstanding they are well acquainted with and make use of the remedies with which others obtain success.

This depends, in the first place, upon a mistaken supposition, of which enough cannot be said by way of caution.

It is the opinion of many physicians that the chief thing they have to do is to make a systematic diagnosis of a skin affection. If this is done, and is perhaps successful, eczema being decided upon, the physician believes he has nothing else to do but to consult an elementary manual or a compendium of cutaneous diseases and

discover what remedy is recommended for eczema—diachylon salve or tar—and then to simply use these remedies and the disease must be cured.

But we have only a few remedies which are efficacious against the morbid process itself, and which, therefore, are applied after a kind of general rule, and, in the cases in which they are indicated, are successful—*e.g.*, arsenic for lichen ruber, cod-liver oil for lichen scrofulosorum, sulphur for prurigo. As for the rest, we know only of methods which can ameliorate and obviate single symptoms, and treatment must therefore aim to secure this object, it being immaterial to which disease the one or the other symptoms belong. With the relief of the separate symptoms the affection *eo ipso* can be obviated, because the disease consists of the sum of the symptoms.

Furthermore, it must not be overlooked that a single morbid process in the different stages of its course presents very different symptoms. For example, the phenomena of eczema are at one time simply redness and desquamation, at another time blebs, inflammation, and swelling, in a third stage moisture and a crust formation. Now, the remedy which would act upon the first-mentioned symptom would cure the eczema also. But the same means applied in the second stage of the same disease would not only do no good. but would do harm, since it would increase the inflammation. The fact that the same process at the same time presents different symptoms in the different diseased places of the skin has a similar significance. The treatment which, because the disease is everywhere the same, as far as classification is concerned, would use the same remedy for all the different symptoms presented by the different diseased places, would be most inefficacious.

We must therefore clearly comprehend and attach the proper value to the existing symptoms with respect to each separate portion of the skin, and, without regard to the general process, must adapt the means and methods of the treatment to them, and the medication must be changed as soon as the phenomena are essentially or gradually changed.

To be prepared for such a task demands great and constant attention and practice in scientific work.

Since we see in the symptoms occurring at a given time the essential indication for the treatment for that time, we are to a certain extent able to treat judiciously cutaneous diseases, the scientific diagnosis of which we cannot make perfectly clear at the instant. We depend upon the visible morbid changes, endeavor to obviate them, are able to give the patient much relief and often to bring about a cure.

But the success of the treatment does not depend only upon the judicious choice of the remedy for the single symptoms and the

special diseased places. This remedy must also be applied in such a way that the result desired locally shall certainly be obtained. In this condition it happens that one physician will obtain a successful cure in twenty-four hours with common olive oil and soap or a simple salve, while with another it cannot be obtained in the same number of days. The approved method then is to be considered well and not to be regarded lightly.

Finally, we must not forget that the successful remedies, applied correctly and at the right place, exercise not only a healing influence upon the symptoms present, but are also able, on account of their physical and chemical properties, to exert a distinct physiological action upon the healthy as well as the diseased skin (an action which takes on the guise of disease), and may also, according to their special nature, power of absorption, and their specific properties, act, quite contrary to our intention, harmfully, or even fatally, upon the kidneys, the heart, and the central nervous system.

As is known, caustics often, contrary to our intention, destroy the healthy skin near the diseased places. Substances which at other times are indifferent or innocuous, such as oils, cod-liver oil, or spring water, when they are used upon the diseased skin can increase or unfavorably change the symptoms and also affect the healthy part of the skin. Hence in the use of medicines all their possible effects are to be considered, and from this consideration it must be decided whether we shall proceed with them.

But all this is conditional upon two different things: first, that the effect which the various remedial bodies exert upon the healthy and diseased skin should be known as accurately as possible; second, that in every phase of the treatment there should be considered systematically beforehand the local effect which it is desired to obtain by means of the medicine, and to lay aside the latter immediately as soon as that effect has been obtained. Every skin is not equally vulnerable or reactive in relation to external injuries and medicinal preparations. If, for example, experience teaches that soft soap must be rubbed twelve times into the skin to produce a general exfoliation of the epidermis, and after four rubbings the skin of the patient appears red and œdematous, it would be very injurious to finish the eight rubbings. The process by which the epidermis has become rubbed off was obtained with four rubbings. To continue the rubbing would be to overstep the desired object and to inflame the skin. Since such a condition might even be dangerous to life, it is clear that through ignorance of the proper method the harmless soft soap may be changed into a dangerously poisonous remedy.

Another very valuable lesson can be deduced from these data.

It is of little importance to collect carefully many prescriptions for the treatment of cutaneous diseases and to store them up in the memory, because the medicine prepared according to these prescriptions often accomplishes nothing in one case, and in another everything. For the successful treatment of skin diseases the following rules only are important:

First, to correctly diagnose the separate symptoms of the disease in each diseased spot and in each phase of the disease.

Second, to determine definitely and to know the alteration which must be produced in these symptoms in order for them to be cured.

Third, to know the different medicines, and the methods of their application, through which such changes can be obtained.

One of the special remedies in the *local* or *external treatment* of cutaneous diseases is water, both cold and hot, pure or medicated with sulphurated potassa or lime, soda, alum, tannin, sublimate, common salt, lead acetate, acetate of aluminum, etc. ; and applied in the form of movable tub baths, douches, steam baths, moist local and general compresses or packs (Priessnitz). Generally, when there is no special object to gain by the use of especially low or high temperature, the warm bath is tempered to suit the comfort of the patient.

The duration of the tub bath is regulated according to popular custom, but may be protracted many hours and days, or even many weeks and months. The last, "continuous" baths, were introduced by Hebra, and were made possible by a bathing contrivance invented by himself, a kind of water bed, the curative effect of, and indications for, which will be treated in detail in the chapter on "Burns."

Generally the effect of water is to soften, to macerate the epidermis, and to loosen up the diseased products, such as scales and crusts, deposited upon the latter. Cold water especially, but also warm and hot, lessens inflammation, as in case of boils, dermatitis, erysipelas, and phlegmon ; but with long contact it again irritates the skin and produces eczema. Therefore it is applied especially as a macerating remedy for the softening and detaching of the scales and crusts, also as a vehicle for the medicated preparations and for the application of soap, as a protective covering for the extensive loss of epidermis (burns, pemphigus), and as an antiphlogistic.

Macerating in effect, in part also specifically healing (in prurigo, pruritus, psoriasis, etc.), are the rubber bandages invented by Colson (1869), Hardy, and Hebra, made from vulcanized rubber ; or articles of clothing manufactured of rubber and rubber linen—finger cots, gloves, jackets, trousers, also caps and face masks (Besnier). Laid

air-tight upon the skin they prevent the evaporation of the perspiration, the latter being precipitated in drops and acting as a macerating agent.

The application of *fats* of all kinds is useful for the softening of the accumulated morbid products. These fats include *olive oil, cod-liver oil, lard, glycerin, petroleum, balsam of Peru, and vaseline.* The last, which is a petroleum product, is a jelly-like substance, yellowish, transparent, easy to rub in and readily liquefiable, tasteless and odorless, soft and flexible, and incapable of producing fatty acids, since it is a mixture of the higher paraffins. With these is also included *lanolin,* a mixture of the chlolesterin ethers of the different fatty acids, which is recommended and prepared by Liebreich, and which is a regular ingredient of horny substances (sheep's wool, horses' hoofs, birds' feathers, and epidermis). It consists of a yellowish-brown, thick, glutinous, greasy substance, which for the practical application and reception of medicinal preparations should be mixed with five to ten per cent of glycerin or fat. When rubbed upon the skin it is rapidly absorbed and makes the skin pliant.

Recently *mollin* has been recommended, a potash soap made of cocoanut oil and tallow, to which a firm consistence is given by the addition of a little soda soap.

H. von Hebra recommends *glycerinum saponatum,* or chemically pure soap to which an ointment-like consistence is given by the addition of eight to fourteen per cent of neutral grain soap.

A greasy vehicle and recipient for all kinds of medicines has recently been made by S. Kahn, *epidermin,* which is made of white wax, glycerin, and water. In somewhat similar manner Schleich's *wax paste* is composed. Paschkis, under the name of *lanolin ointment,* has recommended the following formula : lanol. 66.0, paraff. liq. 6.0, ceresin 1.0, aquæ 65.0.

Recently the fat of wool, *œsypum,* known to the ancients and commented upon by Ovid on account of its disagreeable smell, has been used therapeutically. From every consideration it is dispensable.

English and American authorities (Shoemaker) recommend oleates, which according to their base (zinc, bismuth, copper, silver, etc.) act in an exsiccant, antiphlogistic, or stimulating and macerating manner.

R. Schröter several years ago warmly recommended a product prepared from the distillation of the remains of fossil fish found in Seefeld in the Tyrol, called at first *ichthyol,* and now, from the analysis of Baumann and Schotten, called by Unna sodium sulphoichthyolate. This preparation presents an oily, yellowish-brown, highly disagreeably smelling fluid. It mixes easily with water and

fat and holds sulphur up to ten per cent in chemical combination. Its chemical constitution is not perfectly demonstrated. Ichthyosulphate of ammonium, as well as other derivatives of it, has a therapeutic use in the treatment of eczema and in the most various cutaneous diseases of an inflammable and neoplastic nature.

Tumenol, recommended by Neisser, is obtained from the residues of certain mineral oils by treatment with sulphuric acid. *Thiol*, which is chemically closely allied to it, has lately been recommended as a substitute. This can be used in a two to five per cent solution in water, ointment, and oil, and after several applications causes a brownish drying-up and shrivelling of the epidermis, and from continual application redness and inflammation of the skin.

Fats, as such, or with quicksilver, sublimate, white precipitate, acetate of copper, sulphate of copper, lead acetate, iodine, iodoform, sulphur, tar, carbolic acid, salicylic acid, chrysarobin, pyrogallic acid, naphthol, opium, cocaine, etc., rubbed or boiled to an ointment or spread upon linen, are applied to the skin by means of flannel bandages.

Endeavors have been made to introduce glue (gelatin, Auspitz, Pick, Unna), varnish, and kinds of gum (gum tragacanth) as the foundation of ointments instead of fatty substances. To the gummy preparations belongs the linimentum exsiccans, introduced into therapy by Pick, and prepared from five parts of gum tragacanth and two parts of glycerin to one hundred parts of water.

Among *plasters*, which are the metallic salts of the higher fatty acids, mercurial, lead, and soap plasters are used.

The very adhesive emplastrum diachylon compositum (adhæsivum), as well as the various plasters containing rubber, including the so-called American or Unna-Beyersdorf plasters, and the Turinsky collemplastrum—all of which, like the emplastrum saponatum, can be mixed with salicylic acid, iodoform, quicksilver, etc., in varying proportions—can be used with much benefit for their special and their macerating effects.

Soaps are also of considerable consequence. Chemically they are the salts of the higher fatty acids, especially of palmitic, stearic, and oleic acids. According as potash or soda lye is taken for the saponification of fat, soft potash soap (sapo viridis, soft soap) or hard soda soap is obtained. For this purpose various fats of animal or vegetable origin can be used.

If all the alkali in the soap is united with the fatty acid the soap is neutral. For toilet soaps which are in daily use this state is indispensable. In other cases, however, the presence of a certain amount of free alkali is necessary in order to answer certain therapeutic indications.

The purifying action of soap is not yet understood in all its details. This depends upon the following conditions:

1. Upon the mild macerating action of the alkali salts of the fatty acids, or sometimes upon the much stronger action of the alkali that may be mixed with it or be set free.[1]

2. Upon the property of many soaps of making a lather, which acts mechanically in the process of washing.

3. Upon the ability of the soap to form emulsions with fat.

If for therapeutic ends it is desired to obtain a macerating effect with the soap, such soap should be used, as has been mentioned, as contains a certain amount of free alkali. The principal representative of this class of soap is the already mentioned sapo viridis (soft soap), a jelly-like potash soap smelling of whale oil, which, for the treatment of cutaneous diseases, we prefer to the hard soda soap. This is very useful for softening and separating the thick layer of horny epidermis, as in ichthyosis, callous thickening, psoriasis ; or when the detachment of the entire uppermost layers of epidermis is desired, as in herpes tonsurans maculosus, pityriasis versicolor, and scabies, in which case at the same time the whole brood of itch mites will be killed. The duration of the application must be suited to the object desired. By improper use pronounced dermatitis, and even ulceration, will be caused.

The form of the application is, according to circumstances, the ordinary one in which soap and water are used for the maceration and washing away of fat or for softening the scales and crusts ; or the soap is spread upon the skin like an ointment, in which case it causes the casting-off of the epidermis ; or it is spread upon flannel laid upon the skin, in which case it causes both maceration and deep cauterization.

Recently Unna, in order to decrease the action of the alkali in the soap, has recommended those which, besides fatty acid salts (of which neutral soaps exclusively are made), contain also a certain amount of unsaponified fat. The same part is played in the long since introduced glycerin soaps by glycerin, which is added up to forty per cent to neutral soaps. Glycerin used in this way with a large quantity of water cannot develop its otherwise drying property, since in a state of dilution it is an excellent skin fat.

As the ordinary neutral soaps are the alkali salts of the fatty acids, so the resin soaps are the alkali salts of the resin acids. These soaps, formerly used only for technical purposes, were recently introduced into therapy, owing to the fact that various technically

[1] The fact comes under observation that soaps, in the presence of a free fatty acid (from the secretion of the skin), have a tendency to become decomposed into a fatty acid and free alkali.

inapplicable hydrocarbons of the benzene series, when held in suspension by means of them, have been introduced into practice as antiseptics. Such a preparation is *creolin*. In *lysol* these hydrocarbons are suspended through the alkali salts of the different cresols. Its value, on account of the variability of the preparations, is uncertain.

For therapeutic purposes we use, besides the various kinds of toilet soaps, spiritus saponis kalinus, derived after Hebra's method from the cooking and filtration of sapo viridis with half its weight of rectified alcohol; Sarg's liquid glycerin soap, which is likewise a potash soap; also manufactured soaps containing a certain amount of medicated preparations, like sulphur soap, pumice soap (containing powdered pumice stone or talc), tar soap, and soaps containing tar and sulphur, iodine with sulphur, carbolic acid, naphthol, naphthol with sulphur, etc. Besides these there are soaps in a great number of combinations, according to taste and need, from which medicinal action can be expected.

An important part is always played in our Pharmacopœia by *tar* (oleum empyreumaticum), which is obtained from several kinds of wood by dry distillation. We use tar from the beech (oleum fagi), from the birch (oleum rusci), from the Juniperus oxycedrus (oleum cadinum), as well as ethereal alcoholic solutions of tar, tinctura rusci, prepared after Hebra's method, of the special properties of which we shall speak further in therapeutics. Resineon, a product of distillation from tar, was for a long time in use. Phenol, or carbolic acid, another chemical product obtained from tar, we use in the same way as tar and also as a caustic.

A number of chemically allied bodies—benzoic acid, benzoin, salicylic acid, resorcin (Andeer), chrysarobin (Balmanno Squire), anthrarobin (Liebermann), sozo-iodol preparations, aristol, europhen (containing a large proportion of iodine), dermatol (a basic gallate of bismuth), pyrogallic acid (Jarisch), and naphthol (introduced by me into therapy by the advice of Ludwig)—as well as the remedies belonging to another chemical class, have sometimes a very pronounced therapeutic effect.

Alcohols and ethers (including sulphuric and petroleum ether, chloroform and ethyl alcohol), opium, cocaine, menthol, etc., partly alone, partly in combination with substances in solution with them, serve as *anodynes* against pain and itching.

Rice starch, wheat starch, orris-root powder, pulverized talc, pulverized asbestos, kaolin, and red and white bole are used as *sprinkling powders*, either pure or with zinc, bismuth oxide, and in various combinations which, although they present an indifferent means, yet in the treatment of cutaneous diseases accomplish an important service. With certain forms of disease an indifferent method of treatment is the only judicious one; but to carry out even such a

method something positive must be accomplished as regards remedies and methods of treatment.

Another class of medicines serve as *caustics* for the purpose of destroying inflammatory or neoplastic pathological formations deposited in the skin, or, when applied less vigorously, serve to produce an artificial stimulation and inflammation. These are the vegetable and animal acids—acetic, muriatic, sulphuric, nitric, carbolic, salicylic, pyrogallic, and lactic acids; the alkalies—ammonia, caustic potash, and lime; also chloride of zinc, chloride of antimony, silver nitrate, potassa, Vienna paste, Landolfi's and Canquoin's pastes, Labarraque's and Plenck's solutions, and pulvis Cosmi, concerning the constitution of which and their special manner of working and indication we will speak later.

With these would also be classed galvano-cautery, electrolysis, and electrization.

For *internal use* in certain cutaneous diseases the following have proved efficacious: arsenic, mercury, iodine, iodoform, iodol, cod-liver oil, preparations of tar, quinine, carbolic acid, Zittmann's decoction, pilocarpine, atropine; while other medicines which are used only for improving nutrition and the condition of the blood, or which are designed for specific and general diseases or those affecting separate organs, are administered with the view of reinforcing to a certain degree the local treatment and of preventing relapses. Such remedies are bitters, iron, and ergot, the alkaline, arsenical, and ferruginous mineral waters, bromide of potassium, chloral hydrate, narcotics and hypnotics, also the milk and whey cure, special dietetic remedies, etc., for the ordering of which, with regard to individual conditions, there are many opportunities in the therapeutics of dermatology. In spite of opinions loudly expressed, we make the widest use in the therapeutics of dermatology of these and other so-called internal remedies. But we distinguish between the direct and undoubted curative action of a properly conducted local treatment and the indirect or hypothetical effect of certain internal medicines, and we do not consider it correct to neglect the former for the latter.

On the other hand, the various decoctions of woods, recommended as so-called blood-purifying remedies for eruptions and the predisposition to them, have not the least influence upon them.

Just as little do we find in cutaneous diseases, as such, a reason for forbidding the use of sour, salt, and seasoned meats, fish, alcoholic drinks, etc., since these nutritive remedies can have no influence upon the origin and cause of cutaneous diseases.

CLASSIFICATION OF CUTANEOUS DISEASES.

Thus far we have made ourselves acquainted with a considerable sum of facts concerning cutaneous diseases in general, and we have

so far acquired correct notions in regard to their anatomical disposition, their pathological signification, clinical symptoms and causes, and the methods and means of diagnosing and treating them, that we are now able to enter the fertile domain of special dermato-pathology, so that we may learn to recognize the separate clinical forms of disease.

There is an important question of form to be decided, namely, of the methods which we are to pursue and the classification which we are to adopt in treating of skin diseases.

We know from the historical development of dermatology that the need of a judicious division of skin diseases, which are so very manifold in their form, anatomical basis, causes, manner of progress, etc., has been strongly felt; that at all times attention has been given especially to this point; that in the course of time numerous simple or highly complicated systems of cutaneous diseases have appeared; and that finally we can show nothing to-day which answers in every way the demands of pathology and practical utility.

It has also been shown what principles prevail in the various systems of classification, although it has scarcely ever been possible to carry these principles out swiftly.

It is worthy of notice that since Plenck's unsuccessful attempt at dividing cutaneous diseases only in accordance with the characteristics of their external phenomena, as maculæ, papulæ, bullæ, crustæ, etc., the efforts of most authors who rank as clinical masters in cutaneous diseases have been toward bringing into one harmonious system the anatomical characteristics and the so-called "natural ones"—that is to say, all the terms expressive of the causes, the anatomical and pathological manner of progress, and the rank of dermopathies.

In the execution of this design several authors have fallen into extremes, since they give the first place exclusively to the anatomical conditions. So Erasmus Wilson, who at one time divided cutaneous diseases into those of the epidermis, the rete, the follicles, the vessels, the nerves—a system which must prove most unnatural, since in reality these anatomical structures are very seldom diseased separately. On the other hand, other writers, as Alibert, have considered "naturalness," in the grouping especially, by which the positive scientific basis seems once more entirely lost.

A not improper division is that in which the anatomico-physiological relations are used as a basis, for this is scientific and allows at the same time the grouping of cutaneous diseases according to certain natural groups. Bärensprung has formulated these most precisely, since he divides cutaneous diseases into (1) disturbances of innervation, (2) disturbances of secretion, (3) disturbances of nutrition.

The systematization of cutaneous diseases according to their fundamental pathologico-anatomical characteristics, attempted partly by Rayer, is, as has previously been stated, the work of Hebra. When it is said that this system of Hebra's is founded upon a pathologico-anatomical basis, it will not be necessary to repeat that the system is not a purely anatomical one, as has often been erroneously stated. For pathologico-anatomical. even called clinical-anatomical, is the expression which is limited by pathological processes and the anatomical changes leading to these. In the happy combination of both to one principle of unity lie the character and the excellence of Hebra's system, and upon it rests the possibility of modifying in a measure the progress of pathological and anatomical science without changing its principles.

The author of the system has himself shown that it contains many defects and stands in need of improvement. But up to the present it seems, as a rule, better than all other known systems to answer to the demands of a scientific and practically useful classification of cutaneous diseases. Therefore most of the recent writers on cutaneous diseases accept this system wholly or with slight modifications and also as a foundation for other systems.

We mention next the nominally new systems of cutaneous diseases which have been offered by Neumann, Duhring, the American Dermatological Society, and also by Bulkley; for these are essentially confined to a contraction and a partial transposition of the groups in Hebra's system—changes for which each system expects free scope up to a certain degree.

Auspitz has taken a more radical stand in his system of cutaneous diseases (1881), in which, after criticising the systems in question, and especially that of Hebra, he produces a new system of cutaneous diseases. The attempt to regulate cutaneous diseases upon a neuro-pathological basis finds expression here for the first time. Whether our time is adapted for this yet we will not decide. But it is clear to us that the system offered by Auspitz, in its leading principles, is full of those deficiencies for which the author criticises Hebra's system so bitterly; and also that it is much less practical than the latter, for the reason that it, often upon a highly hypothetical basis of classification, separates processes naturally belonging together and brings into close connection unrelated forms of disease.

Since that time other noteworthy attempts have been made to create a new system of cutaneous diseases which would fulfil all the demands of science and practice. Schwimmer has introduced a new system on the basis of the "neuro-pathological" nature of many cutaneous diseases, without being able to sustain it upon this basis, since only a few skin diseases rest upon such a foundation, with many such an inference is either entirely hypothetically or erro-

neously asserted, and even, when treating it partially, the nature of the process is not exhausted; while Hans Hebra has tried to bring Auspitz's system into practical use, without being able to leave that system unharmed. Hyde's system, which seems to be modelled after the "anatomical" system of Wilson, the classification of Hillairet and of Berlioz, are no more worthy of imitation than that systemless system with which cutaneous diseases are treated in Ziemssen's "Handbook."

So we hold to Hebra's system of skin diseases, which, regarded in every light, seems to us, in the present state of our knowledge, to have the advantage of combining simplicity with exactness, in that it considers particularly the clinical impression of the skin changes which are presented to our notice. But this is neither the exclusive effect of an especial clinical factor nor of a pure anatomical deviation or alteration of function, but the expression of the working together of the sum of such agencies, among which the real course of the illness is classified according to the most obscure, but generally according to the characteristic and most pronounced, eruption.

According to this conception of the diseased processes, the different cutaneous diseases are differentiated according to their especially conspicuous characteristics, which are at one time anatomical and at another physiological or etiological, many of which again belong in so-called natural groups, according to the sum of their natural characteristics. The easy investigation, the sure progress, and the firm hold which this system of Hebra's offers above all others, both in the study and at the sick-bed, guarantees from every possible condition a systematic and varied grouping of the different forms of skin diseases, and merits the esteem of students and practising physicians. According to it cutaneous diseases are divided into twelve classes:

1. Hyperæmiæ cutaneæ—diseases consisting of hyperæmia.
2. Anæmiæ cutaneæ.
3. Anomaliæ secretionis cutaneæ et glandularum cutanearum—skin affections which consist in functional and nutritive disturbances of the sweat and sebaceous glands, or diseases arising from them.
4. Dermatoses inflammatoriæ—inflammation of the skin. In this class belong the largest number of the forms of cutaneous disease, according to their causes, courses, morphological properties, and accompanying characteristics. They are divided into four natural groups.
5. Hæmorrhagiæ cutaneæ—that form of disease consisting of extravasation of blood.
6. Hypertrophiæ, including the skin diseases which consist ana-

tomically of hyperplasia of the whole or single parts of the tissue of the skin.

7. Atrophiæ.

8 and 9. Neoplasmata, and sometimes (8) the clinically innocent and (9) the malignant, which form new growths of tissue. With reference to single cases of the last two classes of cutaneous disease, later researches in micro-organisms have brought forth new views concerning their causes and pathological significance. The further development of these upon the basis of newer and richer facts should in time make possible a more natural grouping of the forms of disease belonging together, without losing the entire contents of this double class and its clinical and therefore its systematic dependence.

10. Ulcerationes—ulcers.

11. Neuroses—cutaneous diseases which, without visible changes in the tissue of the skin, consist purely of a functional disturbance of the skin nerves.

12. Dermatoses parasitariæ, which are conditional upon plant or animal parasites and those cutaneous diseases associated with them in their symptoms. The classes which are arranged according to this system, but signifying in no way groups of disease, are pathologically sharply distinguished from one another. As is known, there are no such demarcations in the nutritive processes, since all the possible changes and transitions from hyperæmia to inflammation, and from this to neoplasm, hyperplasia, gangrene, and atrophy, are not to be defined. But in their clinical characteristics the important functions are very distinctly distinguished from one another, and on this account the above division, as it bears upon these points, is scientific and natural and at the same time practical.

Yet cutaneous diseases, according to boundaries defined by pathological and anatomical characteristics, are separated naturally into special groups. For those characteristics determine, as is already evident from general symptomatology, the essential part of clinical phenomena. But the natural connection is determined by a sum of the symptoms, which, owing to different stages, courses, and peculiar conditions, are of various kinds. According to the demands of these natural connections, cutaneous diseases may be subdivided, from the wide range of the separate classes, into smaller groups naturally and abstractly made.

From a clinical standpoint I do not consider it correct to place hyperæmia in a special group and leave out the inflammatory processes, or to overlook exudation as a special distinctly marked characteristic of a series of diseases, or to ignore anæmia, or not to treat ulceration separately. But I say that a grouping of the single forms of diseases more conformable with the clinical and anatomical

knowledge of recent times would be possible. Corresponding changes in our system will then be met with and will bring us more success. But as for the principal changes from Hebra's system, I think it will be a long time before our general pathology will become fundamentally different.

Now we pass on to the discussion of the special pathology of cutaneous diseases.

SPECIAL CONSIDERATIONS.

CLASS I.

HYPERÆMIÆ CUTANEÆ—CUTANEOUS DISEASE INDUCED BY EXCESS OF BLOOD IN THE SUPERFICIAL CUTANEOUS LAYERS.

LECTURE VII.

HYPERÆMIA OF THE SKIN, ACTIVE AND PASSIVE—IDIOPATHIC AND SYMPTOMATIC HYPERÆMIÆ—ROSEOLA—ERYTHEMA—ANÆMIA OF THE SKIN.

GENERAL CHARACTERISTICS.

UNDER cutaneous hyperæmia we include those forms of disease which, though dissimilar in their clinical relation (*i.e.*, appearance, course, and signification), yet in their general anatomical character are distinguished by the *excessive amount of blood in the most superficial layer of the corium next to the papillary stratum, which is the cause of their phenomena.* It may also be said that it depends solely upon the *injection of the finest vessels, the capillaries and finest arteries and veins.* As soon as, in the course of such a hyperæmia, visible changes of tissue have occurred, the progress of the disease can no longer be included in the category here given.

From general pathology it appears that hyperæmia forms the first step of most nutritive disturbances, especially of inflammation, suppuration, gangrene, hyperplasia, and neoplasm; and that in all cases, whether clinical or histological, there is a sharp line between hyperæmia and the other processes mentioned, whereas, on the contrary, a transition takes place gradually between the first and the last.

It might appear that the systematic presentation of a group of diseases characterized by hyperæmia only would not be correct.

From the practical study of the patient this result appears. Pro-

cesses also belong here which may be developed from the hyperæmic condition to the higher degrees of nutritive disturbance, but which very often stop at this first stage and therefore should be included in it. Other processes are also included which typically do not reach the stage of hyperæmia. Under certain circumstances, with a longer duration of the hyperæmic condition in consequence of a locally increased supply of nourishment, and under certain local conditions which are favorable to hyperæmia—*e.g.*, change of the vascular wall, the extravasation of the coloring matter of the blood and blood plasma, etc.—a visible alteration of tissue makes its appearance. But it must be understood that by the limitation of the forms of disease we have in mind typical groups of symptoms which are average clinical pictures.

Cutaneous diseases consisting of hyperæmia show the following symptoms: blotches, from a lentil to a finger nail in size, situated on a level with the skin or slightly elevated, which are pale pink, vivid blood-red, or dark bluish-red, cyanotic, growing pale under pressure of the finger, uniformly tinged, speckled, or crossed with vascular ramifications (maculæ, roseolæ); or of greater extent, diffuse (erythema congestivum), of an active redness and irregular form. The temperature of the skin at these spots is normal or slightly, even considerably, increased. They are smooth to the touch, greasy like the normal skin, or a trifle firm. Slight burning and itching or other disturbances of sensation accompany their continuance or may be absent entirely. Their course is acute, also cyclic, or very transient (erythema volatile, fugax), or chronic, many times persistent.

They consist of excessive blood injections of the finest vessels of the papillary layer, or also of the upper layer of the corium, and sometimes of the vascular network surrounding the excretory ducts of the follicles. If their course is a limited one they disappear without a trace, or after a short time they are followed by deeper pigmentation or considerable desquamation of the epidermis. Often they cause increased secretion from the sweat and sebaceous glands. Hyperæmia of longer duration leads to œdema of the skin, which, like acute hyperæmia, causes, through occasional increase of the local processes, inflammation, thickening and degeneration of the tissue.

Since in the process of death the skin vessels empty their contents into those of the inner organs, other phenomena, as vascular injections, do not occur in hyperæmia, consequently no trace will be found after death of the symptoms of diseases which belong to this class.

Two kinds of hyperæmia are known in general: when there is fluxion, or the active, and when there is stasis, or the passive. Their

phenomena are best studied on the skin. By the first is understood a strong, active blood flow—*i.e.*, caused by the cardiac or arterial pulse, or perhaps by local irritation, producing an increased filling of the capillaries. Hence the connection between a strong, rapid current of the blood through the circle of vessels affected and the appearance of vivid red and increase of temperature; for the rapidly flowing blood has not time to become carbonized and give up some of its warmth to the surrounding medium.

On the other hand, passive hyperæmia is considered to be an overfilling of the vessels in consequence of obstructed reflex or relaxed vascular walls, with at the same time a diminished rapidity of the flow of the blood, upon which depends the greater carbonization and heat radiation, and still more the dark-red coloring and the depression of temperature.

The cutaneous diseases limited by hyperæmia are divided, according to these two accepted forms, into (1) cutaneous diseases caused by active hyperæmia, (2) those caused by passive hyperæmia.

Only a difference according to these clinical characteristics is meant, since active hyperæmia shows itself under the symptoms of a more vivid redness, accompanied sometimes with increase of temperature, swelling, irritation, burning, and itching; and passive hyperæmia under the symptoms of more livid coloring, cool or lowered temperature, cold perspiration, and the appearance of nervous depressions, as numbness, formication, even anæsthesia, added to the redness of the skin. There is no essential practical difference between the kind of processes taking place in the vascular districts, whether they are produced by local or remote causes. It is shown that active and passive hyperæmia are partly conditional upon the same causes, as in so-called relaxed or paralytic hyperæmia, or *hyperœmia ex vacuo*, "aspiration fluxion," in which cases the vascular lumen becomes extended by paresis of the vascular walls. Further, active and passive hyperæmia, being mutually limited, are shown combined locally with one another; in collateral hyperæmia fluxion is marked in the periphery, and stasis in the centre of the area affected. Finally, the active hyperæmia with the symptoms of rapidly flowing blood is changed into passive with the phenomena of retardation in the local blood stream, which would be the case with a longer duration of the active accompanied by atony of the vascular wall.

Active hyperœmia of the skin is clinically designated as *erythema congestivum*, and is divided into *idiopathic* and *symptomatic*.

The *idiopathic active hyperœmia* represents cutaneous diseases *sensu stricto*. They arise from all general irritations and injuries

which, since they directly affect the skin, cause local hyperæmia. According to the different nature of the injuries we distinguish *erythema traumaticum*, which is caused by the pressure upon the skin of tight clothing, stays, stockings, and bands, these causing great pressure in walking and sitting; also by scratching with the finger nails and by the rubbing of the skin. If these causes are acting only a short time the erythema is transient; but with the repetition and longer duration of such irritation the erythema becomes an inflammatory process, or active hyperæmia is changed into passive. Places on the skin which have been for a long time the seat of erythema traumaticum have a special tendency to inflammatory disease, on account of the relaxation of the tissues caused under such circumstances. Consequently they often become diseased (as in variola, scabies) much more easily than other parts of the skin. Rough, inflamed nodules can be seen on the buttocks of the shoemaker suffering from scabies, owing to the sedentary nature of his occupation; and in women suffering from small-pox the variola efflorescences are much more abundant on the places where the clothes are laced, at the garters and the corset lacings.

Erythema caloricum consists of the most diffuse, at first very vivid and later livid-brown redness, which is caused by the influence of the sun's heat; according to the experiments of Charcot and Wolters they are perhaps occasioned by the influence of the chemical or special waves of warm or cold air in motion (on mountains and marshes), and lead to dark pigmentation and exfoliation. Warm and cold baths increase the vivid redness and transient erythema.

Erythema ab acribus seu venenatum is the name of the redness caused by irritating chemical substances, such as mustard plasters, horseradish, croton oil, cantharides, a number of vegetable extracts and ethereal oils, also many dye stuffs and medicines. The continued action of most of the substances here mentioned changes the erythema to inflammation.

The corresponding idiopathic erythema may be understood as arising in consequence of mechanical, caloric, or chemical poisonous irritations working directly upon the capillaries and finest vessels, thereby causing a rapidity of the blood stream and increased filling with a vivid-red injection, and is accompanied by a feeling of warmth—active hyperæmia—as well as paresis of the vaso-constrictors of the vessels affected or of the capillary wall, then dilatation and excessive filling of the vessels with a bluish-red injection. accompanied by a feeling of coolness—passive hyperæmia. But such erythemata originate near the places and spots which have been caused by direct irritation, as well as reflexly as from scratching. The mechanical irritation is conducted to the central organ. In the medulla oblongata and in the entire spinal cord are found the centres

for the vascular nerves which, partly directly, partly indirectly, run through the sympathetic with the spinal nerves to the skin (Goltz, Vulpian, Stricker, etc.). So it is clear that, as a result of an irritation working on one portion of the skin, another portion remote from the action of the irritation is affected in an analogous manner through dilatation and hyperæmia.

The symptomatically active hyperæmia or symptomatic erythemata are accompanying or secondary symptoms of other general febrile or afebrile conditions of single systems, especially of the central nervous system. They are considered as hyperæmia directly or indirectly excited by irritations of the central nervous system. The best known forms are those resulting from psychical disturbances, as the blush of shame, of anger, of passion.

A sense-perception (or also the adequate representation of such a sense-perception), the sight of a displeasing object, the hearing of a bad word, is perceived by the centre of consciousness. From this follows the disturbance upon the vascular centre and also upon the peripheral ends of the vascular nerves. The effect upon the last is called erythema pudicitiæ, iracundiæ. Erythema is frequently produced also through psychic processes.

In infants and young children erythema appears frequently as the reflex of the excitation of the central nerves and as a symptom of the disease which caused this excitation ; for example, during dentition, in consequence of gastritis. They are either diffuse—*erythema infantile*—or in the form of spots, a lentil to a finger nail in size, distributed over the body—*roseola infantilis.*

It is an error to consider that the blood changes caused by the poison of small-pox, vaccine, the poison of typhus, and the contagion of cholera, acting as an irritant upon the vascular centres, reflected upon the skin under the form of erythema, are the same as the effect of the irritation from a specific poison deposited in the skin, as, for example, the poison of varioloid. This appears in the first stage of *roseola variolosa* or *erythema variolosum*, situated for the most part upon the backs of the hands and on the muscles of the thighs, and will be spoken of later in connection with poison processes.

Roseola cholerica occurs in the asthenic stage of reconvalescence from cholera, in the form of livid, diffused spots the size of a thumb nail. *Roseola vaccinia* sometimes makes its appearance in the course of inoculation with human or animal lymph. *Roseola typhosa* is also no less known. So there exist different organic conditions of the blood and other systems, symptoms of which appear upon the skin as reflex erythema. The names mentioned are referred to by many authors as roseola febrilis, rheumatica, feu-dedents, nirlus, strophulus volaticus, rash, rosalia, measles, German measles.

In the same manner the poisonous action of many medicines is asserted. When taken into the vascular system they cause the outbreak of toxic erythema under different forms, which are known as "medicine exanthemata." There is not much to be said of all these forms of roseola as skin affections, since they do no harm subjectively or leave behind any local consequences. Besides, they have not the least significance upon the course of these diseases when they appear as their symptoms. The knowledge of them is not only pathologically interesting, but also of real practical importance, for the reason that it enables us to quiet the patient and his relatives concerning the slight significance of the affection, and also enables us to avoid mistaking them for measles, scarlet fever, syphilis, and other diseases similar to them, but diseases of great significance.

The *diagnosis* of these, because of the very different processes, from an existing roseola is not always easy. The circumscribed spots, described in respect to their anatomical signification and their course, are not to be mistaken. But in order to judge accurately of their special signification—*i.e.*, their dependence or relation to others and to what other processes of disease—it is necessary to have an exact knowledge of their processes and of all their complex symptoms.

Treatment of erythema is superfluous. For sensitiveness from burning and itching, cool applications can be used—cold water, touching with alcohol, plain or with the addition of carbolic acid 0.50 to 100.00, salicylic acid, and similar preparations.

Passive hyperæmia appears under the form, already mentioned, of a darker redness shaded from dark red, bluish-red, to dark blue, many times of a leaden-gray injection, which disappears under pressure of the finger. The skin is at both times either unchanged or œdematous, of the normal or of diminished warmth.

The most essential thing about passive hyperæmia is always the retardation of the local blood current, which again is the consequence of a disproportion between the mechanical power and the resistance (Virchow). But the circumstances under which this disproportion is brought about are manifold. The mechanical power of the heart itself or of the arteries (as in the case of atheroma) can be absolutely or relatively small, because the resistance of friction in the vascular walls has been increased. The local retardation of the blood may follow because the lumen of the small vessels has expanded; and the latter, again, takes place because of the engorgement of the blood which is caused by the mechanical hindrance of the current; or the vascular walls become more expansible and yielding on account of real illness, or neuro-paralytic condition, or through alteration by the action *ex vacuo*. All these conditions can also be combined in various ways.

The passive hyperæmia will affect first either the capillaries

and the small arteries, or at another time first the finest veins, resembling more the arterial injections or bearing from the first the venous character.

Passive hyperæmia is sometimes the result of those causes which locally affect the skin—injuries, chemical and caloric irritations which have as a consequence active hyperæmia—while after the longer duration of the action of these causes the vivid red is changed into dark bluish red (livedo traumatica, a venenatis, calorica). This condition shows a high degree of relaxed hyperæmia in consequence of a complete atony of the finest vessels affected. They are caused by the action of the already mentioned irritations of the skin, but especially by long-enduring pressure of hard substances, by sitting or lying, by pressure of bands, tight garments, and bindings. Connected with this is the passive hyperæmia in consequence of mechanical obstruction of the venous blood current, the so-called passive hyperæmia. The more nearly peripheral the obstruction lies, the smaller is the vascular region affected by the dilatation and overfilling with blood. The redness belonging to this category is called *livedo*, in contradistinction to *cyanosis*, the common cyanosis, the causes of which lie in the heart or in its neighboring vessels. The acute form of idiopathic passive hyperæmia, *livedo mechanica*, is represented most clearly in the phenomena which are called forth by the putting on of a bandage around the upper arm. Auspitz has experimentally proved these phenomena on the healthy skin, as well as upon such as were at the same time the seat of exanthema, erythema, urticaria, scarlet fever, variola and variola hæmorrhagica, eczema, erysipelas.

Aside from these richly illustrative observations, which must be referred to the distribution of the large vessels, and have been the occasion for discussion of the common pathological questions, Auspitz has made conspicuous the origin of many of the shades of blue, besides the appearance, already noted by Hebra, of vermilion spots which disappear after the removal of the ligature and leave behind brownish pigmentation, as well as the appearance of white spots around the cyanotic or vermilion ones. For the origin of the vermilion spots I find Auspitz's explanation plausible.

The pale or white spots, according to the observations which have been made with the microscope upon the circulation in case of stasis experimentally produced by binding or by embolus, can be explained by the fact that some vascular tracts become quite shut off from the general circulation and remain filled with colorless serum, also appear pale, while others are filled full of stagnant red corpuscles. With the duration of the stasis hæmorrhage occurs, *i.e.*, extravasation of the red corpuscles in the tissue, partly through rhexis, the rupture of the finest vessels, partly by diapedesis. Stricker has

proved that in stasis, through the change of the capillary vessels, red corpuscles find their way in microscopic masses.

The sensation of formication, numbness, lameness is found in the acute stage of *livedo mechanica*. In cases of greater duration and under similar conditions the sensation is normal and the temperature mostly reduced. As a further consequence, redness, inflammation, extravasation of blood (ecchymosis), tissue decay (necrosis), and gangrene (pressure gangrene) make their appearance.

There is a kind of passive hyperæmia, occurring frequently, and for the most part transient, which is caused by tight-fitting bandages, bands, stays, garters, etc. Passive hyperæmia resulting from long-continued pressure continues somewhat longer—*e.g.*, on the buttocks in sedentary persons, and on the sacrum in persons who lie on sick-beds for a length of time. Here occurs, besides, obstruction of the circulation through mechanical pressure, also through paresis of the vascular walls and the weight of the blood, which collects in the most dependent places. Livedo mechanica frequently appears in the lower extremities in chronic cases, in consequence of pressure upon the veins by tumors, exostoses, and gangrene in debilitated persons (*marantic gangrene*).

Collateral hyperæmia also, with the stoppage of a principal part of the circulation through embolus or thrombus, at first active, becomes passive hyperæmia, all the more so the less the local relation allows a rapid equalization of the check in circulation.

The nearer the mechanical obstacle to circulation lies to the heart, or if it lies at the heart itself, the more general is the passive hyperæmia. It is thus called *cyanosis*, morbus cœruleus; the latter is developed by chronic and acute emphysema of extreme degree, ulcers of the mediastinum, and all organic heart failures, which represent reflex stasis of the venous blood.

Many kinds of blueness of the skin, either locally confined or generally spread, depending upon a relaxed or paralytic hyperæmia, are conditioned also upon a primary flexibility of the vascular wall and expansion of the vascular lumen. Next comes the so-called *hyperæmia ex vacuo*, or the impaired support of the vascular walls in consequence of the lessened resistance. Here also belongs the hyperæmia which follows the application of the dry cupping glass. For this an air-tight chamber is used, and the blood, according to hydrodynamic laws, is driven quickly with great violence to the vascular wall or afterward aspirated through it. Just so the vessels, the supporting tissues of which are distorted by the retraction of the scars, as in debilitated persons, become relaxed through the lack of support from the reduced conditions, become expanded and the seat of passive hyperæmia. Thereby the law of gravity is of value in so far as the column of venous blood, where it has overcome in reflux

its own weight, flows more slowly, expanding itself and the vessels so much the easier the more the walls are already relaxed, be it through the causes just mentioned or those about to be cited. This appears especially upon the lower extremities in those persons who habitually stand, causing venous dilatation and livedo, besides the conditions accompanying these and resulting from them, the more easily the less tense the other tissues are.

The further causes for relaxed passive hyperæmia are essential diseases of the vascular walls, those which partly lead to, partly complicate, the varicosities of the lower extremities.

Finally, their cause lies in a neurotic relaxation, *livedo neuro-paralytica*, the material significance of which lies in the fact that the vaso-constrictors become paretic or paralyzed. This can affect restricted vascular districts. In this category is included passive hyperæmia arising from cutaneous irritation and a venenatus, which often follows active congestion ; also *livedo calorica*, which appears with sudden refrigeration of the skin as bluish-red mottlings, or makes its appearance as diffused dark-bluish redness with vermilion markings mixed with vermilion injections, on the end of the nose, the fingers, and the toes of persons who stay for a long period in cold rooms or in the cold out-door air.

Very interesting and classical cases have been reported in which nerve branches of the area affected, pressed by scars, have become irritated and atrophied, or, as a consequence of insufficient function of the vaso-motor centre or separate portions of the sympathetic system, appear as chronic redness of a vivid red, partly cyanotic shade—*local asphyxia*—accompanied with lowered, sometimes raised temperature, sensation of numbness and formication, or, on the contrary, of burning and pain, painful dryness, or the exudation of cold perspiration. Or it may be brought about by the neuro-paralytic influence of the central nervous system, of the central seat of the vascular innervation, establishing in the peripheral parts of the body, mostly symmetrically, on both hands, feet, ears, and the end of the nose, a blueness in anæmic persons and also those persons suffering from brain and spinal diseases, whereby the paræsthesia and functional disturbances usually present themselves in manifold combinations, forms, and intensity ; or in the form of disease known recently as that of Raynaud and that recognized as *myxœdema*. Neurotic asphyxia also appears limited to an arm, whereby at the same time swelling and pain of the cervical sympathetic on the same side makes its appearance, often the direct cause of such vaso-motor disturbance.

From the foregoing remarks it is evident that the chief cause of passive hyperæmia lies always in a slowing-up of the venous blood current, with dilatation of the finest vessels and also of the coarsest

venous trunks ; that the remote causes are partly local and peripheral, partly general and central, situated in the vascular system or outside of it, or in mechanical and neurotic influences.

Correspondingly the course of livedo and cyanosis is at one time short, at another chronic or permanent. In the last case are found significant secondary phenomena and complications, as œdema, and as a further consequence neoplasm of muscular tissue and connective tissue (pachydermia), inflammation, muscular debility, gangrene (symmetrical gangrene, Raynaud), etc. ; while again in other cases peculiar forms of atrophy of the skin and the underlying structures (*e.g.*, the nails on the fingers) arise from such conditions—trophoneuroses, which we will speak of later on.

Concerning the treatment of passive hyperæmia, it is only so far considered as individual symptoms demand attention, and the removal of the remote causes of the disease if possible. Generally, suitable pressure bands for passive hyperæmia may be used locally with success. With very painful hyperæmia which can be traced back to peripheral innervation disturbances, we have often seen the disease disappear after subcutaneous injections of morphine and hydrotherapy (baths near Vienna). Thus when an excitation of the vasomotor centre is indicated, as in paresis of the sympathetic, the internal use of iron, quinine, or ergot is recommended, as well as hydrotherapy and electrification of the sympathetic.

CLASS II.

CUTANEOUS ANÆMIAS.

MORBID PHENOMENA OF THE SKIN CAUSED BY DIMINISHED BLOOD SUPPLY OF ITS FINEST VESSELS.

In connection with hyperæmia I will now speak of anæmia of the skin.

Anæmia of the skin signifies a defective blood-filling of the finest vessels, or a sufficient filling but deficiency in the number of red blood corpuscles or in hæmatin, with or without a simultaneous diminution of white blood cells. In the first instance we have true oligæmia, anæmia, or ischæmia (Virchow); in the last, the conditions known as pseudoleukæmia, leukæmia, and leucocythæmia. The recent developments in hæmatology have revealed in a measure positive characteristics for the differentiation of the known forms of the conditions of the blood. The anæmic (vulgarly speaking, "blood-wanting") skin appears either pale, alabaster white, waxen white, grayish white, dirty white, or yellowish white. These shades of coloring depend upon the gradual or sudden origin of the anæmia, whether it is transient or lasting, whether it is associated with a qualitative change of the blood or general nutrition, and whether it makes its appearance upon a turgescent or flaccid skin. Each radically dark, pigmented portion of the skin, as also the skin of the negro, in the anæmic condition does not look pale, but even darker than normal, because with the loss of blood there is also a diminution of the serum absorbed by the tissue.

The anæmic pallor is associated with local diminution of the temperature of the skin. It is only in certain forms of chronic anæmia that the temperature of the skin may be increased.

Abnormal sensory symptoms, such as a feeling of numbness, formication, complete anæsthesia, chilliness, in rare cases severe pains, are among the accompanying subjective symptoms of anæmias of the skin.

As a general rule there are no striking local changes in nutrition, with the exception of the above-mentioned diminution of the turgor cutis. In some cases, however, we find œdema; in very protracted anæmia changes of secretion and of the formation of the epidermis. The skin may then be dry and brittle, or it may secrete "cold" sweat

or fat and the epidermis desquamates in abundant fine scales which are either dry or greasy (defurfuratio, *pityriasis abescentium*).

Other concomitant symptoms and sequelæ, such as poor general nutrition, loss of hair, gangrene, and a fatal termination in embolism of the peripheral arterial twigs, endarteritis obliterans, etc., do not belong to the anæmia of the skin *per se*, but to those conditions of the organism, of the constitution of the blood, central nervous system, heart, etc., which are the remote causes of the anæmia of the skin.

The immediate *cause* of anæmia of the skin can only consist of an insufficient supply of blood to the finest cutaneous vessels, or of the general poverty of the blood in red blood globules or in hæmoglobin. In the latter event the pallor of the skin is always general ; in the former event it may be general or localized.

Thus, general anæmia of the skin occurs in sudden diminution of the amount of blood after hæmorrhages externally (metrorrhagia, operations) or into internal organs. When this is excessive the coincident anæmia of the brain may give rise to the well-known symptoms of syncope—pallor of the lips and mucous membranes, pallor and coldness of the skin, obscuration of vision, paralysis of the muscles, unconsciousness, cessation of the heart's action and of respiration, and even death. Sudden displacement of the blood, when present in sufficient amount, may also give rise to local anæmia together with hyperæmia of other parts and all their attendant results. This category includes the cases of syncope and sudden death which have been observed after the loosening of Esmarch's bandage. The vessels of a lower limb which have been evacuated by the constriction, and probably rendered paretic by the pressure, suddenly receive a large amount of blood, other parts of the skin grow pale, and the brain also becomes anæmic. This explains the subsequent onset of syncope and of the sometimes surprising fatal termination.

General chronic anæmia of the skin is the result of insufficient formation of blood, either in quantity or quality, such as occurs as a symptom of chlorosis, pseudoleukæmia, pernicious anæmia. scrofula, splenic enlargements, tuberculosis, and protracted, febrile, and exhausting diseases.

When the blood is normal in amount and constitution, general or local anæmia of the skin may be the result of nervous influences—*neuroparalytic* and *neurospastic anæmia*. It must then be assumed that the finest arteries and the capillaries contract and present an obstacle to the entrance of sufficient amounts of blood—*i.e.*, to the normal injection with arterial blood. Apart from the contractility of the arteries, the investigations of Golubew-Tachanoff and Stricker

warrant us in assuming that even the capillaries react to direct irritation by contraction and narrowing of their lumen. Under certain circumstances (as in the chill of a febrile attack) the contraction of the cutaneous organic muscular fibres, which form networks in places beneath the papillæ, may also aid in the contraction of the vessels which enter the papillæ and in the production of pallor of the skin.

In this way we find that local anæmia of the skin, with the symptoms of pallor, diminished temperature and sensibility, develops on the application of cold (ether and chloroform spray) for the production of local anæsthesia, and on the application of the electrical current, and also under the influence of low temperatures, such as cold air, cold baths and douches. Here the local irritation produces contraction of the vessels which are supplied with nerves, and anæmia.

As we have already said in regard to hyperæmia, all these conditions are followed finally by ectasia and distention of the cutaneous blood vessels.

The same effect, with anæmia of the skin, may also start from the central nervous system, and then usually appears as a general pallor of the skin, as in the stage of chill during fever, in the mental excitement of fright, anger, fear, envy, and in syncope in general. This effect may also be reflex—for example, from the cutaneous nerves, as in the pallor of the skin, or even syncope, which may follow even slightly painful stimulation of the skin (slight operations); or from the splanchnic nerves, as in the pallor which accompanies nausea or precedes vomiting in overloading of the stomach, colic, blows on the abdomen.

Mention may also be made of cutaneous anæmia as the result of compression of the finest vessels of the skin. Parts which are compressed by lying upon them, by bandages, etc., appear anæmic and pale, and are the site of formication, numbness, or even anæsthesia. This condition is merely temporary.

In compression of the small vessels by œdema of the tissues the cutaneous pallor is permanent. The skin is then tense, shining, alabaster-like, or has a waxy appearance.

A peculiar form of anæmia, which leads in time to sensory, functional, and trophic disorders (usually of an atrophic character) in the more peripheral parts of the body (hands, feet, ears, nose), has been observed in a few individuals, usually females, in whom congenital narrowing of the vascular system must be assumed.

(Occlusion of large arteries by embolism, the presence of tumors or endarteritis obliterans, rapidly leads to collateral hyperæmia, unless mummification sets in.)

According to the differences in the causal agent, the cutaneous anæmia will be fleeting, prolonged, or permanent.

As a cutaneous affection it possesses chiefly a symptomatic significance, and its prognosis and treatment depend mainly on its causes. We may be called upon, however, to treat the previously mentioned sequelæ, such as pityriasis, alopecia, etc.

The *diagnosis* of cutaneous anæmia is important, not alone in order to fill out the entire pathological picture found in the patient, but also on account of the influence it exerts upon other coexisting dermatoses and morbid formations. In so far as the latter are characterized by congestive redness and turgescence, these symptoms will be absent in cutaneous pallor, and the affection in question will therefore be less marked and will be diagnosed with greater difficulty. Thus psoriasis, squamous eczema, syphilides, etc., are difficult to recognize in anæmic persons, on account of their pale color. In the same way the sudden or subacute onset of anæmia of the skin at once obliterates a material characteristic of many cutaneous diseases. Trivial as this fact is, its observation and correct interpretation were very important. With it Hebra successfully combated the old superstition concerning the recession of cutaneous eruptions. He showed that it is owing to the anæmia of the skin that, for example, a long-standing psoriasis grows pale after a severe hæmorrhage and seems to disappear suddenly, or actually subsides during an exhausting disease which is associated with pallor and diminished turgescence of the skin. With the return of the normal injection and turgescence of the skin the eruption again becomes recognizable or returns.

We should not return to the errors of former times, but should always interpret the facts in the manner just indicated, although the patient and his family may often attempt to lead us astray.

CLASS III.

ANOMALIES OF THE SECRETION OF THE SKIN AND OF THE CUTANEOUS GLANDS—DISEASES OF THE SKIN DUE TO ABNORMALITIES OF THE CUTANEOUS SECRETION AND THE CUTANEOUS GLANDS.

LECTURE VIII.

ANOMALIES OF PERSPIRATION AND OF THE SECRETION OF SWEAT.

PHYSIOLOGY OF THE SECRETION OF SWEAT—CHEMICAL CONSTITUTION OF SWEAT AND ITS MORBID SECRETION—QUANTITATIVE DISORDERS—GENERAL AND LOCAL HYPERIDROSIS—LOCAL AND GENERAL SEQUELÆ AND COMPLICATIONS—TREATMENT—ANIDROSIS—QUALITATIVE ANOMALIES OF THE SECRETION OF SWEAT—ANATOMICAL CHANGES.

THE skin diseases which belong to the third class of our system consist mainly of abnormalities of cutaneous secretion and of the cutaneous glands, and are manifested by (1) functional and (2) nutritive disorders.

I. FUNCTIONAL DISORDERS OF THE CUTANEOUS GLANDS.

The functional disorders of the cutaneous glands constitute anomalies of secretion. But as the secretion has a material influence upon the condition of the general integument, especially of the epidermis, it is evident that with changes in the secretion the condition of the epidermis may also undergo change.

The secretions of the skin are twofold: sweat, the product of the convoluted glands, and fat, which is produced by the sebaceous glands. This is the doctrine which is generally taught, and, on the whole, the statement is true. But no physiologist has yet succeeded, despite numerous experiments, in separating the secretions of the skin—which are not alone derived from the glands just mentioned, but also from the materials exuded from the papillary vascular system—in such a way that the pure product of the sweat glands or of the sebaceous glands could be obtained. The statements made by Thenard, Anselmino, Schottin, Séguin, Funcke, Favre, and others,

concerning the chemical and morphological constitution of perspiration and the fatty secretion of the skin, really refer to a mixture of both, in which sometimes the one, sometimes the other predominated.

Unna, adopting a theory which had been suggested by Meissner, has attempted to show that the watery secretion of the skin, the sweat, is derived solely from the papillary network of vessels, and that the coil glands, which have hitherto been regarded as sweat glands, secrete only fat. This is emulsified, carried off by the papillary lymphatics, and then stored up in the cells of the sebaceous glands.

This purely theoretical notion is combated by various facts which have again been investigated histologically by Max Josef. Thus the sweat glands contain no fat, and they are surrounded, like the renal glomeruli, by a network of vessels to which we must ascribe a similar function—viz., the secretion of watery solutions. Furthermore, Arnozan made the interesting observation that the palm of the hand, which contains sweat glands but no sebaceous glands, is always free from fat.

Nevertheless we cannot entirely deny a fatty secretion by the sweat glands, not alone by those in the axilla (which, as we shall see, correspond more to the type of the ear-wax glands), but also by the coil glands. According to Ranvier and others, the cells of the secreting portion (coil) of the glands contain drops and granules of fat, although it is probable that these are derived from fatty degeneration of desquamated secretory epithelium.

Practically we regard a cutaneous secretion as a product of the sebaceous glands when it exhibits in the main the properties of fat, while a secretion which is chiefly watery is attributed to the sweat glands. Under normal conditions a mixture of both is found upon the skin. This mixture, combined with certain products of exhalation (gaseous and fluid) which escape from the papillary vascular system and through the epidermic covering, forms the perspiratory matter (*materia perspiratoria*) of the integument.

There is no doubt that there may be anomalies of the entire cutaneous perspiration. In some persons the materia perspiratoria is changed quantitatively or qualitatively.

While the quantitative changes are difficult to describe symptomatically, the qualitative changes exhibit more distinct characteristics, which are noticeable chiefly by the sense of smell. Even the laity ascribe to every one a peculiar "exhalation," which is undoubtedly present and can be perceived by the sense of smell.

We know that dogs, which are provided with more delicate olfactory organs, are thus enabled to track their masters. The condition becomes abnormal when the individual has an unusually strong or peculiarly smelling perspiration (*osmidrosis, bromidrosis*). Of such persons it is literally true that they stand in bad odor.

It is difficult to ascertain exactly to what substances the pene-

trating or pronounced odor of the sweat is due. They appear to be mainly volatile fatty acids which are the product of the sebaceous glands, but partly also of the sweat glands and the analogous axillary and ear-wax glands.

Those regions of the body which possess unusually large sweat and sebaceous glands, such as the axillæ, the integument of the genitalia (especially in females), are also the principal site of osmidrosis. This is distinguished as *local* osmidrosis or bromidrosis.

Hebra showed that in many cases foul sweat is not perspiration proper. The latter does not smell worse than in the majority of people, but the foul odor only develops when the perspiratory matters, particularly the sweat, undergo decomposition and form fatty acids, after remaining for a long time on the skin and impregnating the stockings, shoes, and underclothing. This is not true bromidrosis.

Nor does this condition include the peculiar odor of the emanations from the skin when the individual has taken certain articles of diet and drugs (either internally or by inhalation) and the latter are then excreted by the cutaneous glands.

Under certain diseased conditions, such as general cachexia, syphilis, tuberculosis, and the active stage of various acute exanthemata and fevers, the perspiration acquires a more pronounced odor. Some physicians (Heim, Schönlein) regarded this as so characteristic that they claimed to be able to make a diagnosis thereby. It is wiser, however, not to rely too much upon the olfactory organ, and to differentiate scarlatina, measles, and variola by other symptoms than by the smell of fresh feathers, a menagerie, freshly baked bread, etc.

We will now consider the anomalies of the secretions whose source is more distinctly recognized—*i.e.*, the secretion of sweat and of fat.

ANOMALIES OF THE SECRETION OF SWEAT—DYSIDROSIS.

These will be understood more easily after a consideration of the physiology of the secretion of sweat.

We have already stated that the vascular system of each coil gland forms a small network. The afferent arterial branch ramifies into a network which surrounds the coils of the gland, from which a collecting artery emerges. This condition is similar to that observed in the Malpighian bodies of the kidney. Hence the secretion of the sweat glands is also withdrawn from the arterial blood.

This analogy also finds expression in the chemical constitution of the sweat, so far as this has been determined. According to the statements of most writers, and also according to the latest investigations of Heuss, sweat is an acid fluid which contains less solid substances and becomes less acid the greater the amount secreted. Indeed, it may finally become alkaline. Its main constituent is water

(about ninety-nine per cent), in which are found very small amounts of the salts usually present in the body (sodium chloride, lime phosphate, etc.). In addition, lactic acid (in rheumatics), uric acid, urea and its product of decomposition, ammonia, a peculiar hydrolic acid, and indigo have also been found in it. The latter constituents distinctly show the analogy with the renal secretion.

In addition, solid or volatile fatty acids have also been found, and their presence is revealed by their peculiar odor. These are not derived solely from the secretion of the sebaceous glands, which are scattered among the sweat glands, because they have been found in places which contain only sweat glands (the palm of the hand). This is also true of the large axillary glands and of the circumanal glands described by Gay, whose secretion appears to be analogous to that of the ear-wax glands. There is no doubt that to these constituents is due the peculiar odor of certain sweats, particularly in certain regions of the skin.

It has recently been demonstrated by Brunner, of Zurich, and confirmed by Von Eiselsberg, that pus cocci (staphylococcus albus and pyogenes) circulating in the blood are eliminated by the sweat or rather by the general perspiration. In chronic pyæmia these writers found the cocci just mentioned in the profuse sweats produced by artificial stimulation. The curative and prognostic significance of the "critical sweats" of the older writers, and the indication to produce them artificially, thus receive a scientific foundation.

Under ordinary conditions the sweat is secreted in imperceptible amounts. It evaporates, but if evaporation is diminished by impermeable covering, oiled silk or rubber, it may be deposited in the form of drops. In active or passive heating of the body and greater fulness of the cutaneous vessels it appears in clear drops and large amounts.

The secretion of sweat depends not alone upon the increased distention of the cutaneous vessels due to increased action of the heart, but is influenced in great measure by the nervous system. Every one knows that psychical and sensorial excitement of the brain, fear and fright, severe pain, nausea, etc., will lead to the appearance of large drops of sweat upon the brow or the general integument. Contraction of the finest arteries, such as occurs under the influence of cold or a chill, is attended with inhibition of the secretion of sweat, relaxation of the vessels, or, in warm temperatures and during the subsidence of fever, is attended with increased secretion. Hence there is no doubt that the secretion of sweat may be increased or inhibited by local, central, or reflex nervous influences. During the last few years, in which the vaso-motor nerves and their centres have been the subject of numerous experiments, we have learned that the track of these nerves is identical with those of increased secretion of sweat; that the division and stimulation of sympathetic fibres and

of sensory nerves containing them will inhibit or stimulate the secretion of sweat, in the same way that it acts upon the salivary or pancreatic secretion. Irritation of such a nerve trunk will even produce sweat upon an amputated extremity—*i.e.*, one which is cut off from the circulation (atropine checks the secretion of sweat). The sweat nerves of the hind feet of the cat are found in the sciatic nerve. These pass either directly or indirectly(through the abdominal portion of the sympathetic trunk and through its rami communicantes and the anterior spinal roots) to the upper lumbar and lower dorsal cord, where the sweat centre of the posterior limbs appears to be located. The sweat nerves of the forepaws of the cat pass along the ulnar and median nerves and then to the lower cervical cord, either directly through the spinal roots or indirectly through the thoracic portion of the sympathetic. The dominating sweat centres for the entire body are situated in the medulla oblongata (Adamkiewicz) and the cerebrum itself. Coyne has partly demonstrated the existence of peripheral ganglia of the sweat glands: irritation of nerve fibres which pass to the smooth muscular fibres of the larger coil glands causes a sudden production of sweat. In addition to the labors of Vulpian, Betzold, Goltz, Samuel, Ostrumoff, and others, the experiments of Stricker on the tonic vaso-motor centres and collateral innervation, and those of Kendall, Luchsinger, and Navrocki on the influence of nerve stimulation upon the activity of the sweat glands, have shed a great deal of light on the subject.

Pathological confirmation of these conditions is found in cases of abnormal local secretion of sweat, within the distribution of sensory nerves which are paralyzed or irritated (as in migraine), in injuries to nerves, traction by cicatrices as the result of zoster, gunshot wounds of nerves (Weir Mitchell). According to Bouveret's recent experiments and analysis of cases the hyperidrosis appears to be due to stimulation of the cerebro-spinal nerves or to a paralysis of the sympathetic.

There is an undeniable analogy with the neurospastic and neuroparalytic hyperæmias of the skin which have already been described. This is natural in view of the fact that the secretion of sweat is regulated directly by the circulatory conditions of the vascular networks surrounding the coil glands, although there may also be an independent influence by the secretory nerves, like that demonstrated by Claude Bernard with regard to the parotid.

The immediate physiological purpose of the secretion of sweat appears to be heat regulation, because it generally becomes considerable when the temperature of the body is elevated, and by its subsequent evaporation aids in withdrawing heat from the body. In addition an excretory purpose must also be ascribed to sweat. This seems to be expressed not alone by the previously mentioned constituents, but

also by the experience that, under physiological conditions, the secretion of urine and that of sweat is quantitatively proportionate to each other. The more abundant the perspiration the more scanty and concentrated the urine, and *vice versa*.

This every-day experience has undoubtedly led to the view, which still obtains in many places, that the suppression of perspiration, especially when it has been increased pathologically, has injurious effects upon the organism and may produce colds or even more serious ailments.

It cannot be denied that, in the same way as exudations and œdemas are more rapidly absorbed when the renal secretion is increased, this action is also furthered by increase of the activity of the skin. But these secretory conditions are in themselves a result of the subsidence of fever and the associated vascular activity. They by no means warrant the inference that diminished cutaneous transpiration will cause increase of an existing exudation or will cause disease of an internal organ, because the normally acting kidneys excrete a disproportionately larger amount of metabolic products than the sweat glands, whose secretion consists almost entirely of water. The least tenable notion is that of the "recession" of sweat which has already been secreted. This is as inconceivable as the reflux of urine if there are no obstacles in its way. For this reason we do not hesitate to check excessive perspiration when it appears to be pathological. We endeavor to cure such conditions, and have never observed the slightest bad results from such a course. Our sole complaint in this regard is our inability, in many cases, to check the morbidly increased secretion.

It is almost superfluous to say that the sudden cessation of perspiration in a cold draught does not indicate "recession" of the sweat, but a rapid evaporation of the secretion already present.

The secretion of sweat may become pathological either quantitatively or qualitatively.

The former category includes excessive increase (dysidrosis or hyperidrosis) and abnormal diminution (anidrosis).

Hyperidrosis is spoken of as a morbid condition when an excessive amount of sweat appears upon the skin under circumstances in which this does not happen in the majority of individuals. Hence it does not include the excessive sweating produced by increased heat of the body from the heat of the sun or a fire, from bodily effort, etc. Nor do we refer to those conditions which accompany or follow general diseases, acute and chronic fever, tuberculosis and chronic cachexia, and which, in acute febrile conditions (typhoid fever, pneumonia), are also regarded as critical. Between the years 1485 and 1550 five epidemics characterized by excessive sweating were reported in England, France, and Germany. This is known in the history of

disease as *sudor anglicus*. A similar epidemic is reported to have occurred in 1718 in the Picardy and others have been reported by Rottingen (1802) Sulzfeld (1864), Poitou ("Epidemie de Suette miliaire," 1887). This was a febrile, probably contagious disease, associated with eruptions, and in some cases running a fatal course.

The hyperidrosis which is the subject of dermato-pathology is independent of such causes.

It either affects the entire integument (*hyperidrosis universalis*) or is confined to a few parts of the body (*hyperidrosis localis*).

In the diffused form hyperidrosis is found chiefly in fat people, more rarely in those of moderate adipose. Slight bodily or mental effort, a stay in a moderately warm room, mental excitement, confusion, or impatience will produce in such cases a sudden and profuse secretion of sweat. The skin may either feel warm and turgescent, or it may be cool, especially if the perspiration has remained upon the skin for a long time. The latter condition is due to the abstraction of heat by the evaporating fluid.

In some individuals hyperidrosis universalis occurs often, but only for a brief period ; in others it is habitual and continuous. As a morbid function of the skin it usually lasts many years and is found almost exclusively during middle life. It may also occur in youth in cases of premature obesity.

The outbreak of copious perspiration is usually preceded by a disagreeable prickling sensation in the skin, sometimes by a feeling of oppression. Hebra attributes these sensations to congestion of the papillary vessels, which irritates the cutaneous nerves, and this view appears very plausible. After the appearance of the sweat drops the disagreeable sensations subside and the patient feels more comfortable and relieved.

The outbreak of sweat is sometimes accompanied by a cutaneous eruption consisting of moderately itching, very red and firm nodules, as large as a millet seed or a little larger, or of vessels filled with a clear, watery fluid. These constitute the condition known as *sudamina*, prickly heat, heat or sweat vesicles, eczema sudamen, and which occurs in all individuals, especially those with a delicate skin (children), when profuse sweating results from overheating. It cannot be said that the sudamina are the effect of the sweating, in the sense that the latter softens the cutis and irritates the papilla, as happens, for example, as the result of irritating ointments, hot baths, protracted sweating. The sudamina appear almost at the same time with the perspiration ; they appear to be the product of watery secretion from the congested papillæ. Sweat escapes from the excretory ducts of the coil glands, accumulates between the epidermis cells which form the walls of its spiral extremity, and causes mechanical irritation of the adjacent papillary nerves and vessels. In the same

way fluid exudes from the papillary vessels between the layers of epidermis and elevates them into little nodules and vesicles. Hence the eruption has essentially the same significance as the disease which we shall study under the title eczema, and into whose characteristic form it may develop when the skin is irritated by protracted contact of sweat or by improper treatment. Under proper treatment, however, and in cases of temporary hyperidrosis, the nodules and vesicles soon grow smaller The latter are followed by fine furfuraceous desquamation of the raised layers of epidermis, and then the skin returns to the normal.

Although the treatment will be considered in the chapter on eczema, I will here call attention to the fact that, when sudamina are present, everything must be avoided which will cause further production of sweat and irritation. Baths, ointments, warm clothing, heating drinks, and bodily exercise are to be avoided. The skin is cooled by moistening with alcohol, cologne, and the like, and the perspiration is removed by dusting the surface with starch powder.

One of the results of hyperidrosis universalis is the maceration of the cutis and reddening of the skin (*intertrigo*) in those parts which are especially favorable to the retention of sweat and its renewed production. Hence it is found in the adjacent folds of the genital region, beneath a pendulous breast, upon the trunk, etc. This condition may also be intensified into *eczema intertrigo*, which will be discussed later.

Local hyperidrosis is more common and of greater practical importance. It appears as an habitual abundant secretion of sweat upon individual parts of the skin. The most frequent sites are the face (forehead and chin), scalp, axillæ, perineum, palm of the hand, and sole of the foot.

Excessive sweating of the axillæ is especially frequent in women, and, as a rule, is associated with a penetrating odor (osmidrosis). It is annoying on account of the smell and of the discoloration of the clothing, which is impregnated with the perspiration. Eczema results in cases of long standing.

Hyperidrosis of the palm of the hand is a very annoying condition. However often they are washed and dried, the palms of the hands and the inner surfaces of the fingers are immediately covered with perspiration, which even appears in clear drops protruding from the dilated openings of the sweat glands, which are visible with the naked eye.

Habitually sweating hands always feel moist, cool, and clammy. Such a condition lessens the desire of grasping the hand of even the most beautiful woman, and in many cases may have cooled the fires of passion. The affected individuals feel this. They rapidly dry the hands before extending them. Their gloves are at once

moistened and discolored, their handiwork always looks dirty and greasy, because there is no doubt that the sweat glands occasionally produce fat. The patients may even be disturbed to a considerable extent in their calling and practical career. The condition may last for years without producing any local changes in the skin. At most the epidermis appears thin, here and there raised in very superficial vesicles, and wrinkled at the tips of the fingers. In rare cases there appear vesicles as large as wheaten grits, or even pustules. This has led to the description of a special form of disease by Hutchinson (cheiro-pompholix), Tilbury Fox (dysidrosis), Robinson (pompholix), although in fact the symptoms are really those of an acute outbreak of eczema.

This condition is found mainly in young people of both sexes, often associated with chlorosis and poor digestion, coldness of the hands and feet, cyanosis of these parts (local asphyxia). We may then find that the condition is accompanied by very annoying, painful sensations of a burning character. After the lapse of months and years there may develop, either with or without the previously mentioned sensations, profound tissue changes which we shall discuss later under the title keratosis glabra et verrucosa.

These changes are to be considered, not as the result of the hyperidrosis of the palm of the hand (and sole of the foot), but of the local asphyxia and the vaso-motor disturbance. I have also observed the same condition develop in middle life without any demonstrable cause. In the same way the hyperidrosis may also disappear, without any noticeable cause, after the lapse of months or years. It is always, however, an annoying and obstinate ailment.

This is also true of habitual sweating of the feet (hyperidrosis pedum). Any one may occasionally suffer from profuse sweating of the feet and its local results—for example, after a long walk on a hot summer's day. The sweat softens and loosens the cutis, particularly over the adjacent surfaces of the toes and the transition folds, at the tips of the toes and the soles of the feet; the skin becomes fissured and extremely painful. A firm, free step is rendered difficult or even impossible.

The same condition obtains in hyperidrosis pedum, which often lasts from earliest infancy to adult life. It is usually more moderate during cold weather and when the individual remains quiet. As a rule this excessive perspiration has a foul odor (bromidrosis), not because the recently secreted sweat has a penetrating odor, but because it impregnates the footgear and decomposes.

Habitual sweating of the feet is not alone annoying on account of the constant maceration of the skin and interference with walking, but also directly and indirectly on account of the accompanying

bromidrosis, which makes the patient unendurable to those about him.

Sweating of the hands and feet is sometimes combined in the same individual.

We are entirely in the dark with regard to the more remote *cause* of local hyperidrosis. In many cases it may be looked upon as a simple exaggeration of the physiological function of the skin. I have already said that sweating of the hands is associated occasionally with chloranæmia and chronic indigestion.

The immediate cause of the hyperidrosis is always located in the capillaries of the integument, especially of the coil glands and papillæ. These exhibit either active congestion, as in the increased sweating of the axillæ and genitalia due to heat, or passive congestion due to neuroparetic dilatation.

As I have already shown in the discussion of hyperæmia, these conditions are regulated by the vaso-motor (sympathetic, secretory) nerves, and I refer the reader to the remarks made in that connection. We may thus understand how a profuse secretion of sweat, either general or local, may appear under the influence of mental excitement, fright, confusion, or local pains, as the result of a stimulus which is either reflex or starts from the central organs. This has also been shown experimentally. On section of the cervical sympathetic, Claude Bernard found that the vascular paralysis was associated with profuse perspiration in the paralyzed parts. This etiological factor obtains in the numerous cases in which hyperidrosis has been observed after suppurating parotitis, in the distribution of irritated or paralyzed sensory and vaso-motor nerves.

These forms are related to those varieties of unilateral local hyperidrosis which correspond to definite nerve tracts—for example, on the corresponding part of the body, in migraine or after zoster gangræ-nosus, or extending over one half of the body in paraplegia. Such cases have been reported by Hartmann, Er. Wilson, Hebra, P. Guttmann, myself, and others. In one case of migraine I found the forehead on the normal side cool and dry, while the parts corresponding to the distribution of the frontal nerve on the painful side were slightly reddened and covered with drops of perspiration ; in a woman and a man who had suffered from zoster collaris, clear drops of sweat appeared in the corresponding region upon the slightest excitement. In one case of hyperidrosis of the left side of the body Fränkel and Ebstein found stasis of blood globules in the vessels of the corresponding cervical sympathetic. In one case, which affected the left side of the head, Riehl noted, in addition to the same findings in the vessels, interstitial cellular infiltration of the superior ganglion of the cervical sympathetic. According to Riehl's *résumé* of the known cases of unilateral hyperidrosis in the distribution of

the trigeminus, the majority of cases were attended, during the attack, by myosis ; in a few cases mydriasis was present.

Cases of sympathetic paralysis with myosis and vessel dilatation, in which anidrosis was present, have also been reported (Moebius).

I am acquainted with one woman, who suffers from syphilis, in whom, previous to infection and to this day, crossed unilateral sweating was observed. The left side of the face and the right side of the body are covered with drops of sweat on the slightest emotional excitement, while the rest of the body remains dry.

In the majority of cases, however, the etiology of local hyperidrosis remains entirely obscure.

In regard to *prognosis* we possess very few data, with the exception, perhaps, of the purely neurotic forms, and even then it must be made very guardedly.

In the most frequent forms—viz., hyperidrosis of the axillæ, palms of the hands, and soles of the feet—an unfavorable prognosis should not be given, because the disease often disappears spontaneously after lasting for years, and treatment is usually attended with some benefit or even with complete success. Unfortunately, however, many cases resist all treatment.

In the treatment of hyperidrosis of the axillæ, genitalia, and palms of the hands, and in that of the milder forms of sweating feet, we may recommend frequent washings with solutions of tannin (1 gramme to 250 grammes alcohol or water), alum, soda, decoctum corticis quercus (20 : 500), corrosive sublimate (1 : 400), permanganate of potash (5 : 400), soda ammoniate, simple alcohol and ether, or with the addition of extractum aconiti (1 : 200), calumbo, etc. For the hands and feet such solutions may also be used as local baths. In some cases of sweating of the palms of the hands and soles of the feet, excellent results have been obtained from washing with naphthol 1.0, spiritus vini gallici 275, spiritus coloniæ 25. The absorption of the sweat and the separation of contiguous folds of skin must be secured by frequent dusting with powder, amylum tritici. oryzæ, either pure or combined with oxide of zinc, carbonate of lead, salicylic acid, powdered naphthol (1 : 100 amylum). Lint dusted with these powders may also be applied, particularly in the interspaces and furrows of the toes, the genital folds, and the axilla. In axillary sweating the pads of rubber cloth or oiled silk made by dressmakers are very injudicious ; by preventing evaporation they induce increased accumulation of sweat and cutaneous irritation.

The remedies mentioned will usually prove effective in the milder cases of sweating feet. The introduction of powder bags between the toes and the lower furrow of the toes must be repeated several times a day.

In the higher grades of hyperidrosis and bromidrosis pedis Hebra's treatment with unguentum diachyli proves very serviceable.

As this ointment is generally recognized as a valuable part of our armamentarium and is extensively used in practice, we will describe its mode of preparation. It was first made of emplastrum diachyli simplex with oleum lini, later by boiling with oleum olivar. For a number of years it has been made from lithargyrum and oleum olivar. according to the formula: lithargyri 100, olei olivar. 400, heating over a slow fire, and adding sufficient water to make an ointment of the consistence of stiff paste, then adding olei lavandulæ 10.

In treating severe hyperidrosis and bromidrosis pedum this ointment, of the thickness of a knife blade, is smeared upon a quadrangular piece of well-washed coarse linen sufficiently large to cover the foot. The foot, after being well washed and dried, is placed upon the cloth. Between the toes and in the grooves of the toes are placed pledgets smeared with ointment, and the cloth is then wrapped around the foot. The patient must then wear new shoes and stockings. He may then attend to his business, although it is better to remain in the recumbent position, as the ointment then exerts a more rapidly favorable effect. 'At the end of twenty-four hours the linen is removed; the feet are not washed, but merely dried with lint and powder, and then covered with a freshly anointed cloth as on the previous day. This procedure is repeated for ten to fourteen days. The ointment is then discontinued and powder is faithfully dusted upon the foot and into the folds. Within a few days the skin exfoliates in thick, yellowish-brown, parchment-like shreds and the hyperidrosis is cured. Then the foot may be washed for the first time.

For a long time afterward, especially after long walks and during hot weather, it is advisable to powder the feet, particularly the grooves of the toes, or to place powder bags in the latter.

If the result is not entirely successful the treatment should be repeated immediately. Permanent recovery is then obtained with certainty.

Equally favorable results are sometimes obtained from the use of a ten-per-cent emplastrum saponatum, emplastrum salicylicum, or a resorcin plaster.

Neither I nor any one else has ever seen any bad effect upon an internal organ or upon the general condition as the result of the relief of local excessive sweating by means of external remedies. I emphasize this fact in order to furnish you with a weapon against the wide-spread prejudice that dangerous diseases, or even sudden death, may occur when the sweating is checked by treatment, or ceases spontaneously, or even disappears temporarily from sudden cooling. It has also been taught, within recent times, that sweating feet should be relieved cautiously, not suddenly. This is not within our power nor

within that of the writers who furnish the warning. I refer the reader to the remarks made concerning the supposed results of recession of cutaneous eruptions. Both opinions are due to the same false interpretation of clinical facts.

Such objections are made less frequently against the use of internal remedies which are recommended in the treatment of hyperidrosis. These include decoctum chinæ, extractum aconiti, and especially agaricus albus powdered, 0.3–2.0 and 3.0 at a dose; agaricin ½ mgm. at a dose up to 2 cgm. daily; and atropine 0.02, gum tragacanth 1.50, glycerin, pulv. liquir. āā q. s. ut f. pil. No. xx. Sig.: two pills daily. Atropine is also given in solution in increasing doses. The two last-mentioned remedies are occasionally attended with striking, although usually temporary, effects. Tonics and diuretics are also employed, the latter in order to stimulate a vicarious hypersecretion of the kidneys.

Anidrosis is an insufficiency or complete absence of the secretion of sweat. It is associated with a dry, brittle condition of the epidermis, and the subjective sensation of dryness, tension, uncomfortable general sensibility, tickling, and itching.

The insensible perspiration is, however, never abolished. This becomes noticeable as a fluid excretion whenever the skin, however dry it may feel, or even if affected with one of the so-called dry dermatoses (psoriasis, ichthyosis, prurigo, etc.), is covered with some material (rubber cloth) which prevents evaporation. Hence there is in reality no absolute anidrosis.

Anidrosis cutis has not been observed as an independent disease. Apart from the peculiarity of certain individuals, who perspire very little or not at all amidst great heat or on exercise, anidrosis as a pathological condition is always observed as an accompaniment of certain general conditions of nutrition or of skin diseases with well-defined characteristics, such as prurigo, chronic eczema, psoriasis, ichthyosis, and xeroderma. Anidrosis may be diffuse, as is usual in diabetes mellitus and insipidus, in the cachexia of tuberculosis or cancer. The deficient secretion of sweat may go hand in hand with excessive or changed secretion of the sebaceous glands. Or anidrosis, like the dermatosis which it accompanies, may be localized, and in both cases it may be temporary or permanent. In those forms which accompany dermatoses there is a constant relation between the two conditions, so that the secretion of sweat disappears and reappears with the coming and going of the skin disease. For example, a part of the skin which is suffering from chronic eczema is also anidrotic, but the perspiration reappears as soon as the eczema diminishes. This condition has also been interpreted in the sense of dermapostasis, as if the eczema constituted a sort of external deposit because the

sweat and its products were retained within the body. The fact has been overlooked that the hyperæmias which give rise to the chronic eruptions, such as eczema and psoriasis, also carry more products to the coil glands, and that in the same way that excessive serum is exuded and epidermis produced, a greater amount of sweat might also be secreted. If the sweat glands do not functionate under such circumstances it must be due to the nutritive disturbance of the skin which is inherent in the eczema and psoriasis. And in fact the sweat returns with the disappearance of the nutritive change which constitutes the dermatosis.

In extent and localization the anidrosis follows the dermatosis exactly—a fact which will be further considered under the heading of symptomatology of the different diseases.

In addition to local nutritive disturbances, nervous influences may also induce regional anidrosis—for example, in parts of the skin which are subject to paralysis or neuralgic irritation, upon the forehead when suffering from migraine, or on the paralyzed half of the body.

The treatment and prognosis of anidrosis are the same as those of the causal local or general morbid conditions.

Concerning the qualitative changes in the sweat secretion we possess very little positive knowledge, and this is not astonishing when we remember that we know very little concerning the physiological qualities of the secretion. The changes refer to vague changes of smell (bromidrosis or osmidrosis), of color (chromidrosis), or of admixture with other substances.

The necessary remarks concerning osmidrosis and bromidrosis have already been made. To repeat, in some individuals the general perspiration or the secretion of certain cutaneous regions, the axillæ and genitalia, is characterized by a specific odor, while so-called stinking sweat is due to the decomposition of the sweat impregnating the footgear. I have also discussed the statement, made by Heim, Schönlein, and others, that the exhalations from the skin possess a characteristic odor in certain general diseases, such as variola, scarlatina, typhus, etc.

The term *chromidrosis* is applied by writers to those cases in which the sweat has a yellow, green, black, or blue color. The blue color has been attributed (Scherer) to a cyanate combination analogous to Fordos' pyocyanin, to a microscopic fungus (Bergmann) whose gonidia nuclei have a blue color, also to indican and Berlin blue (Apsohn, Bizio). Yellow to orange-red sweat of the axillæ, which has marked discoloring power, is found not very infrequently, especially in red-headed individuals. The hairs are then found covered with small, firm, orange-yellow to brown nodules.

I am unable to decide whether, as Balzer and Barthelémy believe, these consist in great part of the dried secretion of the axillary glands, and whether the cocci ("erythromicrococcus") which they contain have anything to do with the color.

The anomalies which are characterized by special admixtures of a material kind include: *hæmatidrosis*, which is not a true sweating of blood, but the occasional non-traumatic exit of arterial blood from the pores of the skin. This has been observed by Finol, Schilling, Lenhosseck, Wilson, Hebra, and others. Hebra reports that he once observed a spiral stream of blood, 1‴ high, spurt from the opening of a sweat gland on the dorsum of a young man's hand. This is an evidence of the fragility of the capillaries, such as is seen in bleeders. In one case (Tittel), which occurred in an individual who was also predisposed to hæmorrhages into other organs, Wagner proved that the sweat glands were the main site of the cutaneous hæmorrhage, and in an analogous case Franque found blood globules in the escaped fluid. Gendrin and Parrot attribute hæmatidrosis to great permeability of the vessels (diapedesis) as the result of nervous influences, as in hysteria.

The term *galactidrosis* was only employed so long as the belief in "milk metastasis" and "recession of the milk" was entertained, and the puerperal process and the accompanying sweats were attributed to these conditions.

On the other hand, the assumption of *uridrosis—i.e.*, the admixture of urinary elements with the secretion of the sweat glands—is based upon positive facts. Even the older writers, who had no special knowledge of the mechanism of urinary and sweat secretion, spoke of sudor urinosus, on account of the urinous odor of the sweat. Positive findings of urea have been made, in exceptional cases, by Schottin, Drasche, Treitz, Hirschprung, Kaup, Jürgensen, etc. During the cholera epidemic of 1855 Drasche in twelve cases, and Schottin in three cases, collected scales from the integument of the face, forehead, and other parts of the body, which on microscopical and chemical examination were found to consist of urea. Similar observations by Kaup and Jürgensen were made in individuals with atrophic kidneys and in others without any disease of the bladder and kidneys. The scales referred to had appeared upon the skin one to two days before death. At all events, the presence of urea (ammonia has also been demonstrated) is an expression of the vicarious function of the kidneys and sweat glands. This relation has been established still further by the occasional demonstration of albumin in the sweat of certain individuals (Leube), of bilin, biliphæin, and urerythrin.

The majority of urophanic substances—turpentine, tar, balsams, iodine, arsenic, etc.—which enter the blood through the digestive or

respiratory tract and are excreted by the kidneys, are also eliminated in the sweat. This has not led, however, to the creation of special forms of qualitative anomalies of the secretion of sweat.

All the anomalies previously considered are independent of any demonstrable anatomical changes of the coil glands. Virchow, however, states that in phthisical patients who had suffered from profuse sweats he has sometimes found enlargement of the glands and fatty degeneration of the lining epithelium.

It is only in the last few years that any knowledge has been acquired concerning anatomical changes of the sweat glands. Generally they form a part of other histological changes of the skin, as in lupus, carcinoma, lupus erythematosus, elephantiasis Arabum et Græcorum. The findings include dilatation and enlargement of the glands in leprosy (Brücke, G. Simon), atrophy of the glands in corns (Von Bärensprung), degeneration of their epithelium in chronic dermatitis (Gay), inflammation of the adjacent connective tissue in lupus erythematosus, etc. These changes affect only the glands within the otherwise diseased territory, and not the sweat apparatus in general. As a matter of course, the glands are also involved in inflammatory processes of the cutis.

From the dermatological standpoint such findings would not have warranted us in speaking of an independent "inflammation of the sweat glands" in the sense of a *hydrosadenitis phlegmonosa* (Verneuil), and this is also true concerning the occasional inflammations, suppurations, and abscesses of the circumanal and axillary glands, which play the part of sebaceous glands. Recently, however, Giovannini and S. Pollitzer have offered clinical and histological proof, with regard to other regions of the body, of independent inflammation of the coil glands attended by the formation of tumors, abscesses, and cicatrices.

In 1889 Giovannini, under the term *idrosadenitis*, described a case which was characterized by the successive development upon the trunk and limbs of numerous little tumors from the size of a pea to that of a grape. Some of these fluctuated and opened spontaneously, others were opened by operation. Under the microscope they proved to be inflammatory and suppurative nodules of the coil glands and the surrounding parts.

S. Pollitzer (1892) described a case of *hydradenitis destruens suppurativa* and made a careful study of the literature. Painful nodules as large as a pea had developed deep in the corium upon the neck and breast. After a while they extended to the surface, suppurated, and formed cicatrices. The microscope showed inflammation of the sweat glands, due to degeneration of their epithelium. In the writer's opinion Barthelémy's cases of "acneitis," in which similar nodules appeared upon the entire body and also upon the soles of the

feet, belong to the same category as do Lukasiewicz's cases of "folliculitis exulcerans" observed in my clinic. In the latter opinion I am unable to agree.

Tumors due to hyperplasia of the sweat glands, true adenoma of the sweat gland (this includes, perhaps, Lotzbeck's "sweat gland tumor"), occur partly in a pure form (Cahen, a tumor, as large as a walnut, over the sternum in a child of eight months ; Klingel, similar tumors on the external auditory canal—"adenoma sebaceum fibrosum"), partly in combination with epithelioma or lupus. After retraction of the tumors, described by Rindfleisch as mushroom-like and spongy to the touch, cystoid degeneration of the ducts of the sweat glands takes place. In this category belong the cases described in recent years by Balzer, by Jaquet and Darier (1887), as *idradenoma ;* by Török-Unna (1889) as *syringocystadenoma ;* by Philippson (1890) as small nodules, from the size of a millet seed to that of a pin's head, pale to dark pigmented in color or surrounded by a red zone, firm, smooth, often transparent, and situated almost exclusively upon the anterior chest wall or also in the face and upon the eyelids. Darier believes that they have a genetic connection with the sweat glands. Török and Philippson attribute them to a nodular or band-like outgrowth into the corium of embryonal clumps of epithelium. This gives rise at first to small "benign epitheliomata," and then, as the result of colloid degeneration of the internal epithelium, to cavernous and cystoid dilatations which on microscopic section resemble the sweat glands. Philippson considers them identical with colloid milium (E. Wagner), and the "dégénérescence colloide du derme" of Besnier, Feulard, Balzer, and Liveing.

LECTURE IX.

ANOMALIES OF THE SECRETION OF FAT.

PHYSIOLOGY OF THE SECRETION OF FAT—PATHOLOGY—EXCESSIVE SECRETION—LOCAL AND GENERAL SEBORRHŒA—DIAGNOSIS—PROGNOSIS—TREATMENT—DIMINISHED SECRETION—XEROSIS—DISORDERED EXCRETION: ITS RESULTS AS FORMS OF PROLIFERATION, DEGENERATION, AND RETENTION—COMEDO—MILIUM—MOLLUSCUM VERRUCOSUM SEU CONTAGIOSUM—ATHEROMA.

We now come to the consideration of those diseases of the skin which consist of a pathological change of the second form of cutaneous secretion—*i.e.*, the secretion of fat.

You will remember that the fat, which is intended physiologically for the lubrication of the skin and hairs, is not produced by the sebaceous glands in the same way that sweat is produced by the coil glands. Sweat escapes from the capillaries of the papillary body and sweat gland, or is secreted by the secretory cells of the latter, whence it passes to the surface as a finished product.

The production of fat in the sebaceous glands takes place in another way. As in the regeneration of epidermis in the rete, young cells are constantly forming deep within the sebaceous glands, probably from proliferation of the epidermis cells which line the interior of the walls of the lobules. In their advance toward the lumen of the individual lobules and of the gland, a part of the protoplasmic contents of these cells is converted into fat and the cell walls become dry and brittle. The fat appears at first in small drops, which later coalesce into larger ones, in the interior of the cells. These fatty cells and their detritus are gradually pushed, by the cells behind them, into the excretory duct of the gland, and finally reach the surface of the integument. A proliferation of epidermic cells then takes place from the sebaceous glands, as it does from the rete, from whose germinal layer the glands are developed. Because the cells in their passage undergo a fatty change and crumble during proliferation, the fatty contents are set free.

Under normal conditions this cell exfoliation is no more striking than is that of the rete, and the free fat is only noticeable from the physiological lubrication of the surface of the skin and of the hairs.

In pathological cases, however, the sebaceous secretion may appear in large masses which consist almost entirely of fatty epidermic cells.

Mention has already been made of the fact that the coil glands, especially the axillary and ear-wax glands, also secrete fat in slight amounts.

The fat secretion of the skin may be abnormally changed in two ways, as regards either its secretion or its excretion.

The secretion may be either abnormally increased or diminished.

The former condition is known as *seborrhœa* or steatorrhœa, fluxus sebaceus—*i.e.*, a disease which is characterized by the escape and accumulation of abnormally large amounts of fatty secretion upon the surface of the integument. It appears upon the surface either as an almost purely oily covering, or as a deposit of very fatty scales which form thick, discolored crusts, or a thin, varnish like covering (seborrhœa oleosa s. adiposa, acné sebacée fluante of Cazenave). Or the deposited masses form fatty but at the same time rather dry, brittle scales of epidermis (seborrhœa sicca s. squamosa s. furfuracea, acné sebacée sèche of Cazenave). Both forms may occur separately or they may be combined in the same individual, and are either confined to certain parts of the body (seborrhœa localis) or are generally diffused (seborrhœa universalis). The symptoms and sequelæ of the malady differ greatly according to these conditions and also according to the location in hairy or non-hairy parts of the integument.

The scalp is probably the most frequent site of the disease (seborrhœa capillitii) in nurslings and adults of both sexes. Its product on children's heads is a yellowish-brown, variously discolored mass, which is either cheesy, brittle, and fatty to the feel, or is dry, hard, and lamellated. It covers the entire scalp in a thin layer or in large, irregularly nodular agglomerations, or it adheres in the shape of sharply defined, insular foci. If the masses of sebum are removed the scalp appears pale and moist. Within a few minutes it is again covered by a parchment-like, thin, shining coating, the product of the freshly secreted fat. Or the skin is somewhat reddened and tender, inasmuch as the corium is thin and loose; or we may find bleeding places or eczematous patches which are devoid of epidermis and secrete a serous, sticky fluid. The latter condition depends upon the macerating and irritating influences which are exerted upon the epidermis and papillary body by the cutaneous secretions which are retained and decomposed by the fat crusts. The hairs embedded in the fatty mass fall out very readily on traction, and may even fall out spontaneously after the affection has lasted a long time. In insular seborrhœal deposits this may give rise to disc-shaped baldness, which may be mistaken for alopecia areata.

The crusts develop as a continuation of the seborrhœa and more

copious regeneration of epidermis which are found over the entire body of the fetus and new-born (vernix caseosa, exfoliatio epidermidis neonatorum) during the first few weeks of life, and consist of fat, dust, desquamated epidermis, and hairs. This condition may continue until the age of two or three years. When the secretion of fat diminishes the crusts are raised from the skin, are pushed forward by the growing hairs, crumble and fall off.

Even in adults seborrhœa capillitii may be attended by the formation of massive deposits which glue the hairs together. The product of the seborrhœa sometimes appears as a shining white, lamellated, asbestos-like mass. But it occurs most frequently in the shape of thin, dirty-white, branny scales which undergo constant desquamation (pityriasis capillitii).

In adults seborrhœa of the scalp is often the outcome of previous local inflammatory processes, such as erysipelas, acute and chronic eczema, variola. The pityriasis form is especially frequent as a symptom of acute and chronic anæmia, in puerperal women, and in poorly nourished persons of both sexes. It is also very common after preceding or during still existing syphilis (seborrhœa syphilitica). In not a few cases it is an idiopathic affection without any demonstrable cause. It lasts months and years, and recovers spontaneously or as the result of treatment, either temporarily or permanently, It is always attended by loosening and abundant falling out of the hair (effluvium capillorum). When the affection lasts for years permanent thinning of the hair and baldness (alopecia furfuracea) become noticeable.

In the face the chief sites of seborrhœa are the forehead, nose, temples, and chin, and the beard in men. Many persons, especially brunettes, suffer during the period of puberty. However often the face is washed with soap, it again appears fatty and shining forthwith ; and if the air is laden with dust it also looks dirty, because the particles of dust adhere more readily to the fatty skin. The cases described as seborrhœa nigricans palpebrarum (Neligan, Wilson) and blepharomelæma (Law) refer simply to these dirty and blackish deposits of sebum. The secretion of fat is favored by heat. In many cases the fatty deposit upon the face develops suddenly. In some chloranæmic and neurasthenic individuals, men and women, facial seborrhoea appears in the form of oily, sweaty drops upon the nose and forehead, not alone after overheating, but also suddenly as the result of nervous excitement. This vicious circle may lead to considerable mental depression, inasmuch as the patients devote their entire attention to this condition, imagine themselves scrutinized by every one, and dread intercourse with others. Increased falling out of the hairs of the eyebrows and beard as the result of localized seborrhœa is a not very infrequent occurrence.

Upon the nose, adjacent parts of the cheeks, and the root of the nose, thick, dirty, yellowish-black crusts sometimes form from the desiccation and accumulation of seborrhœal products. The nose may look as if enclosed in a bag of papier maché. Such cases have often been regarded as malignant neoplasms. If the crusts are carefully removed from the edges (which is always easily done) we shall find that prolongations pass, like roots, from the lower surface of the sebum crusts into the dilated openings of the sebaceous glands. These crusts are merely the superficial accumulations of the fatty masses which press out of the follicles. Finally, the same regions, particularly the grooves of the alæ nasi, the region of the eyebrows, the concha, the hairy part of the face, are often the site of a seborrhœa sicca. The skin is moderately red and covered with thin, dry, but firmly adherent scales which are continued into the follicles. The integument, when freed from the deposit, is pale or moderately red, shining, contains larger pores (the gaping openings of the sebaceous glands), and is readily encrusted again ; in rare cases it is moist or bleeding in places. The injection of the skin is sometimes more pronounced. Hebra has described these forms as seborrhœa congestiva. It has since been ascertained that this condition may continue as such for years, but that it is often the preliminary stage of lupus erythematosus, a disease which we shall subsequently discuss in detail.

The forms of facial seborrhœa which were last described are due occasionally to a previous inflammatory process, especially erysipelas and variola. Some cases have a causal relation to the development of puberty, to anæmia following losses of blood, to febrile diseases, etc. Others do not permit of an etiological explanation, but are to be regarded as the expression of an individual peculiarity of the skin.

The complications and sequelæ of facial seborrhœa include local eczema, dilatation and inflammation of individual sebaceous glands, the development of comedos and acne, and in a few cases the process known as lupus erythematodes, which is attended by cicatricial changes in the skin. All forms of seborrhœa of the face, after lasting months or years, usually recover spontaneously or yield to suitable treatment.

Among other local seborrhœas I will first mention that of the umbilicus. Large amounts of fat and epidermis are apt to accumulate in the retracted depression of the umbilicus. This mass has a rancid odor, and the products of decomposition give rise to inflammation. Next comes seborrhœa genitalium. It is difficult to decide whether this is always due to a more copious secretion of fat or to a local accumulation of normal desquamative products of epidermis and fat. The latter explanation appears more probable in regard to

the glans penis and inner surface of the foreskin, because they contain very few glands, and the condition in question develops chiefly in cases of narrow or phimotic prepuce. The fatty and rancid secretion (smegma præputii) is especially apt to accumulate in the coronary sulcus. It may lead to painful erosions of the foreskin and glans and to the discharge of purulent secretion (balanitis, balanoposthitis).

Under analogous conditions the clitoris and its prepuce and the vulva are also the site of inflammation, a feeling of burning, and purulent discharge which simulates gonorrhœa. I have often seen the acute onset of such seborrhœa (balanitis and posthitis) in young, feeble children, and in adult females who had long been bed-ridden from disease.

Seborrhœa universalis is much rarer than the local forms. In the new-born it is represented by a greater amount of vernix caseosa, which continues to be reproduced during the first few days after birth. It encrusts the skin and gives rise to tension and the development of painful fissures. When this condition affects the entire integument the latter has a brownish-red color, even a few hours after birth, shines like silk or as if varnished, or, according to Hebra's simile, like a half-browned sucking pig. Painful fissures develop in the face, starting from the angles of the mouth, upon the joints, and in the gluteal folds. The rigidity of the nose and mouth and the painfulness of the rhagades render nursing impossible. The children die in a few days from inanition and loss of heat, unless relief is afforded by inunction and softening of the incrustations and by artificial maintenance of the temperature of the body. This condition is correctly termed ichthyosis sebacea or seborrhœa squamosa neonatorum. It is equivalent to the ichthyosis congenita of some writers, and is allied on the one hand to extrauterine exfoliatio epidermidis neonatorum (which will be described later), and on the other hand to certain forms which appear to be fœtal inhibitions of development. Under the title incrustatio seu scutulatio, Steinhausen described a case of this kind in a child belonging to the Berlin Museum. Analogous cases have been described by Sievruk, Vrolik (1854), Löcherer (1846), Kyber and Hans Hebra (1881). There are also undoubted cases of ichthyosis congenita, in the sense of fœtal ichthyosis, such as Caspary, I. and others have observed.

In adults seborrhœa universalis may occur in the form of fatty, shining scales which are constantly exfoliating and chiefly cover the trunk and the extensor side of the limbs. This is found mainly in old people, also in younger, marantic individuals, and hence is called pityriasis tabescentium. There are also rarer forms, known as cutis testacea or ichthyosis sebacea, in which the greater part of the integument, especially of the trunk and the extensor aspect of the limbs, is covered with greenish-brown and blackish crusts. Corre-

sponding to the deep grooves and lines of the skin, the crusts break up into plates and patches; in some places they are thin, in others they are heaped up and project like horns. These crusts may also be detached. Apart from moderate redness the skin appears normal, but we find numerous dilated openings of the sebaceous glands, into which the crusts extend with thread-shaped prolongations.

The *diagnosis* of seborrhœa is generally quite assured if we take into consideration the symptoms just described. At times it is attended with certain difficulties, especially in view of the manifold character resulting from the different forms, intensity, and localization of the disease. As the reader is yet unacquainted with the differential symptoms of the affections which come into question, I will confine myself to a few hints. Seborrhœa of the scalp may be mistaken for all diseases of the skin which are attended with the deposit of crusts and scales, and to which, in former times, the etiologically unmeaning term *tinea* was applied—for example, eczema squamosum and impetiginosum, psoriasis, herpes tonsurans, and favus With regard to the two latter affections, the deciding point is the exclusion of their distinctive feature, viz., the microscopically demonstrable fungus. With regard to the former, we must supplement the clinical history with the changes which are also present upon other parts of the skin.

Seborrhœa of the face must be differentiated from eczema, psoriasis, and lupus erythematodes. The latter gives rise, in addition to redness of the skin, to cicatricial retraction. In seborrhœa of the genitalia, especially when balanitis and erosions of the glans and foreskin are present, we must not forget the possibility of coincident syphilitic infection or of diabetes, in which the sugar in the urine may be the cause of the affection. Hence the prognosis should be made with caution—*i.e.*, a sufficient period of observation should be taken.

Seborrhœa universalis in the new-born cannot be mistaken, while that in the adult may be mistaken, for ichthyosis. In seborrhœa the crusts can be completely removed, mechanically and by softening, and the skin appears moderately red, speckled with large pores, but otherwise normal, elastic, smooth. The disease is curable Ichthyosis always begins in childhood and is incurable. The scales can be removed with difficulty and imperfectly, the skin is thick and warty, traversed by deep furrows (hypertrophy of the skin and papillæ), and the crusts are rapidly reproduced.

The prognosis of seborrhœa, whether general or local, is favorable. The disease can always be improved rapidly and in the majority of cases can be permanently cured. Apart from the local disfigurement, the annoyance from the tension, development of painful rhagades, and the occasional complication with eczema, comedos and

acne in the face, the disease has no bad effects on the general condition. But, as we have already mentioned, ichthyosis sebacea of the new-born may imperil life.

In the *treatment* of seborrhœa the main indication is foreshadowed by the principles which I have laid down in General Therapeutics. As we always have to deal with deposits of (secondary) morbid products. scales and crusts of fat and epidermis, the first object of treatment is their removal by softening and loosening.

They are softened and broken up most rapidly by the action of fluid fats, and are then removed by washing with soap and water.

Among the solvent fats we may recommend olive oil, cod-liver oil, petroleum, butter, lard. Additions of zinc, white precipitate, carbolic and salicylic acids, etc., are unnecessary. The oil or fat is always the chief thing. It must be used in such amounts and in such a way that the desired object is attained most rapidly and completely.

The method may be varied according to the situation and severity of the seborrhœa and the circumstances of the patient.

In seborrhœa of the scalp the oil is applied with a hard brush, a piece of sponge, or a pledget of lint, is rubbed into the crusts by pressure and friction, and the head is then covered with a flannel cap or an uncolored fez. The oil is applied in this way four or five times a day and allowed to remain over-night. Within twelve to twenty-four hours the crusts are softened in great part, to such an extent that they crumble under the fingers and may be detached. Nurslings must be handled very gently. In such cases a more prolonged course is immaterial, and very much depends upon relieving the fears and prejudices of the mother and nurse by mild and gentle measures. In adults the treatment may be aided by cutting the hair. In female patients this should be omitted, as it is unnecessary and disfiguring.

When the crusts and scales are completely softened and separable they are washed off. We may use any ordinary hard toilet soap or soft soap. If the skin is tender and delicate, as in children. glycerin soap is preferable; in adults, Hebra's spiritus saponatus kalinus. This contains alcohol, which dissolves the fat and probably has a stimulating effect on the tonus of the sebaceous glands. It is prepared according to the following formula:

℞ Saponis viridis.... .... 100 grammes.
Solve leni calore in spir. vini..... ..... 200 "
Filtra et adde,
Olei lavandulæ,
Olei bergamott ... .āā 3 "
M. Filtra. D. S. Spirit. saponat. kal.

In washing the parts use a coarse flannel cloth or a bathing

sponge, upon which the fluid soap is poured, the hard soap is rubbed into a lather. Lukewarm and cold water is to be used freely. In this way the head may be thoroughly cleaned. At the close the soap is removed by means of irrigations with lukewarm or cold water, and the head is then dried. During this procedure it is noticed that a considerable proportion of the hairs, which are matted together by the masses of fat and crusts, is detached, and that many a patient who appeared to be previously well provided with hair has become almost bald. Patients are at once inclined to attribute this loss of hair to the "powerful remedies." You are already acquainted with its real cause. The seborrhœal process itself causes loosening and falling out of the hairs. Many cases of alopecia are due solely to pityriasis capitis, but we may promise a sufficient restoration of the hair after the recovery of the seborrhœa, unless the condition has lasted for years.

The skin, when washed clean, looks moderately red and shining, and grows more tense the more it dries. The resulting disagreeable sensation, the fissuring of the thin corium, and the regeneration of the sebum deposits are prevented by the application of oils or pomades. For example, olei olivar. 50, bals. peruv. 1; or ung. emoll. 25, zinci oxid. 0.50, olei baccar. lauri gtt. 5. After the corium has been restored to sufficient thickness at the end of a few days, and the skin has lost its tenderness, the scalp must be brushed for several weeks with spir. vin. gall., either pure or with the addition of acid. carbolic. 0.15 : 100 (or acid. boracic. 3, or acid. salicyl. 3). This should be done daily or three times a week, according to the amount of the newly produced scales.

As the soaps and alcoholics remove the fat from the corium and make it brittle, it is advisable to employ from time to time some bland oil or fat. This after-treatment may be necessary for weeks or even months.

Balanitis is not relieved by mere frequent washings, as is often recommended. It is better to insert charpie or strips of linen, dipped, for example, in amyl. oryz., talci veneti, āā 50.0; kaolini pulv., magnes. carb., āā 5. If weeping, raw places are present the strips of linen may be dipped in astringent solutions or ointments, such as aeruginis 0.15, aq. font. 25; or plumb. acetici basici 0.50, aq. font. 30; or unguent. emoll. 20.0, zinci oxidi 0.25.

After this description of the local treatment of the more frequently observed forms of seborrhœa, very little need be said concerning other localizations, especially upon hairless regions of the skin. In all cases softening of the deposits, detachment and washing, and the subsequent use of alcoholics with occasional washing and anointing, are necessary to success. The mode of application depends upon locality and circumstances. For example, thick crusts

on the face are removed most rapidly by the application of oiled cloths or pieces of linen smeared with ointment or simple plaster, and by keeping these in position by means of less permeable material.

The so-called dusting powders, such as amylum, talcum venetum pulverizatum, kaolin, tannin, etc., are occasionally serviceable—for example, when inserted between the prepuce and glans, for dusting upon a face smeared with pomade in order to avoid the glistening appearance, etc.

General seborrhœa is treated in a similar manner. A child suffering from cutis testacea must be vigorously rubbed with oil or grease, or wrapped in cloths smeared with a bland ointment. This must be done methodically, the limbs, the toes, face, etc., being wrapped in separate cloths, which are kept in position by a flannel mask and bandages. In order to maintain the temperature of the body the child is kept in poor conductors of heat, such as down and woollen coverings. It should be washed daily with soap in a warm bath, and, after drying, again treated with oil.

Similar treatment is adopted in ichthyosis sebacea of adults. In order to soften the crusts the patient is anointed for several days with soft soap or cod-liver oil and placed in woollen blankets, or he wears flannel or rubber clothing. After the crusts are softened, treatment with daily baths, soaping, douches, and again inunctions is continued until the skin has regained its normal structure.

In view of the fact that some local seborrhœas, especially of the head and face, may be due to remote causes, anomalies of other organs or of the general nutrition (particularly chronic gastricism and chlorosis in women), the treatment, in part internal, must also be directed against those causes. Bitters, such as gentian, rhubarb, alkaline and ferruginous waters, iron, etc., are useful, in addition to dietetic and climatic measures, and must be continued for a long time in order to prevent relapses of the seborrhœa.

The internal administration of cod-liver oil or morrhuol (one to two capsules daily) is useful in the seborrhœa sicca universalis of scrofula and tuberculosis.

There is very little to say concerning the diminished secretion of fat, or *asteatosis cutis*. The skin which is deficient in physiological lubrication has a dry, fissured, occasionally finely desquamating corium (*pityriasis simplex*). This condition is rarely idiopathic and independent. It is generally a part of some other congenital skin disease—for example, xeroderma, ichthyosis, prurigo; or of some acquired disease, like elephantiasis Græcorum, psoriasis, lichen ruber. It is rarely general, but is confined to larger or smaller patches of skin. It may be permanent or temporary and changeable like the causal skin disease.

Asteatosis cutis is often produced artificially by the influence of agents which permanently withdraw too much fat from the epidermis. This is done by soap and lye upon the hands of washerwomen, by chemicals in certain trades. The palm of the hand usually exhibits a thick and brittle, inelastic and fissured corium. The patients keep the fingers flexed, and complete extension, even passive, cannot be performed. In people who daily wash the entire body with cold water containing lime salts and saltpetre, the skin also becomes scaly, dry, and destitute of fat; pruritus and eczema are not infrequent results.

The duration and curability of asteatosis cutis depend upon its causation.

We know of no mode of treatment which will stimulate the activity of the sebaceous glands. In addition to the removal of the primary cause, the cure of the coincident affection of the skin, and the avoidance of everything which removes fat from the skin, the sole object of treatment is to supply the integument with fat by inunctions of cod-liver oil, lard, etc. But as all fats irritate the skin as soon as they become rancid, they must be frequently removed with soap and water. Hence it is best to recommend inunctions of vaseline or lanolin.

We will now consider a few interesting forms of skin disease which are due to disordered excretion from the sebaceous glands—anomaliæ excretionis glandularum sebacearum—or forms of fat retention. They are due to the fact that the secretion of the sebaceous glands, epidermis and fat, is not carried externally, but is retained within the excretory duct or the gland itself. With the secretion of the sebaceous glands the downy hairs, which are physiologically exfoliated, sometimes remain in the excretory duct.

The attendant conditions are manifold, in part very complicated, in part entirely unexplained.

The simplest conditions are those of mechanical interference with excretion. When the common excretory duct of the hair bulb is plugged by foreign bodies, such as tar or dust, or that of the sebaceous gland is obliterated by cicatrices, the retention of the secretion is readily understood. Inasmuch as the sebaceous glands continue to secrete epidermis cells and fat under such circumstances, the retained products will mechanically dilate the excretory duct and gland. The chemical changes in these products will irritate the gland and may induce more abundant proliferation and inflammation. These are the simple retention forms from mechanical causes when the excretory duct is occluded, as in tar comedos, milium in the vicinity of cicatrices, and some atheromata.

The same forms may also occur when the excretory ducts are

open. We are then compelled to assume, in addition to hypersecretion (proliferation), a qualitative change in the sebum as the cause of its retention. This is so much the more plausible because, as a matter of fact, the enclosed epidermis masses exhibit, under such circumstances, a different chemical constitution from the normal secretion, as in milium and molluscum sebaceum.

Instead of undergoing the physiological fatty changes, the secreted cells become cornified, like those of the rete (as in ordinary milium), or they undergo colloid degeneration, as in colloid milium, or amyloid or hyaloid degeneration, as perhaps in molluscum contagiosum. Both conditions prevent the destruction and evacuation of the secreted cells.

In many conditions, especially in impaired nutrition (chloranæmia, scrofula, etc.), I would regard as a third predisposing factor of the retention of fat a diminished tonus of the cutaneous muscles, the arrectores pilorum, which send branches to the sebaceous glands.

From the dermatological standpoint the chief forms are comedo, milium or grutum, and molluscum verrucosum, sebaceum, or contagiosum. Atheroma, cholesteatoma, and cryptoliths belong rather to the domain of surgery.

Comedones (acne punctata) are dirty, whitish-yellow to brown or black specks in the skin, from the size of a needle point to that of a pin's head, which correspond to the free openings of the ducts. They form the visible extremity of a plug which fills the common excretory duct, and rarely project much above the level of the skin. Upon lateral pressure the plug is forced through the opening in the shape of a sinuous body. With the dark-colored upper extremity, like a head, it resembles a worm, and hence the term fleshworm.

The usual site of comedones is the integument of the forehead, nose, temples, chest, and back. They are sometimes present in enormous numbers, scattered or arranged in groups of two (double comedo), three or four (multiple comedo, Ohmann-Dumesnil), or even in wart-like, nodular masses (sebum warts, Hebra; comedo discs, Ribbentrop). They are also found in other parts of the body, especially upon the integument of the penis, very rarely upon the glans (E. Lang).

Now and then a comedo develops in every one. After lasting a longer or shorter period the plug is loosened and is expressed by renewed secretion or by mechanical pressure and friction while washing. The opening of the gland gapes for some time. Numerous and persistent comedones constitute an annoying and disfiguring ailment. Although the individual comedones are discharged and very rarely leave punctate cicatrices as the result of obliteration of the follicles, yet the disease appears to remain stationary in some

individuals on account of their continued recurrence, and may be very distressing in social and business intercourse.

As a rule it develops in males and females at the period of puberty; in the former it often lasts to the age of twenty or thirty years, while in the latter it commonly disappears at an earlier period. It is often combined with seborrhœa oleosa faciei, and also gives rise to inflammatory acne as a result of the irritation exerted by the substances retained in the gland upon surrounding parts.

The *causes* of the development of comedones are in part the same as those of facial seborrhœa (chlorosis, cachexia). Occasional causes are occlusion of the mouths of the glands by tar and dirt (tar factories), and insufficient cleansing of the skin with soap and water when the secretion of fat is copious.

It is difficult to find a cause for comedones which develop apart from such conditions. The most natural plan appears to be to look for the cause in the anatomical relations.

The comedo consists of a peripheral covering, formed of epidermoidal cells, which encloses a mass of fat (cholesterin), fatty and broken epidermis cells, together with downy hairs (three to twelve) and acari folliculorum. In old, inspissated, dry, and brittle comedo plugs I have often found the bodies which are described as peculiar to molluscum. If the fat is extracted by the aid of alcohol and turpentine, the hairs and epidermoidal elements are left over, and particularly the peripheral part of the plug, in the shape of a tulip-like pouch. The cells composing the latter are derived from the mucous layer of the excretory duct and the remains of the root sheath; the constituents of the interior of the comedo. with the exception of the downy hairs, are derived from the sebaceous glands.

Unna ascribes the blackish-brown color of the head to ultramarine as a normal element of the cutaneous secretions, but this has been denied (Krause). In my opinion cornified epidermis and fat are always discolored in this manner when they remain in contact with the air for a long time, and the dirt from the atmosphere aids in producing the color.

From these findings, as well as from the clinical appearances, the comedo appears to be situated in the excretory duct of the sebaceous glands or in the common duct of these and of the hair bulbs.

In the favorite *sites* of comedones, the forehead, nose, back, etc. (which are also the localities for lanugo hairs), the conditions are such that the sebaceous glands open free with a wide excretory duct. The hair follicles form an appendage of the sebaceous glands and empty at an obtuse, occasionally at a right, angle into the excretory duct of the sebaceous gland. The tip of the hair coming from the hair bulb impinges against the opposite wall of the excretory duct, and sometimes even rolls downward (Fig. 16, Biesiadecki). In this way it may

cause irritation at this point and induce proliferation of the lining epithelium of the duct, and the covering of the sebaceous contents is thus produced. This would also explain the development of comedones at the period of puberty, because it is well known that a more vigorous growth of hair takes place at this time. The lanugo hairs are produced and exfoliated more rapidly. While the hairs growing from the follicle cause irritation, the older hairs, which are shed physiologically from the papilla, enter the wide excretory duct of the sebaceous gland and remain here in the mass of cells, cellular detritus, and fat, and form part of the comedo (Fig. 16, *b b'*).

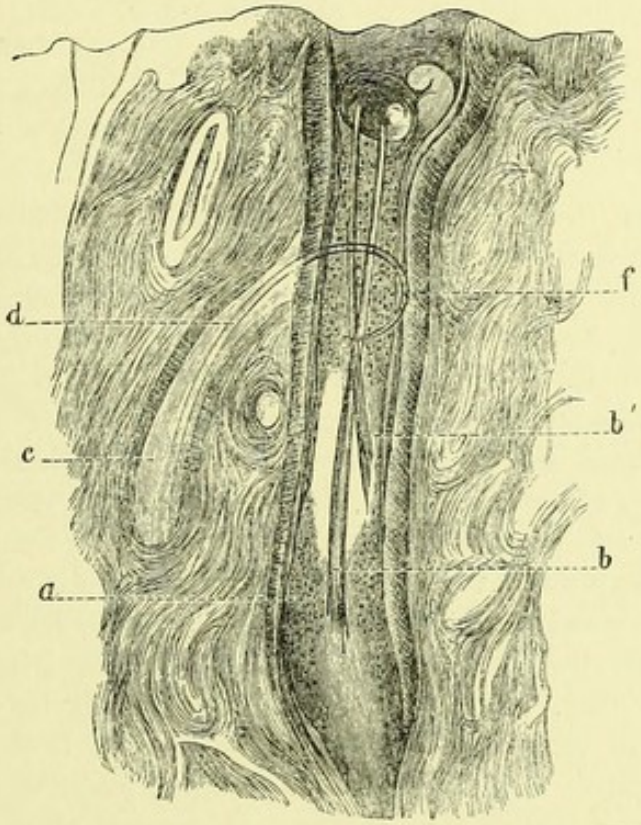

FIG. 16.—SECTION OF A COMEDO.

a, duct of a sebaceous gland, filled with secretion; *b b'*, two lanugo hairs contained in the duct; *c*, small hair follicle opening into it; the hair (*d*) impinging against the opposite wall of the duct and turning downward.

In other parts of the body—for example, the limbs—where the sebaceous glands empty into the hair follicles, the excretory duct of the latter is common to both and is the site of comedones.

The development of double and multiple comedones depends upon a communication of two or more adjacent excretory ducts. Ohmann-Dumesnil explains this by compression atrophy; Török, by previous operative or suppurative destruction of the septum.

We have already said that the mechanical plugging of the excreory duct by tar, dirt, etc., may give rise to the development of come-

dones. I am inclined to believe, however, that diminished tonus of the walls of the duct is responsible in all these cases.

The *treatment* of comedones consists in their removal. This is done by squeezing them between the two thumb nails, or we may employ Hebra's so-called comedo expressor. This consists of a conical metallic tube, four centimetres long, whose narrow end has a blunt rim, and the upper part has two lateral eyelets. The narrow end of the instrument is pressed rapidly upon the skin, vertically over the comedo, and the latter is thus forced into the cavity of the tube. Useful instruments have also been devised by Piffard, O. Simon, and Unna. In addition we employ antiseborrhœal remedies—viz., washing with soap, application of alcoholics, etc.—in order to diminish the secretion of fat and stimulate the tonus of the glands. Good results are also obtained from the modes of treatment employed in acne (to be considered later), which is usually associated with comedones.

Milium, or *grutum*, consists of rounded, spherical little bodies, of a yellowish to milky-white color, of the size of grits to that of the head of a pin, which are scattered through the skin or project slightly, shine through the corium, and are firm to the touch.

Their main site is the delicate integument of the eyelids and their vicinity, the cheeks and temples; next the border of the lips; upon the male genitals, the penis and scrotum, but particularly the corona glandis, which is sometimes encircled by milium granules; finally, the female genitalia, especially the inner surface of the labia minora.

If the integument over a milium granule is slit with a fine knife the spot will bleed moderately, and the granule as a whole may be expressed with the thumb nails from its nest or lifted out with the tip of the bistoury. It sometimes adheres firmly by a narrow pedicle to the skin (the hair bulb), and this must first be torn. The granule is round, finely lobulated, smooth, and can be easily compressed, when it splits into scales. It consists of a simple or lobulated peripheral envelope, a delicate membrane, with contents of dry epidermis cells. These are arranged, like an onion, around a central epidermoidal, fat-containing nucleus, like the so-called cancroid corpuscles. The latter, however, contain proliferating cells.

The milium consists of a single sebaceous gland or of several lobules of a gland, and hence is always covered by a thin layer of the corium with its papillæ and the rete. These must be incised in order to remove it. For this reason the epidermis accumulating within the gland or its lobules gives rise to distention. Philippson and Robinson also assume that in some cases the milium is not situated in preformed cavities, but is unconnected with sebaceous glands, hair follicles, or covering epithelium, and is composed of concentrically arranged horny cells which are embedded free in the connective tissue and are unmixed with fat.

The *causes* of this affection are varied. When it develops upon a healthy skin and the excretory ducts are patent, there is no reason to assume a mechanical interference with the excretion of the glandular secretion. It appears as if a chemical disturbance takes place, so that the cells, instead of undergoing a fatty change and then crumbling—this is favorable to their evacuation—simply become cornified, like the cells of the epidermis, and remain stationary.

A similar condition seems to obtain in certain superficial inflammatory processes of the skin. Bärensprung, Hebra, and I have observed the rapid development of many hundred milium granules during the course of an attack of pemphigus in a man, and I have seen them in a child, æt. six years, suffering from pemphigus (in the places where the pemphigus vesicles had healed), and also in a man after the termination of an attack of erysipelas. They were arranged in delicate groups and rings on the arm, the dorsum of the hands and fingers, and the integument of the abdomen. After a number of weeks, in these cases, the granules exfoliated in part, and some may have remained permanently.

On the other hand, a purely mechanical cause may be assumed for those milium corpuscles which develop at the edges of cutaneous cicatrices, whether the latter follow lupus, syphilis, or burns. Here some of the lobules of the glands are evidently shut off from the excretory ducts by the cicatricial bands, and the cells, which continue to be secreted for a while, accumulate in their cavities.

When the sebaceous gland empties into the hair bulb the milium occasionally forms a cyst-like dilatation of the latter. Indeed, Virchow and Rindfleisch expressly state that the hair bulb is the site of milium—the opening according to Virchow, the fundus according to Rindfleisch. The latter view is probably incorrect, in view of the remarks we have just made concerning the site of milium and its mode of development.

The *treatment* of milium is especially desired by female patients whose faces, particularly if the complexion is delicate, are disfigured by a large number of milium granules. The best remedial procedure is incision of the skin over each nodule with the tip of a fine bistoury and then expression of the milium The point of incision bleeds very little and heals without leaving a trace. When there is an acute and enormous development of milium, as has been described, in cases of pemphigus and erysipelas, I produced redness and moderate inflammation of the skin by the application of soft soap, and as a result the milium granules rapidly exfoliated. Hence I conclude so much the more positively that in these cases there was free communication between the milium nodules and excretory ducts. If this communication were obliterated, then at the most the granules could

only be discharged in time as the result of atrophy of the overlying thin layer of the corium.

Mention may be made of the rare colloid milium described by E. Wagner, in 1866, in a woman of 54 years. The forehead, nose, and adjacent integument of the cheeks and temples were traversed by longitudinal and transverse ridges, upon whose summit were situated numerous firm nodules as large as grits and which looked like vesicles. They could not be ruptured by the strongest pressure. It was only after the overlying layer of skin was pricked that the contents appeared as a pale yellow, homogeneous, dully glistening, translucent mass which resembled firm colloid. According to Wagner these were milium granules whose epidermic contents had undergone colloid degeneration. There were no recognizable epidermis cells, although a few delicate hairs were found. In speaking of tumors of the sweat glands (page 120) it has already been mentioned that Philippson considers Wagner's case and all other analogous cases (six) as formed, not from the acini of the sebaceous glands, but from embryonal collections of epithelial cells which had been left over in the corium.

I will now discuss the so-called *molluscum contagiosum* (Bateman), or *molluscum verrucosum*, as a pathological condition which naturally belongs in this category, although from a purely anatomical standpoint it must be included among the benign epithelial tumors.

Under the term molluscum, Bateman originally described tumors which undoubtedly consisted in part of distended, cystoid, degenerated sebaceous glands, with thickened walls, filled with fluid fat and epidermic débris; the openings of the glands were obliterated or visible, even permeable for probes (sebaceous-gland tumors). These might be properly termed molluscum atheromatosum. In fact the term "molluscum" was also applied to connective-tissue tumors of firm consistence, which will be considered later under the term "fibroma molluscum." At a later period Bateman recognized a "peculiar variety" of molluscum, which he described and pictured as round, smooth, shining, often transparent nodules, from the size of a pin's head to that of a pea. From the larger ones pressure discharged a "milky" fluid through a hardly discernible opening. To this form he afterward (text to his Plates, xxxi., 1817) applied the term molluscum contagiosum, because he inferred from his cases that the disease was communicable between children and their nurses and mothers, and that probably the "milky fluid" was the carrier of contagion.

Molluscum contagiosum appears upon the skin in the shape of round, hemispherical, white, shining, almost transparent, warty

projections or little tumors. They vary from the size of a pin's head to that of a pea, and are occasionally surrounded by a narrow red zone. Even the smaller ones, and still more distinctly the larger ones, present in the middle an umbilicated depression which corresponds apparently to the mouth of a follicle. They look very much like a varicella eruption, with which they are apt to be confounded.

If such a nodule is compressed between the thumb nails the contents escape and leave a shallow depression whose base bleeds considerably. The lesion consists of several round, smooth, white lobules, which are attached to a short stem like a little bunch of grapes. Sometimes the entire lobulated nodule escapes in such a way that the epidermic covering remains intact, perforated merely by a small central opening, and remains connected with surrounding parts. It is only after the firm covering bursts that the nodule can be compressed between the fingers. We then find a mushy, lamellated mass which consists, under the microscope, of finely divided, flat epidermis cells, fat globules and crystals. In addition it con-

FIG. 17.—"MOLLUSCUM CORPUSCLES," PARTLY OR WHOLLY CONTAINED IN THEIR EPITHELIAL COVERINGS, SOME ENTIRELY DENUDED.

tains large, ovoid, non-nucleated, peculiar dull glistening bodies, partly free, partly filling an epidermic covering; sometimes inside, sometimes outside the latter (Fig. 17). These bodies are known as "molluscum corpucles." These have been carefully studied, because, since Henderson and Paterson called attention to them, the opinion has been generally entertained that they are peculiar to molluscum and are the carriers of the contagion, which had already been assumed by Bateman.

The nodules are usually quite numerous. They are found upon the penis and scrotum and the labia, and for this reason a relation to clap has been assumed; also upon the trunk and limbs, particularly on the flexor surfaces, upon the face and neck, very rarely on the scalp and the mons veneris. They occur singly or in large numbers (twenty, fifty, one hundred, or even more), of various sizes, disseminated or closely aggregated in places, or forming nodular tumors as large as a quarter or even larger (Lutz, Geber, Vidal).

Very little is known concerning their development, except that they usually appear imperceptibly. They are generally found upon

the patient as completed structures. More rarely we have an opportunity of observing their gradual development from very small red papules. They run a chronic course, lasting weeks, months, or years. Many of the smallest ones disappear. The large ones are occasionally scratched and fall out from the bleeding base. Others are exfoliated by painful inflammation and suppuration of surrounding parts, and leave a scar. As we have already remarked, others continue unchanged for years. Their presence is unattended by pruritus or pain, apart from those which undergo inflammation. They are more frequent in children than in adults, and their development appears to be favored by eczema, prurigo, profuse perspiration, maceration of the skin. Under such circumstances I, as well as other writers, have observed an acute development over large areas.

The numerous theories held concerning these structures are evident from their varied nomenclature. They have been described under the terms subcutaneous and endocystic condylomata, condyloma porcelaneum, sebum warts (Hebra), molluscum epitheliale (Virchow), acne varioliformis (Bazin), molluscum verrucosum (mihi), epithelioma contagiosum (Neisser), acne molluscum contagiosum (Vidal and Leloir). The notion of their contagious character was originally called forth and has been maintained by the fact that they have been repeatedly known to develop at the same time or in rapid succession in several persons, especially children, who were intimately associated with one another. The question has been decided by the positive experimental inoculations which were carried out by Retzius, Vidal, Haab, and recently by Pick (1891), in an exact manner, free from all objections. Hence the contagious character of molluscum is proven clinically and experimentally, and the adjective "contagiosum" is justified. Still the question of the carriers of contagion is as yet undecided. As a matter of course, its solution was attempted by histological examinations, but hitherto, as it seems, without any decided results.

Upon transverse section (Fig. 18) the molluscum exhibits a lobulated structure, like a sebaceous gland : a connective-tissue limiting wall, which sends into the cavity septa, principal and secondary, and laminated epithelioidal contents. For this reason the majority of the earlier observers, and recently Vidal and Leloir, have regarded the structure as a distended sebaceous gland with proliferated and peculiarly changed epithelioidal contents. I, and formerly Virchow, believe that the epithelial proliferation which leads to the formation of molluscum contagiosum begins in the rete of the excretory duct of the follicle. On the other hand, a large number of observers (Retzius, Bizzozero, Manfredo, C. Boeck, Lukomsky, Thin, Taylor,

Geber, Caspary, Sangster, etc.) attribute it to proliferation and lobular outgrowth of the interpapillary rete cells.

If we examine under the microscope the laminated epithelioidal contents of a lobule which opens toward a central cavity (Fig. 18), it is found to consist peripherally of "enchyma" cells arranged in columns, and, further toward the centre, of cells whose protoplasm, beginning in the vicinity of the nucleus, becomes homogeneous and shining (according to Renaut this change is keratinic; according to

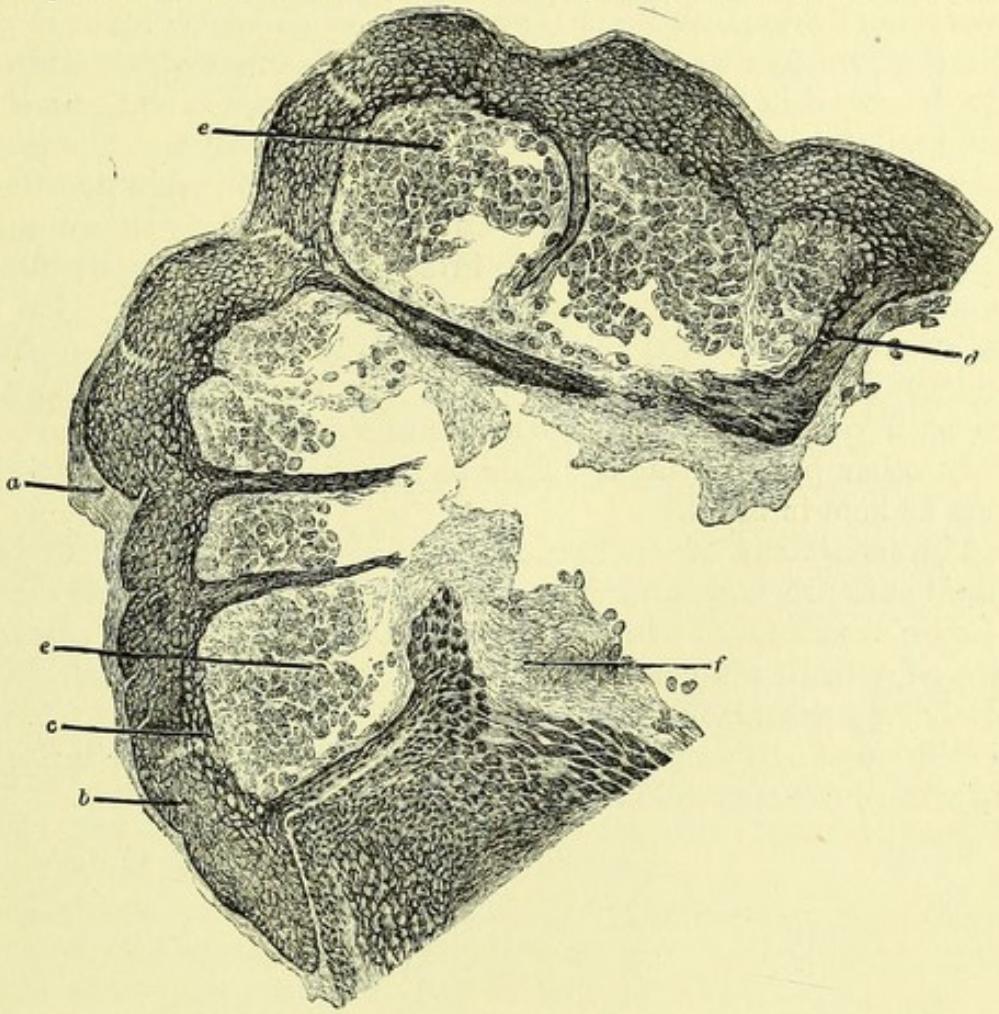

FIG. 18.—MICROSCOPICAL SECTION OF A NODULE OF MOLLUSCUM CONTAGIOSUM.

a, capsule of connective tissue ; *d*, the septa ; *b*, unchanged, *c*, altered epithelium ; *e*, molluscum corpuscles ; *f*, fibrin.

Török and Tommasoli it is due to a peculiar degeneration). The outer zone of the cells is cornified or has been made rigid by some special chemical change. The bodies, dull or shining, in the substance of the enchyma or rete cells, in the deepest layers, are the above-mentioned molluscum corpuscles. The closer the cells approach to the centre of the lobule the larger these corpuscles appear, until finally they fill the distended cells completely, the latter rupture, and the molluscum corpuscles then project partly from the cell or are free in the cavity.

While they have generally been regarded as the peculiarly degenerated protoplasmic portion of the epithelial cells, Bollinger stated that they probably consisted of migrated gregarinæ, basing his opinion on the assumed identity of molluscum contagiosum and so-called "fowl-pox." In the latter the gregarinæ are unmistakable, but Neisser and I believe that they are not identical with the molluscum contagiosum of man.

Nevertheless Neisser, who was corroborated later by other observers (Touton), adduces numerous reasons which make it probable that the molluscum corpuscles are gregarinæ whose migration into the rete cells induces the epithelial proliferation and tumor formation, but are themselves hindered in their typical development by the extensive cornification of the surrounding epithelial mantle and are thus made to undergo degeneration. Accordingly the gregarinæ would constitute the carriers of infection from one individual to another. Vidal and Leloir also describe a network due to the normal cornification of the other rete cells, and this is said to enclose the "gregarinæ" on all sides. Up to this time, however, no proof has been offered that these corpuscles are living organisms, and every other possible mode of clinical and experimental infection must be kept in mind.

The *treatment* of molluscum nodules is mechanical. The individual ones are expressed with the thumb nails, or, if their number is large, are removed with the sharp spoon. The bleeding places are covered with Bruns' cotton and rapidly heal. If many mollusca are closely aggregated, shrinking and exfoliation may be produced by the application of soft soap or some other substance which gives rise to a superficial, diffuse inflammation.

CLASS IV.

INFLAMMATORY DERMATOSES.

SKIN DISEASES DUE TO EXUDATION AND INFLAMMATION.

LECTURE X.

GENERAL REMARKS ON EXUDATION AND INFLAMMATION.

EXUDATION AND INFLAMMATION IN GENERAL—CELL FISSION: ITS RELATIONS TO THE FORMER AND TO THE FIXED AND WANDERING FORMED ELEMENTS—SYMPTOMS OF CUTANEOUS EXUDATION AND INFLAMMATION: THEIR COURSE AND TERMINATION—RESOLUTION, SUPPURATION, HYPERTROPHY, ATROPHY, DEGENERATION.

GENTLEMEN: I am about to make you acquainted with a series of skin diseases which, on account of their great frequency, will occupy you more than all other dermatoses in your practical career.

Varying extremely in their appearance, course, causation, and significance, they are all based upon a common anatomo-pathological foundation—viz., exudation and inflammation. They constitute exudative or inflammatory processes—κατ' ἐξοχήν. We must therefore consider the significance of these processes with regard to general pathology and to the integument in particular.

You are aware that the conception of inflammation has always dominated medical studies. Its symptoms were first observed upon the skin, as is shown by its traditional characteristics—viz., redness, heat, swelling, pain, and impaired function. From the observations made upon the living inflamed skin the conception of inflammation was also extended to the internal organs.

For two thousand years the only attempts to explain the nature of these symptoms were speculative in character. Pathological anatomy first sought for the character of the inflammatory process in the material changes of the inflamed tissues.

Until the fifties of this century Rokitansky and his pupils regarded the exudation in the inflamed tissues as the essential anatomical symptom of inflammation, and the exudative process itself as equivalent to inflammation. The latter was supposed to be inaugurated by a disorder of circulation which began as hyperæmia, increased to

stasis, and then led to the escape of exudation. The latter characterized the height of the inflammatory process. The formed elements—cells, nuclei, "exudative corpuscles"—in the exudation or inflammatory product were supposed to develop from the plasma of the exudative fluid, as it were by spontaneous generation. At the same time it was held that all these elements were incapable of further development. The exudation was differentiated from another discovery in the inflamed tissues—viz., the inflammatory new-formation, the tissue vegetations. This was not regarded as an attribute of the inflammatory process itself, but as a sequel. The inflammation proper reached its typical termination with the exudation. The tissue vegetation was produced from the pre-existing connective tissue elements (vessels as well), the result of the stimulus of the exudative fluid, and hence constituted a sequel, a termination of the inflammation. The pus cells whose development accompanied the inflammatory tissue vegetation (granulations) were attributed, like the corresponding elements of the original exudation, to a renewed exudation which escaped from the new blood vessels that kept pace with the formation of granulations.

It is thus seen that the inflammatory process, which was supposed to end, on the one hand, with the original exudation, was assumed, on the other hand, to continue, inasmuch as the pus formation of the granulations was derived from a new exudation. This exudation, of course, could only be the product of an inflammation.

The discovery that the fixed connective-tissue elements are capable of proliferation, and Virchow's theory of cellular pathology which was based upon this fact, drove the previous conception of the significance of the exudation into the background. The appearance of formed elements (cells, pus) and the formation of permanent tissues in the inflamed parts were attributed to proliferation of the connective-tissue corpuscles. The exudation—*i.e.*, its fluid part—could not be overlooked, however, and it was also evident that it was derived from the blood vessels. Nevertheless its escape was not explained by the action of the vessels or heart; it was supposed to result from the proliferating vegetation of the tissue elements (Virchow). Attention was thus diverted from the processes in the vessels, from the circulatory disturbance and its direct consequence, the exudation, to the processes in the tissue elements themselves.

For a time the attention of histologists was held by a series of newly discovered phenomena in the tissue processes. In the first place, by Recklinghausen's discovery of the wandering of pus cells (exudate cells); further by Cohnheim's demonstration that during the inflammatory process swarms of white blood globules pass from the lumen of the vessels through their walls and migrate into the tissues; Stricker had already shown that red blood globules occa-

sionally follow the same course, and Waller made the same observation in regard to white blood globules.

Cohnheim's discovery of the migration *en masse* of the white blood corpuscles was identical with the proof of an inexhaustible supply of pus cells. White blood globules, pus cells, exudate corpuscles, cannot be distinguished from one another and must be regarded as identical. Virchow's doctrine was thus assailed, especially as he had furnished no real proof that the connective-tissue corpuscles divide and that the pus cells develop from them. The migration of white blood globules by diapedesis could be observed under the microscope by every one.

Facts soon accumulated with surprising rapidity. Thorough investigation proved that the pus cells develop by endogenous formation ; furthermore, that the epithelial cells also produce pus cells by endogenous formation, and also proliferate by division of their nuclei and protoplasm (Buhl, Rindfleisch, Oser, etc.) ; finally, that wandering cells may pass from the connective-tissue spaces into the epithelial layers.

The proliferating power of the connective-tissue corpuscles, which had been doubted by Cohnheim, has received undoubted demonstration at the hands of Recklinghausen and Stricker-Norris (for corneal corpuscles and the connective-tissue corpuscles of the tongue). Cell fission in inflamed tissues was also proven for the muscular elements, nerve cells, the elements of the vessel walls, bones, tendons, etc. In short, observation taught that the living elements are in part capable of movement, in part become capable, and that they divide and proliferate. A better knowledge concerning the internal processes in division of the cells and nuclei has been obtained in recent times, thanks to the labors of Heitzmann and others, but especially of Flemming and Rabl. These conditions, introduced into recent pathology as "karyokinetic" phenomena, will be discussed with the processes of wound-healing and cicatrization.

It was not owing to this general cellular proliferation that the clinical doctrine of inflammation received its most fatal blows. It had long been known that such proliferation does and must occur, although its intensity and extent were not appreciated. The most damaging features for the clinical doctrine were the biological independence manifested in the productivity of the individual elements ; the theory that the tissue elements react to a direct stimulus by proliferation ; and the fact that these phenomena within the tissues occur independently of the general nutritive processes, particularly of the local circulatory conditions to which we had been accustomed to refer all nutritive changes.

Virchow himself, the founder of the new pathology, had not left vascular action entirely out of consideration. On the contrary, as

required by clinical observation, he postulated hyperæmia as the cause of the inflammatory nutritive disturbance. He also emphasized the fact that not every hyperæmia is followed by inflammation. Nor did he undervalue the importance of the exudation, but he regarded it as the result rather than the cause of the tissue changes. Cohnheim's discovery pointed distinctly to the disturbance of circulation. The migration of white blood corpuscles took place under its influence. Nevertheless, in following their migration and subsequent fate, and in the interest excited by the proliferation of the fixed connective-tissue elements, the relation of the blood vessels to all these processes was forgotten.

This one-sided view, which was destructive to clinical doctrines, seems to have been abandoned. On the one hand, the statements concerning the proliferating activity of the stable connective-tissue elements have been confirmed and supplemented by new experiments. On the other hand, the exudation has again been relegated to its proper place in the series of inflammatory processes, and stress laid upon the implication of the vessel walls in the process (Samuel, Stricker, Billroth, Ziegler).

The way to further advance has been paved by the investigations on lymphagoga (Rogovier, Haidenhain), on the differences between transudation and lymph, and on the dependence of the latter upon the (secretory) function of the tissue cells (Klemensiewicz). It must be assumed, accordingly, that the circulatory disturbance which follows a direct irritant or one conveyed through the nerve tracts always inaugurates the process of inflammation. Then an exudation of fluid and formed elements from the blood vessels takes place, and the nutritive disturbance in the tissues develops, either at the same time or as a result of the exudation. The exudation—or preferably the exudate—is not alone necessary as food or building material for the newly produced elements, but its fluid portion, as we may infer from Stricker's experiments, also serves as a mechanical irritant for the stable tissue cells, inasmuch as it stimulates them to new vital activity, to proliferation.

The chain of phenomena is thus completed and restores the clinical picture of inflammation. The temporary interruption of its continuity has been serviceable to the whole. The individual links in the chain have been taken into the different workshops, and there, in accordance with actual facts, have been rewrought, strengthened, and again welded together.

Upon the general integument the previously considered histological signs of inflammation, the circulatory disturbance, the exudation, and the nutritive disturbance of the tissue elements appear

clinically with unusual distinctness and are attended by the following symptoms:

1. *The Circulatory Disorder as Hyperæmia.*—It appears as redness, of varying depth and extent, which disappears under the pressure of the finger. It is an expression of the increased fulness of the smaller and smallest vessels; as such it is associated with increased heat in the parts.

Alone, the hyperæmia does not constitute the initial symptom of inflammation, but only in relation to a subsequent exudation. The latter only supplies the previous hyperæmia with its significance. In other words, not every hyperæmia leads to exudation—*i.e.*, not every hyperæmia forms part of inflammation. This fact, which is recognized by all physiologists and experimenters (Brücke, Virchow, O. Weber, Billroth), must be especially emphasized from our standpoint.

There is a hyperæmic condition of the skin which leads to exudation very rarely or not at all. Not alone the absence of exudation, but the fact that such hyperæmias run a typical course, stamps them as special forms of disease. Hence, in order to lay due stress upon the clinical facts, we have arranged these hyperæmias in a special category (first class of skin diseases). Inflammatory redness is either diffuse and extends over larger areas, or it is confined to individual points, mainly in the vascular tracts of the cutaneous glands. Whether the hyperæmia involves only the superficial papillary vessels, or also extends to the vessels of the corium, is also important.

Finally, the circulatory disturbance may run an acute or chronic course. In the former event the rise of temperature is often considerable (to 41° C. or more); in the latter it hardly varies from the normal.

2. *The Exudation*—the escape of fluid (serum) and the corpuscular elements (white blood cells) of the blood from the vessels into the tissues.

It is an evidence of inaccurate clinical observation to ignore the importance of "exudation" and the "exudate" in the internal processes of inflammation. For this reason I am well satisfied to be accused of adhering, in this respect, to the standpoint adopted by Hebra in 1844. Indeed, it would be very desirable if our knowledge were not confined to the mere fact of exudation, but if it could also be extended to the differences in the chemical constitution of the exudate in different processes. That such differences exist is placed beyond doubt if we bear in mind the differences which the exudations of urticaria, eczema, erysipelas, dermatitis, pemphigus, etc., manifest in their appearance, course, and influence upon the tissues.

The exudate distinguishes a large series of processes from others

likewise inflammatory, and we cannot overlook it either as an objective sign or as a clinical criterion.

In the corium the exudation is manifested by swelling (increased volume), and, if more developed, by infiltration which is painful on pressure; at the surface it is shown by loosening and projection of the epidermis in the shape of little nodules and vesicles whose contents consist of exudate; after detachment of the epidermis the fluid appears upon the surface in drops or greater quantity. This fluid shows the chemical and microscopical characteristics of inflammatory exudation. Under such conditions the exudate appears as a yellowish-white, sticky fluid of a feebly alkaline reaction. A large amount of albumin is precipitated by boiling or the addition of nitric acid. The microscope shows a larger or smaller number of formed elements (a few red, more white blood globules, free nuclei). In the air it dries into yellowish, brownish, honey-like or gum-like crusts.

As a rule the swelling, infiltration, and free exudation correspond in localization to the causal hyperæmia, sometimes diffuse, sometimes confined to individual points. In the latter event the exudation appears chiefly around the openings of the different follicles, or escapes from the openings, after it has taken place, into the lumen of the glands or follicles.

The amount of exudation is not always directly proportionate to the intensity of the hyperæmia. Profuse exudations appear in apparently slight hyperæmias, and *vice versa*.

The question of the further fate of the exudate can only be answered in so far as it can be separated from the nutritive disturbance which it inaugurates. Rapidly developed exudates which are poor in formed elements are occasionally absorbed so rapidly that they could hardly produce any noticeable disorder of the nutritive conditions of the tissues (erythema exsudativum multiforme, urticaria). Under such conditions it is difficult to assume any active implication of the stable tissue cells (proliferation of the connective-tissue corpuscles). When the exudate is more abundant or richer in cells and remains for a longer time within the tissue spaces, or when it is frequently renewed, or under circumstances which cannot be clearly defined but are due to the nature of the process in question, a tissue change results which is more or less recognizable clinically and is manifested by proliferation of the tissue elements.

The exudate, with its constituents (serum and migratory cells), is not alone employed as available material in the proliferation of the tissues, but also takes part actively by the division of its cellular elements. The further fate of the exudate is intimately connected with that of the inflamed tissues. In other words, the nutritive disturbance is identical in both and can only be regarded as a whole.

3. *The Nutritive Disturbance* of the inflamed tissues. Upon

the skin this is manifested clinically by the infiltration of the corium, the formation of nodules, either over an extensive area or confined to solitary points. In the more superficial layers it appears as an excessive formation, accumulation, and exfoliation of the epidermis and the epidermic structures of the cutaneous glands (desquamation, also increase of pigment); in all of the tissues of the skin this process may consist of loosening, softening, and destruction of its elements with the symptoms of suppuration, tissue necrosis, gangrene, or it occurs as hyperplasia or atrophy, with or without the phenomena of retrograde metamorphosis.

Microscopical examination shows that the first nutritive disorder of the inflamed skin consists, apart from the separation of the individual tissue elements by the fluid exudate, in the production of numerous cellular elements which accumulate within the tissues attacked by inflammation (cellular infiltration). The newly formed cells are generally round, oval, or spindle-shaped bodies, as large as a white blood globule, with a large, strongly refracting nucleus; some possess two or more smaller nuclei and finely granular protoplasm. They are derived in great part from the blood vessels, whose walls are morbidly changed in the inflammatory process and therefore more easily traversed by the blood globules. Hence they appear in the immediate vicinity of the capillaries and in the adventitial spaces of the vessels at the beginning of inflammation (and also in many neoplasms). In a measure, however, they are derived from fission of the cells and their nuclei, or of the exuded corpuscles, and also from the former fixed tissue elements, connective-tissue corpuscles, the parenchymatous epithelial cells of the glands, the epithelial cells of the Malpighian layer.

All the new-formed cellular elements (young cells, inflammation cells, granulation cells, infiltration cells) exhibit the biological characteristics of the white blood globules which have migrated from the blood vessels into the tissues—*i.e.*, they are capable of movement and are sometimes found remote from their place of origin. Those produced in the corium may be found within the Malpighian layer (wandering cells).

Inflammation may terminate in various ways. After the cellular proliferation and the infiltration of the skin have lasted a longer or shorter time, the termination may be in

(*a*) *Resolution.* The process gradually subsides and the skin returns to the normal condition.

The hyperæmia first diminishes, and with it the exudation is also lessened. This checks the supply of material and the stimulus to proliferation of the fixed tissue elements—*i.e.*, to the new formation of cells.

The fluid and cellular exudate in the tissues gradually disappears.

When situated on the surface, in the epithelial layer, it disappears partly by evaporation and desiccation. The mechanically loosened and detached epithelial cells are cast off as dead bodies; those within the corium are probably removed by absorption. The cellular elements may be absorbed either in their integrity or perhaps after fatty degeneration.

(*b*) *Inflammatory hypertrophy.* The mobile, amœboid cells, after the diminution of the hyperæmia and exudation, are converted into fixed elements—in the corium, into connective tissue. vascular structures, etc.; in the epithelial layer they increase the number of cells. This hypertrophy is a frequent result of chronic inflammation, such as chronic dermatitis, eczema, psoriasis, chronic (lymphatic) œdema, and is apt to be associated with the various forms of degeneration and retrograde metamorphosis which will soon be described.

(*c*) *Suppuration.* This termination is characterized clinically by the acute softening and destruction of the tissues, attended by the production of pus, a thick, greenish fluid.

Pus has a feebly alkaline reaction, like every exudation, and consists of an albuminous fluid (pus serum) in which cellular elements (pus corpuscles) are suspended. The appearance and structure of the pus cells correspond with those of exudation cells. But, on the one hand, they are present in relatively larger numbers than in the so-called exudates, and on the other hand a larger proportion are provided with two or more nuclei and with fatty granules. Hence it may be said that pus is an exudation in which cellular proliferation has been unusually acute and abundant. In fact, a clear, transparent exudate (for example, in a pemphigus vesicle) may become purulent and cloudy when the proliferation of its cellular elements becomes excessive.

Two questions have always engaged the attention of pathologists: 1. Whence is pus derived? 2. In what way does the destruction of the tissues, noticed in suppuration, take place?

The first question is answered when the pus is put on the same plane with the exudate. Like the latter, the fluid elements of the former are undoubtedly derived from the vessels, and its cells partly from the vessels, partly from the fixed tissue elements. The rapid increase is due to proliferation of the lymph cells. This is shown by the frequent finding of multinuclear pus cells which are undergoing division. More positive proof would consist of the demonstration of karyokinetic phenomena in them, as will be shown later in regard to the epithelial cells.

The manner in which the tissues are destroyed has been explained at different times in different ways. Formerly pus was erroneously supposed to be able to cause destruction of the tissues by contact. At the present time the destruction of the tissues in suppuration

is otherwise explained. The softening and destruction of the cell forms must be regarded as a part of the process of cell proliferation and pus formation, not as its result. The connective-tissue corpuscles and the epithelial cells produce young cells, pus cells, and their substance is lost in this very new formation. As Stricker expresses it: "Many tissues lose their functional peculiarities as a result of the inflammatory process, and are so modified in their biological character as to become fitted for productive purposes—*i. e.*, they become mobile, increase in size, and undergo complete or partial division" Purulent destruction is accordingly the expression of the acute extensive cell proliferation.

With regard to the destruction of the fibrous part of the connective tissue, recent investigations have shown (as Virchow hinted at an earlier period) that the bundles of connective-tissue fibres are traversed by fine processes of the cells (the connective-tissue corpuscles), which form an anastomosing network. During inflammation these processes, like the cell bodies themselves, swell up, probably at the expense of the intercellular gelatin-forming substance of the fibres, and finally occupy the entire tissue space. It is found that this substance disappears in proportion as the processes of the cells become thicker and the meshes of the network formed by them become narrower. Finally pure pus is produced. This process has been demonstrated in the cornea, the tendons, and also in the tissue of the cutis (Ravogli).

Some of the pus cells are washed away by the after-coming exudation, others are desiccated or are distended by imbibition and burst. Other parts undergo fatty degeneration and are absorbed. Finally, the youngest cells, which have suffered least from chemical and mechanical injuries, which are found upon and in the area of the suppuration, in the basal tissue of the wound (plasmatic layer of Thiersch), return to their normal stability as the inflammatory proliferation diminishes, and are converted into fixed tissue elements.

Losses of substance in the corium, due to suppuration, are replaced by young cicatricial (connective) tissue, and the young elements reproduce connective tissue, blood vessels, and nerves, although not in their physiological grouping.

Losses of substance which involve the epithelial layer alone (herpes, pemphigus) heal without a cicatrix, because the restitution of homologous cellular structures alone is requisite in such cases.

Mention may here be made of the other possible terminations of inflammation: necrobiosis in the form of gangrene, which is distinguished from purulent destruction of the tissue elements by the necrosis of larger masses of tissue; progressive molecular destruction of the tissues in fibrinous exudation (croupous, diphtheritic inflam-

mation); the various forms of atrophy and degenerative destruction of the tissues, such as fatty, waxy, mucoid, and colloid degeneration, cheesy degeneration, and calcification of the inflammatory products and infiltrated tissues. I will content myself with their mere mention, because they will again be considered when we meet with them among the diseases of the skin, and their significance is already known to you from your study of pathological anatomy.

The very large majority of skin diseases belong to this class, which is characterized by exudation and inflammation. In all these forms we will not alone find the phenomena of exudation and inflammation in the clinical picture, but can also demonstrate them with the microscope.

There are no anatomical differences among the various forms of this category, except as regards the intensity, spread, and special localization of the inflammatory change. In some processes it occurs chiefly in circumscribed foci, in others it is diffuse; in some it affects only the upper layers of the corium, in others its entire thickness, in still others it involves chiefly the glands and surrounding parts. The finer histological conditions are approximately the same in all forms, in the papule of variola as in the nodules of eczema, prurigo. lichen urticatus, and psoriasis, in the vesicle of herpes as in that of eczema, etc. This fact diminishes the importance which some have attached to histological details. They afford us an instructive insight into the finer changes of the tissues, and hence some explanation of the clinical phenomena, but offer no differential signs between the various analogous (inflammatory) diseases of the skin.

The latter differ very materially, however, in their clinical characteristics, in their causation, course, appearance. significance, and results as regards the skin and the general system. Upon this basis the inflammatory dermatoses are arranged in various groups and subdivisions, and the study of the individual forms is facilitated.

In order to give a preliminary view of the exudative dermatoses, I will here mention that they are naturally divided into two principal groups, one which runs an acute, typical course—*i.e.*, according to a definite and well-known rule—while the members of the other group run in the main a chronic course

Among the acute exudative processes the so-called "acute exanthemata" (measles, scarlatina, and variola) form a natural group of contagious diseases, as opposed to another group of non-contagious acute forms. Among the latter one series is characterized by the predominance of vaso-motor changes of vascular tonus (angioneuroses), which occur clinically as mere redness or associated with moderate serous exudation—erythema forms : erythema multiforme, erythema nodosum, pellagra, acrodynia, roseola, urticaria. In a

second series of acute diseases with a typical course the inflammation is excited by a neuritic disturbance and the exudation occupies the foreground, inasmuch as it leads to the development of vesicles : herpes facialis, progenitalis, zoster, iris et circinatus, miliaria, pemphigus acutus In the third series the signs of inflammation (redness, heat, swelling, pain, and all their possible terminations) receive full expression—true dermatitides or inflammations of the skin. The forms included in this category may be produced by chemical, traumatic, or dynamic causes—idiopathic dermatitis (dermatitis traumatica, venenata, calorica)—or they may be the symptom of a local or general toxæmia due either to bacterial organisms or to certain chemical poisons. The latter may be of animal origin, in the broadest sense, and may be derived partly from the human body, partly from animals—infections of the skin : erysipelas, furuncle, anthrax, pseudo-erysipelas, cadaveric infection pustule, and the pure zoonoses, malignant pustule and glanders. The inflammation may attack only the superficial layers of the skin and terminate in resolution, as in the ordinary forms of erysipelas ; or it may penetrate to the deeper layers and lead to suppuration, as in the phlegmonous variety. This may be diffuse as in the latter process, or circumscribed as in furuncle.

In addition to the large number of acute inflammatory forms there is an equally large number of chronic dermatitides, which may be arranged in several subdivisions, according to special characteristics. There would be no advantage in enumerating them at the present time. It will be preferable to arrange them in a comprehensive scheme after you become thoroughly acquainted with all the different varieties.

We will now investigate the acute inflammatory processes of the skin, and first the acute contagious forms, the so-called "acute exanthemata."

A. ACUTE EXUDATIVE DERMATOSES.

I. ACUTE, CONTAGIOUS, INFLAMMATORY DERMATOSES.

LECTURE XI.

ACUTE EXANTHEMATA.

THE CHARACTERISTICS COMMON TO THE ACUTE EXANTHEMATA—MEASLES.

MEASLES, scarlatina, and small-pox constitute the group of the so-called acute exanthemata, or exanthemata *κατ' ἐξοχήν*. These forms of disease are produced by specific contagion, which, in addition to an affection of the entire organism and an acute, febrile, typical course, are characterized by specific changes in the general integument and a regular rotation of the local and general symptoms.

The coincidence of the general symptoms of the exanthemata is manifested in the following points :

1. Their *contagious* character, inasmuch as they develop solely as the result of infection and are communicable to others.

2. From the moment of infection to the outbreak of the disease there is a certain interval of relative well-being (*incubation*).

3. The outbreak of the eruption is preceded by *fever*, whose intensity and duration bear a certain regular proportion to the former.

4. The changes in the general integument, apart from their specific anatomical character, exhibit a regular *type* as regards course, development, duration, and recession.

5. During and after the eruptive stage the involvement of the entire organism is shown, apart from the fever, by numerous other accompanying disturbances and sequelæ, so that the process is stamped as an *infectious* disease.

6. The acute exanthemata often occur in *epidemics*, because the contagion is regenerated n the individuals who are attacked and is conveyed to others.

7. As a rule they attack an individual only once.

The characteristic changes in the general integument, the cutaneous eruption, forms, from a semiotic and pathological standpoint, the main feature of the disease, around which the other symp-

toms are grouped in regular order. This enables us to fix certain boundaries in the course of the eruption, which define the various stages. These are usually described as follows :

1. The period of *incubation*—the interval between the demonstrated or assumed infection and the occurrence of the first morbid symptoms.

2. The *prodromal* period. This is manifested by the first febrile outbreak and its attendant symptoms. It continues until

3. The period of *eruption*, which begins with the appearance of the characteristic cutaneous eruption. This passes directly into

4. The *florid* period, which corresponds to the duration of the fully developed cutaneous affection. With the recession of the eruption begins

5. The period of *desquamation*, *exsiccation*, or *decrustation*, which passes into convalescence and recovery.

On account of the acute course of the exanthemata the various stages last but a few days, although there is considerable variation, according to the character and intensity of the process and according to idiosyncrasy. The stage of decrustation in variola sometimes lasts for weeks.

Despite the striking similarity in their general characteristics, there are three specific forms of exanthemata, each of which possesses a distinct contagion and a special pathological character. To a considerable extent, indeed, they exclude one another, both individually as well as epidemically.

With regard to the latter, it is a matter of experience that when one disease is epidemic the other is absent or occurs only spasmodically in the same territory, and that epidemics of measles, scarlatina, and variola follow one another. It is also true, as a rule, with regard to the individual, that he cannot be attacked by two exanthemata at the same time, while other skin diseases, such as eczema, psoriasis, scabies, and even constitutional and infectious exanthemata (syphilis), may occur at the same time with measles, scarlatina, or variola.

Of late years, however, many observers, especially pædiatrists, have reported the coincident occurrence of two acute exanthemata—measles with scarlatina, or variola with scarlatina. In such cases the measles and scarlatina do not develop at the same time, but, for example, while the measles eruption was still recognizable, violent fever suddenly developed, followed on the third day by a distinct scarlatinal eruption in addition to the other symptoms of that disease, so that the fever must be regarded as the prodromal fever of the second exanthem. In all cases there was a rapid succession of the two exanthems, but in reality they were coincident. The contagion of the second exanthem, in view of the average period of incuba-

tion, must have been present in the organism during the development of the first exanthem.

Like other physicians, Hebra had observed the succession of two acute exanthemata, one developing as soon as the other had disappeared, but he did not observe the coincident existence of two exanthemata as is claimed by many pædiatrists. There is no doubt that many of these statements are founded on error, inasmuch as an increased development of an already fading eruption, or a complicating erythema or urticaria, was regarded as a second exanthem. But all these reports of cases cannot be combated in this way, although no instance has come under my observation. The possibility of the coincidence of two acute exanthemata is not to be denied, however, because when they rapidly follow each other the two contagions must have existed in the organism at the same time.

MEASLES, MORBILLI.

Measles (*morbilli, rubeola, rötheln, rougeole,* etc.) is an acute, contagious disease with a typical course, which is characterized by the appearance of separate, irregular red patches and nodules upon the general integument. It is attended by fever and catarrh of the respiratory passages. The disease was long known in literature (Rhazes), but Sydenham and Morton (1670–1674) were the first to furnish a thorough description.

The principal *symptoms* are the eruption, fever, and catarrh of the respiratory passages. The symptoms develop according to a well-defined, regular type. The more the course of the disease corresponds to this type the more normal it is regarded. There are also abnormal cases which exhibit notable deviations from the normal type.

The beginning of the disease must be reckoned from the time when the individual absorbed the infectious material, either directly or indirectly, from a measles patient. This begins a period of *incubation*, which passes unnoticed, as a rule, on account of the absence of symptoms. It lasts on the average eight days; in rarer cases it is more prolonged, even to twenty-one days. The average duration of incubation has been determined in part in cases in which the source of infection was accurately known, in part in experimental inoculations of the measles virus by means of the lachrymal or nasal secretion of measles patients. In rare cases incubation is signalized by a feeling of malaise, and toward its close, and immediately before the beginning of the second stage, there may be a moderate and atypical rise of temperature.

The *prodromal* period is characterized by fever and catarrh of the mucous membranes. It begins with fever, with a chill followed by a hot stage and the well-known febrile symptom-complex—a feeling

of dulness, pains in the joints, headache, a hot, dry skin. with a temperature of 39°–40° C., rarely vomiting. Initial convulsions are extremely rare in view of the fact that the disease occurs chiefly in children. By the second or third day the fever has disappeared almost entirely or there are moderate nocturnal exacerbations, so that the patient appears to be perfectly well.

On the other hand, there is an increase in the catarrh of the mucous membranes, which has attacked the upper portion of the respiratory tract. This is pathognomonic of the approaching measles. It begins upon the nasal mucous membrane, then attacks the conjunctiva, the mucous membrane of the palate, pharynx, and larynx. Frequent sneezing, injection and chemosis of the conjunctivæ, a feeling of pressure in the frontal region, are the first symptoms. These are soon followed by an abundant flow of tears, photophobia, increased secretion, and occasionally hæmorrhage, from the nasal mucous membrane. The nares are often plugged, the face swollen, the lids œdematous. Dark-red dots and patches and large vascular tumefactions often appear on the soft palate, and to the expert furnish a characteristic sign of the oncoming measles. Tonsillar swelling and difficulty in swallowing are usually insignificant. A dry, hollow, even barking and spasmodic cough, with moderate expectoration of mucus, and hoarseness, reveal the catarrh of the larynx and trachea. At this time the eruption is still absent.

After the prodromal stage has lasted three to five days, rarely longer (even eight to ten days in anæmic, scrofulous children), the stage of *eruption* begins with a renewed exacerbation of fever and the characteristic outbreak upon the skin.

The eruptive fever constantly rises to 40° or 41° C. It remains at this height until the next stage, and, as a rule, reaches its maximum at the height of the eruption on the second or third day—*i.e.*, about the fifteenth day after infection. With the fever there is also an increase of the general symptoms, the swelling and redness of the face and the catarrh of the respiratory passages.

The eruption appears first on the face, upon the forehead and temples, rapidly extends (within twenty-four to thirty-six hours) over the neck, back of the head, trunk and shoulders, and appears last upon the limbs, being more profuse upon the extensor aspect, especially over the joints.

In the *florid* stage (fourth to sixth day of the disease) the fever and eruption attain their maximum. The former subsides soon after the eruption has reached its height. At this time the eruption has ts deepest color, and continues to spread over the trunk and limbs, while it remains stationary on the parts previously attacked. The palms of the hands and the soles of the feet always escape. The acme of the eruption hardly lasts longer than twelve to twenty-four hours.

The measles eruption appears in the shape of bluish-red to yellowish-red, flat or somewhat raised patches, as large as a finger nail, and growing pale under the pressure of the finger ; or fine, red nodules which correspond to the mouths of the follicles, and which resemble the papules of variola. In places the patches coalesce over larger areas, but normally colored streaks and patches of skin always persist between the extensive spots of redness. General confluence is never observed. The edges are sometimes jagged and irregular, often semicircular, but always distinctly marked. In the new-born it is sometimes observed exceptionally in the shape of fine red points. Where the patch pales under the pressure of the finger the skin has a yellowish tinge, which becomes darker with the duration of the eruption. It is accompanied occasionally by moderate burning or itching. After the eruption has remained at its height for a few hours it begins to pale, as a rule in the order of its development, and leaves a yellowish-brown to brown pigmentation.

In the meantime the fever has notably subsided. The swelling of the face and the catarrhal symptoms have also diminished in severity, the least improvement being noticed in the larynx and trachea. The disease thus passes into the stage of *desquamation*, which is non-febrile and is accompanied for a while by the constantly diminishing catarrhal affections. Sleep and appetite return.

Upon the integument, moderately moist from perspiration, branny desquamation appears at the former sites of eruption, most distinctly on the uncovered parts, the face, neck, and hands. This often continues more than two weeks, counting from the beginning of the eruption, and is occasionally accompanied by decided itching. The patient has then recovered completely.

This is the normal course of measles.

Cases also occur in which there are anomalies of one or another symptom. These may either lead to mistakes in diagnosis or prognosis, or they may be dangerous in themselves, or, on the other hand, of no significance. These anomalies may be due to individual peculiarities, to special conditions of the organism, particularly anæmia, tuberculosis, and scrofula, or to unfavorable external conditions, such as miserable quarters, poor food and nursing. The anomalies may also be due to the peculiarity of the individual epidemic. In such an event they are often observed and form part of the peculiar character of the epidemic.

Thus the eruption may appear first on the trunk and other parts of the body instead of the face, or it may be incompletely developed. Instead of lasting two to three days it may continue unchanged for seven to ten days, and is then especially dark and leaves more pigment. During the crisis it may suddenly fade and then reappear at the end of one to three days (secondary measles). This should not

be mistaken for another attack of measles, in which there must be an interval of several weeks, or at least the entire desquamative stage, between the first and second eruptions. The sudden disappearance of the eruption is always the result of a febrile complication, not its cause. If the complication rapidly subsides the eruption may reappear. This will not happen if the complication—for example, a pneumonia – lasts a considerable time. Inasmuch as dangerous, or even fatal, complications often cause the rapid disappearance of the eruption, it is easily understood that this symptom is regarded as an unfavorable omen. The statement that the efflorescence may reappear during desquamation is incorrect, and arises from confusion with erythema and urticaria.

Finally, the eruption may be entirely absent. while all the other symptoms—the normal type of fever, the catarrh of the mucous membranes, and the opportunity for infection, such as contact with measles in other members of the family or the prevalence of an epidemic—warrant the assumption that the case is one of measles. We then speak of *febris morbillosa sine exanthemate ;* when the eruption is present, but the febrile symptoms are wanting, we speak of *morbilli apyretici.*

There are also deviations from the normal in regard to the morphological character and the intensity of the eruption. In addition to the two varieties of eruptive elements of normal measles there are also vesicular measles, confluent measles (which have a favorable prognosis), and hæmorrhagic or black measles. A few petechiæ—*i.e.*, spots due to an extravasation of blood and not disappearing under the pressure of the finger—may occur in every profuse eruption, associated with the ordinary patches. They constitute merely an expression of the changes in the vessel walls which attend every active hyperæmia, and their greater permeability or fragility in places. In hæmorrhagic measles, however, such extravasations of blood predominate. They then indicate a dangerous condition, because they are usually part of the general disposition to hæmorrhages and are associated with profuse and obstinate hæmorrhages from the nose (rhinorrhagia), stomach, and intestines, bloody sputum (an evidence of lobar and lobular pneumonia), with general decomposition of the blood and corresponding febrile symptoms. At first the pulse is full and frequent, but soon becomes weak and thready. Unconsciousness and rapid collapse set in and generally lead to a fatal termination. These hæmorrhages are a frequent symptom of typhoid measles—*morbilli typhosi, s. nervosi, s. putridi.* This anomaly of the clinical history belongs to the florid stage.

Even if the eruption is not hæmorrhagic the general course may be changed by increase of the individual symptoms or complications of all kinds, and may give to the process a typhoid character. As a

rule this typhoid measles is accompanied by enlargement of the spleen and copious evacuations from the bowels.

Among the complications of measles, severe and exhausting epistaxis sometimes occurs during the prodromal stage. When the eruption is hæmorrhagic the epistaxis may continue during the florid stage and constitute the prelude to a typhoid condition, with high fever, dry tongue, and signs of cerebral pressure ; or it may be the forerunner of scorbutus, which may be left over after severe measles An allied condition is shown by stomatitis and noma, with extensive gangrene of the buccal mucous membrane, septic pulmonary infarctions, and pulmonary gangrene. Laryngitis, so-called false croup, belongs to the prodromal stage, and is characterized by hoarse, barking, or spasmodic cough, occasionally by inspiratory spasm. This condition is merely the expression of an intense catarrhal affection of the larynx and disappears with the outbreak of the eruption. Croupous or diphtheritic laryngitis, true croup, occurs during the florid stage of eruption or during desquamation, is shown by renewed increase of fever, and is characterized by the well-known membranous deposits on the pharyngeal mucous membrane and by barking, hollow cough. In the majority of cases it proves fatal by suffocation, dissolution of the blood, pulmonary complications (croupous pneumonia and bronchitis), or cerebral complications.

Next to catarrhal or croupous disease of the trachea and bronchi, pneumonia is the most frequent complication of measles. The lobar variety occurs chiefly at the height of the disease, or even at the onset ; the lobular variety is a frequent sequel. With the development of the latter the eruption becomes pale. The majority of pulmonary inflammations run a favorable course, unless they are a continuation of croup.

Miliary tuberculosis with the symptoms of acute hydrocephalus occasionally occurs even at the beginning of measles, and rapidly proves fatal.

With regard to the complication of measles with other skin diseases, those which had formerly been present, such as chronic eczema and psoriasis, partly disappear during the measles, or they recover entirely, or they may reappear during convalescence. Urticaria, erythema, or a few large vesicles (Steiner) are occasionally observed.

As in the case of other blood poisons, it is difficult to furnish an exhaustive enumeration of all the possible complications and sequelæ. Among the more frequent sequelæ are *ozæna, chronic laryngeal and pulmonary catarrh, intestinal catarrh, ophthalmia, chronic inflammation of the glands* at the angle of the jaw, of the bronchial and mediastinal glands (Widerhofer), terminating in suppuration or cheesy degeneration, scrofula, anæmia, etc. *Renal affections* are extremely rare, while *whooping cough* is very common.

The anatomical changes in the eruption of measles are very simple. From the clinical viewpoint they consist of injection of the finest vessels situated around the mouths of the follicles, or of those of various groups of papillæ, with moderate serous transudation. Post mortem these signs disappear. Changes in the epidermis and papillary layer which warrant an inference of cellular proliferation have not been found hitherto with the microscope; and this is easily understood, in view of the slight intensity and brief duration of the clinically visible changes. M. Simon mentions greater prominence of the part of the cutis corresponding to a nodule of measles. Mayr and Hebra explain the nodules as an inflammation of the sebaceous follicles. It seems to me that they correspond mainly to the excretory ducts of the hair bulbs or sebaceous glands, but that in many places they are due to swelling of individual papillæ and the overlying rete. This also agrees with the investigations of J. Neumann. In measles combined with petechiæ or vesicles the anatomical changes are similar to those found in other processes. The changes in the blood and internal organs found post mortem correspond to the complications observed in the clinical history.

The *cause* of measles resides in a peculiar contagium, in exposure, and the predisposition to its reception. Until recent times the contagium of measles could not be demonstrated physically. It was assumed to be of organic nature, perhaps a vegetable organism like the cocci or bacteria. P. Canon and W. Pielicke claim to have found, in the patients of P. Guttmann's wards in Berlin (1892), the bacillus of measles in the blood, nasal mucus, sputum, and conjunctival secretion, and they regard it as probably identical with the one previously described by Cornil and Babes. This finding has not yet been corroborated by other writers.

The contagium of measles, like that of all contagious diseases, regenerates and proliferates in the infected organism, which thus becomes a source of infection to others. The carriers of the measles contagion are the excretory products of the catarrhal mucous membranes, the secretion of the nose, the sputum, tears, and also the blood (Home, Speranza, Katona). Experimental inoculations with blood have produced an outbreak of the disease after the normal period of incubation. The exhalations from the lungs and skin during the prodromal and eruptive stages also carry the contagion, which is thus diffused through the atmosphere. Opinions differ in regard to the production of infection during the period of desquamation.

The predisposition to measles is approximately equal in all individuals, but is absent in those who have had the disease. Still numerous cases have been reported in which two or even three attacks were experienced at intervals of several weeks or months or years. Children in the first year of life and old people seem to have

a slighter predisposition. The greatest number of cases occur between the ages of two and ten years, so that measles is pre-eminently a "children's disease."

Acute febrile diseases do not protect against infection with measles, but its outbreak is usually delayed under such circumstances until the former have run their course. Pregnant and puerperal women are greatly endangered by the disease. Cases have been reported in which the child had an eruption of measles at birth when the mother had been attacked by the disease about the time of parturition.

Infection occurs from direct contact with a measles patient or from inhalation of the latter's exhalations. The period of time necessary for infection seems to vary greatly, according to individual predisposition and the intensity of the contagion. The latter seems to vary according to the epidemic and the individual case. It is certain that a very fleeting contact may suffice. The contagium adheres to clothing and utensils, and may thus be carried by healthy persons to others. But in this respect, as well as regards its duration of life, the measles contagium appears to be less active than other viruses, so that the patients and dwellings do not seem to preserve their infectious character beyond the duration of the last case of measles.

On account of its direct and indirect infectiousness, measles, as a rule, attacks all the children in the family who have not already suffered from the disease, and it thus spreads to a large number of others. It occurs, therefore, in epidemics. In large cities sporadic cases are hardly ever absent, and more extensive epidemics develop every three or four years. In regions which are remote from the paths of travel, and in which an epidemic has not prevailed for a long time or not at all—*i.e* , when many or all of the inhabitants have not suffered from the disease—measles, when once introduced, attacks the larger part of the population, old and young. Although the same contagium must be assumed for all epidemics, certain ones are characterized by special mildness of the symptoms, others by their severity and dangerous results.

Catarrhal affections of the air passages, bronchial catarrh, influenza, and especially whooping cough have often been observed as the forerunners of an epidemic of measles, and such affections usually persist after the subsidence of the epidemic.

The *diagnosis* is based upon the combination and the regular mode of development of the catarrhal symptoms, the fever, and the eruption. The latter is very similar to the prodromal eruption of smallpox, which is also preceded by catarrh and fever. Hence a cautious physician will make a diagnosis of measles on the first day of the eruption only when the catarrhal symptoms and the known exposure to the disease favor this opinion. Otherwise it is preferable to wait

until the next day. In a case of variola the nodules would then have developed more distinctly or have been converted into vesicles, while the nodules of measles do not change in this way. Scarlatina presents an entirely different appearance. Miliaria and roseola papulosa have a great resemblance to measles. In the former, however, the catarrh and fever are absent or they are very moderate, and the fever does not run the regular course as in measles.

In recent times a differential diagnosis has also been made between measles and *rötheln*. Since the sixties this term has been applied more frequently than in former times to an acute, contagious, often epidemic eruption in children which closely resembles measles but is attributed to a different contagion. In 1877 Buchmüller reported an extensive epidemic in our immediate vicinity, but the symptoms do not differ from those of mild measles. The transition of such forms into true measles has not alone been demonstrated by the opponents of the theory of their specific character (Kassowitz), but has also been acknowledged by its advocates (Gerhardt). Its occasional occurrence in children who have already suffered from measles is not surprising, inasmuch as characteristic measles may attack the same individual two or three times. These facts and my own observations have led me, as well as Hebra and many others, to regard the eruption known as rötheln as belonging to measles.

I must admit, however, that Kassowitz, who formerly denied the specific character of rötheln, has recently made observations which tend to overthrow this opinion. Even men of great experience, like Henoch, hesitate to express an opinion on the subject.

At times the symptomatic roseola of various diseases, such as typhoid fever or erythemata, especially so-called drug eruptions, may be mistaken for measles.

The *prognosis* of ordinary measles is favorable. Recovery is always to be looked for in normal cases and epidemics, and in otherwise healthy individuals.

In complicated cases the prognosis only becomes serious or absolutely unfavorable in so far as the complications exert an untoward influence on the course of the disease, either *per se* or on account of the individual organization of the patient. In regard to the febrile complications I subscribe fully to Thomas' statement: "The prognostically important anomalies are unusually high fever and delay in its critical resolution, unusually abundant and highly colored as well as anomalous eruption, unusually severe catarrhal affections, finally complicating diseases of the internal organs or a bad general condition." Great importance must be attached particularly to the severity and course of the fever. The value of temperature measurements lies in the fact that they afford the best and most certain

means of distinguishing normal from anomalous cases, of ascertaining the onset of anomalies and complications, and of judging their significance.

The prognosis also depends on individual and epidemic influences. Children in the first year of life, old people, and those already ill, especially pregnant and puerperal women, are put in the greatest jeopardy. Certain epidemics also furnish a large number of severe complications and fatal results, while in others almost all cases run a normal, typical course. The mortality thus varies very much, from one to five per cent or more. The most disastrous are seen in those epidemics which attack a population previously free from measles.

Death as a direct result of measles rarely occurs in the prodromal stage, more frequently during the florid stage, in so-called typhoid measles, *morbilli asthenici, synochales.* It may also occur in all stages, and even long after the disease has run its course, as a result of the complications and sequelæ previously described.

The best *treatment* of normal measles is that which avoids all unnecessary and useless measures. Rest, a well-aired room at a temperature of 14°–15° R., moderately darkened in case of photophobia, a regimen suitable to the fever and the subjective symptoms, form the best plan of treatment. In excessively high temperature of the body cold sponging, or even methodical wet packs, may be employed. Recession of the eruption need not be feared as the result of such measures ; its sudden disappearance is due to the occurrence of some serious complication.

Daily washing of the body and change of linen will be a source of comfort to the patient. Inunctions with bland oils are indicated in itching of the skin. When the children are entirely free from fever they may remain out of bed and take ordinary food.

Complications and sequelæ are treated according to their character, without paying any regard to the eruption.

After desquamation—*i.e.*, about two weeks from the beginning of the disease—the patient may take a lukewarm bath and be allowed free communication with others without any risk.

Prophylaxis against measles and its spread is not very serviceable. Inoculation with the blood and secretions of measles offers no advantages, because it produces, not an insignificant local process, but the general disease. The majority of physicians recommend that children suffering from measles be not separated from the others, because it is generally assumed that the latter will be attacked sooner or later, and strict quarantine is rarely practicable. As the patient may infect others during the prodromal stage, when no eruption has appeared and the diagnosis cannot be made, the disease will usually develop in the other children, even if they are removed prior to the appearance of the eruption but have been present during the catarrhal symptoms.

LECTURE XII.

SCARLET FEVER—SCARLATINA.

SCARLATINA (*febris scarlatinosa, angina maligna, rosalia*) is an acute, contagious, febrile disease, characterized by an inflammatory affection of the throat and a scarlet-red eruption upon the general integument. Sennert and Döring described it at the beginning of the seventeenth century, but Sydenham (1670–1674) was the first to give an authoritative description.

As in measles, some forms run a normal (typical), others an abnormal (atypical) course. The typical diseases are divided into the periods of incubation, the prodromal, florid, and desquamative periods.

The period of *incubation* is shorter, on the average, than in measles. It usually lasts about eight days, often not more than four to five days. In exceptional cases it may be extended to three to five weeks, or it may be very short, hardly twenty-four hours. In ascertaining its onset we are restricted to the demonstration of an opportunity for infection from another case, because experimental inoculations have rarely been performed, on account of the possible dangers of the resulting disease, and, when performed, have been in part unsuccessful, in part have shown very different periods of incubation. During the period of incubation, as a rule, there is no disturbance of health. Occasionally, for two to three days prior to the prodromal stage, there is slight fever, with malaise and a feeling of dulness in the head.

The *prodromal* stage begins with sudden high fever. The temperature reaches 40° C. or more, the pulse is 140 to 160 in a minute. At the same time we notice angina, redness and swelling of the tonsils and soft palate, which exhibit dark-red points, and extending with less severity to the pharynx, hard palate, more rarely to the nasal cavity, larynx, trachea, and conjunctiva. The tongue is thickly coated. Difficulty in swallowing, malaise, and the symptoms of fever—viz , thirst, nausea, headache—are present in moderate degree. Sometimes the concomitant symptoms are very severe. Vomiting, drowsiness, convulsions (in children), headache, delirium are observed not infrequently, and although they indicate a more

intense affection of the nerve centres, they are not decisive with regard to the further course of the disease.

This stage lasts twelve to twenty-four hours, sometimes two to three days, and is distinguished from the *eruptive* stage merely by the appearance of the eruption. The fever and attendant symptoms continue unchanged or even increase in severity.

The eruption appears first upon the neck and clavicular region in the form of closely aggregated, pale-red or deep-red dots as large as the point of a needle. Seen from a distance they unite into a diffuse and uniform redness. At this time the color is not scarlet, rather a bright red. But the finely punctate appearance and its boundary at the edge of the jaw permit a probable diagnosis of scarlatina. The face, although reddened by fever and somewhat turgescent, is not the site of the eruption. In exceptional cases patches appear upon the forehead and temples, but the vicinity of the mouth always remains pale (Thomas).

In the *florid* stage the eruption spreads rapidly over the back and chest, the upper and lower limbs, with especially distinct development upon the palms of the hands and soles of the feet. It is composed of the characteristic fine dots and has a pronounced scarlet color. Upon the limbs the eruption is usually discrete, and in some places appears in patches as large as a lentil. It disappears under the pressure of the finger, and the skin is somewhat œdematous. On the second day it attains its greatest extent and severity. It continues for one to three days, sometimes five to seven days, the intensity of the color frequently changing, especially with the exacerbations and remissions of the fever.

The fever and general symptoms continue. The anginal symptoms are sometimes moderate—diffuse redness, moderate œdema of the velum and uvula. Occasionally there is intense inflammation and a gray or diphtheritic coating upon the tonsils. In children this occasions great discomfort and may become directly dangerous to life. The buccal mucous membrane is diffusely reddened; the tongue, after exfoliation of the gray coating, has a dark flesh-colored and villous appearance (cat's tongue); the cervical glands are often distinctly swollen; the skin is dry, hot, burning; the urine is scanty and often contains albumin and renal epithelium (Eisenschitz).

After the eruption has remained at its acme for from one to three days it begins to fade from the neck downward, with coincident diminution of the febrile and other symptoms. The angina alone continues unchanged, or even grows more severe. Within four to eight days the eruption has faded, leaving a yellowish-brown pigmentation; the skin has a normal temperature and perspires; the angina and other symptoms are subsiding; the restlessness and sleeplessness have disappeared, and appetite returns.

The *desquamative* stage now begins, and is shown by detachment of the epidermis corresponding to the site of the eruption. It is removed in larger or smaller lamellæ (membranous desquamation), especially on the fingers, where it may come off like the fingers of a glove (desquamatio siliquosa), sometimes followed by the nails. In parts which perspire more freely the skin comes off in scales (furfuraceous desquamation). The desquamation is complete in two weeks and the epidermis uniformly regenerated. In a few cases alopecia or grayness of the hair has been observed. The entire process, from the prodromal stage to the termination of desquamation, lasts from two to three or five weeks. This clinical history obtains in mild epidemics or in sporadic cases in otherwise healthy individuals. But scarlatina exhibits, much more often than other acute exanthemata, very considerable variations which entail an anomalous symptomatology and course.

The period of *incubation* may be unusually short, four to five days; more rarely it may be abnormally long and last several weeks. The latter condition is observed particularly in rachitic or otherwise enfeebled children. The prodromal stage may be entirely absent or it is apyrexial. The angina is then rarely absent, but it may be overlooked on account of the absence of fever. The eruptive stage then appears to develop suddenly without prodromes. The eruption may be irregular, appearing first on the trunk, over the joints and those parts which are kept warm or are subject to pressure; or it is very scanty and, according to some observations, unilateral; or it may appear suddenly over the entire body. In paralytic limbs it either remains absent or, on the other hand, it is more strongly developed and lasts a longer time.

In some cases the eruption lasts barely a few hours, so that it is often entirely overlooked. Then the tonsillitis and those circumstances which point toward infection, possibly the sequelæ (desquamation, dropsy), alone reveal the character of the disease. An allied condition is *scarlatina sine exanthemate*, with complete absence of eruption and desquamation, while the angina and all the other symptoms of scarlatina may be present and may even be attended with fatal results.

The eruption may also continue for an unusually long period (one to two weeks or even longer). This is usually associated with frequent fading and recrudescence of the eruption, with the production of patches which do not disappear under the pressure of the finger, and even of petechial patches. A return of the eruption after complete or nearly complete desquamation has also been reported. In such cases, however, we probably have to deal with erythema and not with a true scarlatina eruption.

The desquamation may also be unusually delayed or protracted,

or it may be very intense—*i.e.*, in the shape of extensive and thick callosities.

There are numerous variations in the morphological characteristics of the eruption. In *scarlatina lævigata* the patches are more prominent and shining ; in *scarlatina papulosa* and *miliaris* there is a distinct formation of nodules and vesicles upon the diffusely reddened skin ; in *scarlatina variegata* large patches start from the individual red dots, and by their darker color contrast distinctly with the paler color of the general eruption. In *scarlatina hæmorrhagica s. septica*, hæmorrhages—at first punctate, later growing to the size of a dollar or the palm of the hand—appear upon the scarlatinous parts of the skin, in children chiefly on the trunk, in adults upon the neck and over the joints ; this is associated with scorbutic manifestations in the buccal mucous membrane.

With regard to the coincidence of scarlatina with other acute exanthemata, measles and variola, there is no doubt that the virus of these different diseases may be received into the body at the same time and may develop their specific effects. This is manifested, however, by the appearance of one eruption upon the skin when the other is already diminishing. But cases in which one eruption—for example, scarlatina—is interrupted by an outbreak of measles, and, after the fading of the latter, again returns, are probably susceptible of another interpretation.

Erythema, urticaria, a few vesicles, pustules or petechial patches, occur occasionally in scarlatina, but have no further significance than that of local increase of vascular distention, exudation, and permeability of the vessel walls. Chronic eruptions, such as scabies, eczema, psoriasis, subside during the acme of scarlatina, but return with the advance of convalescence.

The series of further anomalies which scarlatina may exhibit is as large as that of the different local and general symptoms. There is hardly a single one which may not develop into the most prominent feature of the entire symptomatology, so that the clinical history may be materially changed. Such abnormal symptoms appear as complications if they pass entirely beyond the bounds of the ordinary clinical history, or as sequelæ when they continue beyond the ordinary period of duration or begin at that time.

The most frequent complication is an abnormal severity of the angina (*angina scarlatinosa maligna*). Even during the prodromal stage and at the beginning of the eruption there is parenchymatous inflammation of the tonsils, the mucous membrane of the palate and pharynx, and of the submucous connective tissue. The condition is accompanied by marked interference with deglutition, patency of the mouth, abundant flow of saliva, high fever, swelling of the face, restlessness, delirium. The enormously enlarged tonsils are applied

to one another and may cause suffocation. The formation of an abscess in one or more parts of the tonsil is a relatively favorable termination. A more dangerous outcome is suppuration of the submucous connective tissue. Retropharyngeal abscesses may prove fatal directly or as the result of burrowing of the pus. Fatal results are rapidly produced when the inflammatory infarct of the tonsils and the palatal mucous membrane leads to gangrene, shown by the well-known gangrenous odor of the breath. The gangrene, starting from the tonsils, extends very rapidly over the palate, the mucous membrane of the mouth and pharynx. Death takes place after coma, convulsions, ichorous discharge from the nose and mouth, acceleration and feebleness of the pulse. The eruption continues during the high fever which accompanies the gangrene, and only pales when the pulse becomes weaker.

In other cases the angina is aggravated by diphtheria of the pharynx (*angina diphtheritica*). The dirty-yellow, fibrinous deposits may extend over the pharyngeal mucous membrane and through the posterior nares to the Schneiderian membrane. Exfoliation of the membranous deposits often takes place. Greater danger arises when the larynx is involved in the diphtheritic process (*laryngitis crouposa*). This terminates fatally by suffocation, complicating pneumonia, gangrene. and decomposition of the blood, or, even after detachment of the membrane, by sudden paralysis of the nerves and vessels.

All these affections are accompanied by increased swelling or inflammation of the salivary glands and the glands of the lower jaw. This sometimes terminates in extensive inflammation of the surrounding connective tissue. The region of the cheeks, jaw, and neck is occupied by a firm swelling, which presses against the larynx and makes the opening of the mouth impossible. Abscesses constantly form in a number of places ; the local and general symptoms may subside and recovery occur. In other cases rapidly spreading gangrene develops and terminates fatally by exhaustion, high fever, or erosion of the large vessels of the neck and hæmorrhage. Again, an indolent swelling may persist for months despite an otherwise favorable course.

Affections of the intestinal tract, shown in mild cases by moderate diarrhœa, may assume a dangerous character from intensification into croupous enteritis. This is attended by profuse bloody mucous discharges, rapid exhaustion, lowering of the temperature, and death.

Catarrhal and suppurative otitis media, perforation of the drum membrane with secondary ankylosis of the ossicles, inflammation and caries of the mastoid process with resulting phlebitis and meningitis, which run a chronic course or terminate rapidly in death, not

infrequently complicate scarlatina even in its early stages ; they also occur as sequelæ.

Any angina scarlatina parenchymatosa, gangrænosa, or diphtheritica may be the cause of all previously mentioned complications, and also of numerous others, especially hæmorrhages, embolism, pyæmia, and metastatic inflammations in all tissues and organs.

Even though life has not been destroyed by any localized process (including angina and eruption), the blood-poisoning produced by the scarlatina contagion may cause death by giving rise to general decomposition of the blood. This condition is manifested in the symptom-complex known as typhoid scarlatina, *scarlatina septica* or *hæmorrhagica*. With regard to the intensity of its symptoms it is divided into two forms (Mayr, Hebra).

In the first form great muscular weakness and mental confusion are manifested at the outbreak of the fever. In the eruptive stage there is an increase of the symptoms of cerebral involvement—viz., frequent vomiting, delirium, mental hebetude, coma, convulsions, dilatation of the pupils. Bronchial and tracheal râles soon develop, the tongue is red and dry, the abdomen tympanitic, the spleen moderately enlarged, the urine scanty. The pulse, which is very rapid, becomes weaker, the temperature falls, and death follows in the course of twelve hours to five days. In the rapidly fatal cases the eruption is absent ; otherwise it is irregular or appears suddenly in large, very deeply colored patches which soon become livid or hæmorrhagic.

In the second form of typhoid scarlatina the cerebral symptoms are more moderate (according to Löschner) and the disease as a whole runs a slower course. The eruption develops regularly, although often associated with petechiæ or miliary vesicles. It is not until a later period that the symptoms grow more severe. Albuminuria, tympanites, and diarrhœa set in, then coma, feeble pulse, and diminution of temperature, followed by death.

The notable post-mortem findings in such cases are : gray granulations on the meninges (more frequent after a slow course of the disease); congestion of the brain, lungs, and intestines ; swelling of Peyer's patches, the solitary glands of the intestines, and the mesenteric glands; finally an accumulation of serum in the serous cavities.

Renal disease is probably the most frequent complication of scarlatina. In the majority of cases, even in those which run a normal course, the urine contains albumin and renal epithelium, and sometimes evidences of fatty degeneration (Eisenschitz, Steiner, etc.). Catarrhal and parenchymatous nephritis develops, however, chiefly during desquamation and at a later period, thus constituting a sequel. Even the most favorable case of scarlatina may finally terminate fatally as the result of such a complication.

Pneumonia, desquamative and croupous (the latter as a result of the extension of laryngeal croup), inflammation of the serous and synovial membranes (pleurisy, pericarditis, peritonitis, joint affections) are among the rarer complications. Keratomalacia may occur as a sequel of septic scarlatina and gangrene. Less importance attaches to epistaxis, aphthæ of the mouth, suppuration of individual glands of the lower jaw and neck, when they constitute an isolated complication of normal scarlatina.

The *sequelæ* of scarlatina are those affections which have begun during the disease, but continue independently after the period of desquamation, or begin after the latter, although they are due to the changes in the organism peculiar to scarlatina.

The first category includes chronic ozæna, either in the shape of simple chronic nasal catarrh, or more intense inflammation which may lead to ulceration, purulent discharge, caries and necrosis of the turbinated bones and septum, erysipelas, and even gangrene; otitis with its possible results, such as destruction of important structures, permanent impairment of hearing or deafness, or even fatal caries; enlargement of the tonsils; chronic intestinal catarrh and secondary impairment of nutrition; metastatic affections of the joints and serous membranes; inflammation and suppuration of the lymphatic glands, skin, and subcutaneous cellular tissue in various parts of the body. The parotid and submaxillary glands are especially apt to enlarge slowly and remain for months, or even for a year or two, as indolent infiltrations of the periglandular connective tissue. All these affections may lead to a chronic disturbance of general nutrition, especially to scrofula, or even to an acute process with a fatal termination.

The most frequent and dreaded sequel of scarlatina is the renal affection, which leads in rare cases to sudden death from uræmia after symptoms of acute Bright's disease. Or it may lead to the gradual development of dropsy in different parts of the body (sometimes confined chiefly to the lower limbs), general anasarca, and ascites. The majority of cases with late dropsy recover; others lead to serious complications or may terminate fatally as the result of acute hydrocephalus, hydrothorax, hydropericardium, or œdema of the glottis.

In accordance with the general plan of these lectures I have merely given the chief factors among the complications and sequelæ of scarlatina, but even these considerations will serve to convince you of the varying and uncertain character, and at the same time of the danger, of the scarlatinous process.

The *prognosis* of scarlatina must always be regarded as doubtful. I am acquainted with no disease which is more insidious, and we may be surprised by the most dangerous symptoms in every case and at any time. A case which begins normally and is attended with

moderate and typical symptoms may suddenly terminate fatally from uræmia, cerebral paralysis, or one of the previously mentioned complications. Even after it has run a favorable course it may be protracted by sequelæ and metastases, or it may subsequently become dangerous, perhaps proving fatal. Under all circumstances, therefore, the physician must remain sceptical, must regard even the slightest complication as not devoid of importance, and must not declare the patient free from danger until all the symptoms—even those which are protracted beyond desquamation—have entirely disappeared, the urine is entirely free from albumin, and the functions of the organism have become entirely normal.

Special attention should be paid to the course of the fever and the eruption. Very high fever is always a serious indication, and still more its complication with notable cerebral excitement or depression. A distinct eruption, which develops at the right time, with moderate fever and catarrhal angina, furnishes the most favorable symptomatology. Parenchymatous angina, inflammation of the submucous connective tissue of the pharynx and of the neck, are very dangerous; diphtheria, croup, and gangrene almost always terminate fatally. The sudden disappearance of the eruption indicates severe disease of internal organs—*e.g.*, the lungs or brain. Delayed appearance of the eruption, with constant high fever and brain symptoms, is also a bad sign. Scarlatina with a mixed eruption is often followed by obstinate bronchitis and pneumonia. Scarlatina miliaris, characterized by the development of vesicles upon the already existing eruption, indicates pyæmic blood-poisoning. A few petechiæ or epistaxis are not ominous if the other symptoms are moderate; but high fever, delirium, stupor, and gangrene are indications of general dissolution of the blood.

Careful attention must be paid to the urine from the beginning, and the presence of albumin determined by daily examinations. Albuminuria is not a dangerous symptom *per se* and is hardly ever absent. But its presence must make us alive to the danger of uræmia and its possible termination, or of subsequent dropsy and protracted convalescence. This, as well as every other complication, must be judged according to its pathological significance and its possible results, then utilized in making a prognosis. The general character of the epidemic also furnishes some data in regard to prognosis. According to the statements of some experienced pædiatrists no epidemic is benign, although some are characterized by the unusually large number and danger of the complications and sequelæ. Toward the end of an epidemic the cases are generally milder. During the prevalence of croup, typhoid fever, dysentery, and cholera sporadic cases of scarlatina are also more dangerous.

Finally, the individual conditions as regards age, the general constitution, and other complicating diseases must also be considered.

As a general thing young people are in greater danger than older ones, although the latter may also die very rapidly. Poorly nourished persons, children who have suffered from frequent sore throat, women in the puerperal state, and convalescents from variola or typhoid fever are put in great jeopardy by scarlatina. I will conclude these hints with the admonition that although the physician should not keep the family in constant alarm concerning every case of scarlatina, still he should not be assured of recovery until the last complication and sequel has entirely disappeared.

Concerning the anatomical changes of the eruption nothing can be said that is not evident from its clinical appearance: in the normal forms, hyperæmia and moderate exudation; in nodules and vesicles, correspondingly greater exudation and cell proliferation within the papillæ and rete; in petechiæ, free extravasation of blood into the papillæ and cutis. In the cadaver the hyperæmia disappears. The other anatomo-pathological changes in the skin and various tissues and organs correspond to the affections which appear during the clinical course of the disease.

The *etiology* of scarlatina has not been more definitely settled than that of the other acute exanthemata. There is no doubt that the disease is due to a specific contagium. The opportunity for infection everywhere and in the majority of cases of scarlatinal disease proves that the disease develops only from contagion. This must also be assumed in the sporadic cases, whose source of infection cannot always be demonstrated.

Infection of healthy individuals by inoculation with the blood, scales, and secretions of scarlatina patients has succeeded in a few cases, but has often been followed by a severe general disease, so that such experiments have been abandoned. Others have failed to produce infection. Miquel alone states that he produced merely local inflammation and protection against subsequent infection. Numerous bacteria have been found (Cose and Feltz, etc.) in the blood of rabbits who rapidly succumbed after injection of the blood of scarlatina patients. It is very doubtful whether they are related to the virus of scarlatina.

The scarlatina virus is volatile and fills the respiratory sphere of the patient. It is also contained in the blood, probably also in the desquamative and excretory products of the patient, and adheres to objects which may carry it to remote regions. Infection may result from inhalation of the air impregnated by the contagium, direct contact with the patient and his secretions, and contact with other persons and objects to whom the virus adheres. The most remarkable statements have been made concerning the prolonged vitality of the virus, its resistance to changes of locality, to extreme heat and cold. The disease has been transported many miles through the

agency of healthy individuals or of objects—clothing, even letters—which have been brought from scarlatina patients or their vicinity. The disease has attacked individuals who have moved into a room which had been occupied months before by a scarlatina patient and had then been thoroughly cleaned and disinfected.

The virus seems to be exhaled by the patient in the eruptive but not in the prodromal period. The infective power of scarlatina lasts longer than that of measles, and may even continue for some time after the cessation of desquamation, if sequelæ, such as dropsy, are left over.

The predisposition to the scarlatina virus is less marked than to that of measles. For this reason, as a rule, only a few members of a family are attacked, rarely all the children. The predisposition appears to be greatest between the ages of two and seven years, but, with the exception of old age, no period of life is exempt. Whether the affection can be acquired *in utero* and appear in the new-born has not yet been settled. A single attack seems to afford life-long protection against another infection—at least cases of a second infection are extremely rare and open to doubt.

Scarlatina occurs more frequently in a sporadic form and is always present in large cities. Some maintain that diphtheria and scarlatina exhibit mutual relations with each other. The closer propinquity of individual cases and the consequent increased opportunity for further infection give rise, in large cities, to epidemics of scarlatina every three or four years. These never attain the proportions of an epidemic of measles. Variable development, protracted disappearance, and greater persistence at its height are characteristics of scarlatina epidemics. They may be mild or characterized by special complications and malignancy. The disease is sometimes carried to secluded localities by patients, convalescents, or objects which contain the virus. In such places it may remain sporadic or develop into an epidemic.

The mortality varies from five to twenty-five per cent, according to the character of the prevailing epidemic.

The *diagnosis* of typical scarlatina is assured by the characteristic eruption, its punctate redness and demarcation from the face, the early angina with the punctate redness of the palate, the fever, and the desquamation following the paling of the eruption. In abortive cases, or in those which prove fatal before the outbreak of the eruption, the diagnosis will be aided by the fact that an epidemic is prevailing, by the demonstration of an opportunity for infection, and by the presence of albumin in the urine. Other obscure forms, such as scarlatina without eruption, are recognized by the angina and the prevalence of an epidemic ; scarlatina with a fleeting eruption is diagnosed by the distinct desquamation or by certain sequelæ, such as mumps or dropsy.

Morbilli is differentiated from scarlatina by the macular character of the eruption, its presence in the face, and the catarrhal symptoms; erythema by the absence of angina, the variability in the form of the eruption, and the mild degree or absence of fever. Quinine eruptions, which are often very similar to scarlatina, may be differentiated in the same way. Purpura variolosa must also be distinguished from scarlatina.

In the puerperal state a disease known as *scarlatina puerperalis* (Helm, 1837) sometimes occurs and as a rule proves fatal. This should not be confounded with scarlatina in puerperal women, which merely signifies scarlatina during childbed. The former disease is an erythema, confined chiefly to the abdomen, occasionally localized in other parts of the body, and sometimes general. It is characterized by diffuse, light-red to dark-scarlet redness, heat of the skin, especially of the abdomen, which is often covered with numerous miliary vesicles. Tenderness of the uterus, scanty and foul-smelling lochia, a typhoid condition with high fever and dry tongue, are usually present. The erythema is the expression of a pyæmic process starting from metrophlebitis, and readily distinguished from scarlatina by the above-mentioned signs. It generally terminates in death. The vesicles are often found in the cadaver. Their site may be marked by punctate exfoliation.

In the *treatment* of scarlatina we possess no remedy which will destroy the activity of the virus emanating from a patient or already contained in the organism. Those who consider the nature of scarlatina, as well as of the majority of infectious diseases, mycotic, may believe in their ability to antagonize the virus within the body by the internal administration of salicylate or borate of soda, carbolic acid, chlorinated water, etc. It is a fact, however, that the outbreak of scarlatina, after the reception of the virus, has never been prevented by these remedies or by the mineral acids which were formerly employed.

Hence the first indication in treatment is subjective and objective prophylaxis. By the former term I mean the protection of a non-infected individual by prompt and thorough isolation from the source of disease. By objective prophylaxis is meant the attainment of the greatest possible innocuousness of the focus of infection by its isolation and by disinfection of the rooms and objects with which the patient has come in contact.

In accordance with modern experience the disease itself can only be treated symptomatically. In normal cases the purely expectant plan is the best. The patient is placed in a large, well-aired room at a temperature of 14° to 15° R., and kept in bed under light covers; he may receive cooling drinks, meat broths, milk, stewed fruit; in annoying angina pieces of ice or ice-cream are grateful, and a gar-

gle may also be used. Washing the body and changing the underclothing and bed linen have only good effects.

The patient should not be permitted to leave the bed until the pulse has been normal for several days and the skin is soft and perspiring. After the cessation of desquamation a lukewarm bath is taken every second or third day. The convalescent should not go out of doors until the end of the fourth or fifth week, when desquamation is entirely completed and albuminuria is no longer present.

In all cases of high fever and alarming cerebral symptoms I favor hydrotherapeutic treatment. Whether this should be adopted in the form of lukewarm or cool half-baths, washings or packs, etc.. must depend upon the individual case and the judgment of the physician.

I can merely mention by name some of the remedies and methods indicated in the vast number of complications, such as stimulants in cerebral pressure and collapse ; tonics and opiates in diarrhœa ; quinine, antipyrin, antifebrin, digitalis, etc., in high fever and acceleration of the pulse ; ononis, acetate of potash, iodide of potassium, diuretin, etc., in scanty diuresis, etc. All these affections are treated according to the well-known principles of medical and surgical pathology, irrespective of their occurrence in the clinical history of scarlatina. I would call special attention, however, to the swelling of the parotid and submaxillary glands, which may last from a few months to a year or two, and comes under dermatological treatment. I have often obtained surprisingly rapid results from the application of emplastrum hydrargyri puri, or this mixed with equal parts of emplastrum cicutæ. The application of iodoform 1 to collod. elasticum 15 may also be tried.

LECTURE XIII.

SMALL-POX, VARIOLA.

HISTORY—VARIOLATION AND VACCINATION—VARIOLOSIS—VARICELLA—TYPICAL VARIOLA—VARIOLA VERA—ATYPICAL VARIOLA WITH FAVORABLE COURSE.

SMALL-POX (*variola, petite vérole, blattern, vajuolo*) is the term applied to an acute contagious disease of a typical course, with an eruption of nodules, vesicles, and pustules on the skin, and accompanied by fever and an implication of the general organism.

Among the acute exanthemata variola is the most important from a pathological and epidemiological point of view. It is of special interest to dermatologists on account of the pronounced changes in the integument. The eruption constitutes the main feature of the disease and is decisive in regard to diagnosis and prognosis.

The history of small-pox shows that laymen and physicians, and indeed all who are concerned with the sanitary welfare of mankind, have always recognized the great importance of this disease. It is very probable (Moore), although not proven, that small-pox is an ancient disease and that it travelled from Eastern Asia, from China and Hindostan, to the Mediterranean coasts and then to Europe. Gregory states that Procopius (544 A.D.) gives the first accurate historical accounts of epidemics in Arabia, Asia Minor, and Egypt. At all events the disease must then have spread very rapidly over the Mediterranean coasts. According to Hecker, Gregory of Tours (581 A.D.) described an epidemic, which may be regarded as variola, which extended over the entire south of Europe. More distinct statements are made by Rhazes (900 A.D.), who also reproduces remarks on the disease by the Egyptian physician Ahron in the sixth century. It was clearly described by the Arabians, and, to judge from some of the manuscripts in the British Museum, it was known as *variola* (diminutive of *varus*, nodule), although this term is generally ascribed to Constantinus Africanus (1087).

During the Crusades the active communication between the different nations aided in the spread of the disease and also in the general notion of its infectiousness and dangerous character. Syphilis, which

appeared epidemically toward the end of the fifteenth century, was called big-pox, or "grande vérole," to distinguish it from small-pox, or "petite vérole." At the end of the fifteenth century small-pox and the dreaded "black death" entered the newly discovered transatlantic countries. During several epidemics millions of people fell victims on both sides of the Atlantic. Numerous hospitals were erected for sheltering and isolating those attacked by the disease. The dread of infection gave rise to numerous precautions which would now be regarded as inhuman—for example, the complete isolation of every house containing a small-pox patient, by nailing a notice on the door.

In the latter part of the seventeenth century Sydenham published valuable papers on the pathology of variola, and during the eighteenth century light was thrown on its clinical aspects by Boerhave, Van Swieten, Cotugno, De Haën, Hoffmann, Sauvages, and others.

An important peculiarity noticed by all observers was the fact that in certain epidemics the disease was usually mild with slight affection of the skin, while in others there was marked lesion of the skin and other organs and the cases often ended in death. Sydenham had applied the term *variolæ regulares* to the epidemics of 1667–1672, and *variolæ anomales* to those of 1674–1675. As it was known that one attack protected against a subsequent one, the notion arose of directly infecting individuals at a time when the mild forms of the disease prevailed. In this way developed the practice of inoculating human small-pox—*i.e.*, variolation or inoculation.

The genesis of this method is unknown. According to Eimer it was practised in the Orient as early as the eleventh century and was undoubtedly transplanted thence. It is known that Lady Montague, the wife of the English ambassador in Constantinople, inoculated her son there in 1717, and in 1721 inoculated her daughter in England with the contents of variola vesicles. The method then extended to the Continent of Europe, stimulated by the example of the ruling families.

When inoculation was performed, papules developed, on the third to fourth day, upon the site of inoculation, and in its vicinity on the following days. These developed into vesicles and pustules, and on the tenth to eleventh day a general small pox eruption, which usually ran a mild course. Although such individuals were generally protected against another attack, variolation could not be maintained permanently. It was evident that inoculated individuals led to the spread of variola contagion and to the production of epidemics in the same way as those who were attacked accidentally. Hence inoculations were strictly prohibited toward the end of the last century.

In the meantime Jenner, in 1798, had brought into prominence inoculation with cow-pox (vaccination). This produced only a few local pustules and no general disease; further, it gave rise to no

infection, at a distance, of other individuals, and nevertheless protected against variola. This brilliant result secured for vaccination the esteem of all intelligent and impartial physicians and laymen. I shall not enter here in detail into the question of vaccination, because I shall again consider it in the chapter on prophylaxis.

Jenner made no such claim himself, but many of his pupils entertained the hope that the possibility of inoculation with variola is absolutely *nil* after vaccination. It soon appeared, however, that some vaccinated individuals were attacked by small-pox, although usually in a milder form, after the lapse of a few years. In order to rescue the theory such attacks were called *varioloid* or *varicella*. In time, however, the belief gained ground that vaccinated individuals may also suffer from small-pox. But as such cases were usually mild and differed in some respects from the variola vera of non-vaccinated individuals, they were regarded as mitigated or modified (variola modificata) by vaccination. In fact, however, all these cases are genetically and pathologically the same; vaccinated as well as non-vaccinated may be attacked by variola vera, although much more rarely; mild varioloid and varicella in the vaccinated may give rise to severe small-pox in the non-vaccinated; and, finally, non-vaccinated individuals may suffer as mildly as the vaccinated.

With regard to *varioloid* all opinions agree. This term is applied to mild variola, whether it occurs in the vaccinated or the non-vaccinated.

With regard to *varicella*, however, opinions differ greatly. Before the introduction of vaccination it was known as varicella, variolæ spuriæ, water-pox, chicken-pox, etc. Since the last century it has been regarded both as identical with variola (Thomson), as a form of variola modified by vaccination, and as an entirely distinct disease. It is impossible to form a decisive opinion from the conflicting statements in the older literature. Eisenschitz, who has collated these data, has arrived at conclusions with which I, as well as others (Kassowitz), are unable to agree. In addition to Hesse, Trousseau, and Vetter, many recent pædiatrists (Thomas, Steiner, Lothar Meyer, Gerhardt, Monti, Fleischmann, Henoch, etc.) maintain that varicella occurs only in children and is entirely distinct from variola. The arguments advanced by these writers have been opposed by Hebra, Kassowitz, and myself with arguments which appear to me sufficiently strong to deny the existence of a varicella distinct from variola. I cannot enter into details, but I recognize only a single form of variola, derived from a single virus, which may occur with more or less severe, even fatal symptoms, and at other times runs its course as an insignificant disease. With Hebra I regard it as practicable to recognize three classes of variola, according to their degree of severity—viz., variola vera, varioloid, and varicella. It must

always be remembered, however, that these forms are identical and that one form may give rise to another in another individual.

Varicella runs its course in fourteen days or less, varioloid in three to four weeks, and variola vera in four weeks or more.

We will now pass to the symptomatology of small pox.

Although you may read numerous valuable treatises on small-pox, it will be difficult to form in your own minds an harmonious picture of the affection, due to the often considerable variations in the symptomatology. This is due rather to the disease itself than to the writers on the subject. While assistant in the clinic I had the opportunity of observing five thousand cases of small-pox, and I thoroughly understand that such differences of opinion are unavoidable. Not alone does each epidemic present a great variety of cases, but individual symptoms are especially prominent in this or that epidemic or district. One whose observation is restricted is therefore apt to regard accidental symptoms as characteristic.

I make these remarks in order to show that a description of small-pox in sharply defined groups is apt to exhibit a schematic appearance. In Nature there are numerous transitions between the mildest and most dangerous forms.

But if we strike an average among thousands of cases we may divide them into two forms—viz., the typical or normal and the atypical. Both forms include mild and severe or fatal cases.

In small-pox with a normal course the stages found in other acute exanthemata are distinctly marked by corresponding symptoms, and this is particularly true of the eruption.

VARIOLA VERA.

The stage of *incubation* lasts two weeks, as a rule, occasionally longer (three weeks), more often only ten to twelve days. During the early part of this period the patient feels well, but toward its close malaise, anorexia, and restless sleep are sometimes noticeable.

The *prodromal* stage begins suddenly with a chill, generally in the evening. As a rule this is attended by high fever (40° to 41° C.), and followed by vomiting, headache, great restlessness, delirium, sometimes violent convulsions (in children), and severe pains in the small of the back. The latter are so violent that the patients complain immediately, imagining that they are suffering from an injury or inflammation. This symptom is very characteristic, although not entirely pathognomonic. The fever continues on the second or third day, a feeling of heat alternating with chills; otherwise it increases somewhat as do the other symptoms, particularly the pains.

The mucous membrane of the palate and pharynx is often reddened diffusely or in patches, the tonsils are swollen, and there is difficulty in swallowing. On the third day red, elevated papules

are visible here and there on the buccal mucous membrane, in places where the eruption will appear later.

In some patients an eruption (roseola variolosa, erythema variolosum, or prodromal eruption) appears on the second or third day upon the general integument This was first described accurately by Hebra and shown to be a prodrome of variola. I have observed it in all forms of the disease, but it is especially frequent in certain epidemics.

It appears in the shape of bright-red to dark-red, irregular, sometimes sharply defined dots, patches, and streaks, which are flat or sometimes slightly prominent. They grow pale under pressure and rarely itch or burn. They are found particularly in the groin and adjacent inner surface of the thighs (femoral triangle of Simon), the pubic and hypogastric regions, the extensor aspects of the knees and elbows, the phalanges, dorsum of the foot (along the extensor of the great toe, Simon), the axillary folds and clavicular region, the lateral aspect of the loins. They may also appear occasionally in any other part of the trunk or limbs.

In the region of the groin, pubes, and axillæ the erythematous region often contains dark-red patches, from the size of a dot to that of a lentil, which do not disappear on pressure (hæmorrhages, petechiæ). During the next few days these pass through the well-known stages of green, yellow, and brown pigmentation.

The erythema is changeable in many places; in others it is more permanent, and, while gradually paling without desquamation, lasts until the first day of the eruptive stage, rarely longer. In such cases miliary vesicles, or even large vesicles and wheals, develop upon the erythematous patches. As a general thing this erythema is more frequent in females and in the young and vigorous. In the former it is especially apt to be petechial and associated with profuse menstruation, whose appearance has been precipitated.

Neither the intensity of the febrile symptoms nor the extent of the prodromal eruption furnishes any data in regard to the severity of the beginning variola. It is true that those parts which were the site of erythema, particularly the femoral triangle, usually remain free from the variola eruption. A very dark redness of the entire abdomen, if it continues undiminished into the eruptive period and contains recurring hæmorrhages, is an ominous symptom, as Hebra has shown. Apart from this condition I have generally regarded erythema variolosum as indicative of an attack of moderate severity.

After the prodromal period has lasted three, rarely four, days, the *eruptive* period begins: bright red, conical, firm papules, as large as a pin's head or larger, appear first upon the face and scalp, more slowly upon the trunk and limbs, the palms of the hands and soles of the feet. They are attended by a feeling of pricking, pressure, and pain. On the trunk some are surrounded by a rosy-red

zone as large as a finger nail (hyperæmic halo), and this is a characteristic sign of variola. The papules usually form around the openings of the cutaneous follicles. If the prodromal eruption is still present there is a striking resemblance to morbilli papulosi, and on this day it is hardly possible to make the diagnosis.

With the appearance of the papules the febrile symptoms, as a rule, suddenly subside. But they continue to a marked degree if the eruption has developed to a notable extent upon the mucous membrane of the pharynx and larynx, or if they are deep, numerous, and closely aggregated (precursor of variola confluens).

The general severity of the disease is decided by the appearance of the papules in normal variola. If only a few have appeared by the fourth or fifth day, and the fever has disappeared almost entirely, the entire disease will run its course in twelve to fourteen days (varicella). If the papules are more numerous but are disseminated, and there are large intervening patches of free skin, the eruption will disappear completely within three weeks (varioloid).

In the cases of typical variola vera the papules continue to multiply on the first and second days of the eruptive stage, and the intact islets of integument grow smaller and smaller. Those which appeared first (upon the face) have meanwhile enlarged and become converted into transparent vesicles by the accumulation of clear serous fluid. Many exhibit in the centre a shallow depression (umbilication).

The first appearance of vesiculation begins the *florid* stage, which is reckoned from the sixth day of the disease. During this time the fever is very moderate, the pulse 96–100, and the majority of papules are converted into vesicles.

Beginning with the eighth and ninth days the contents of the vesicles grow cloudy in the order of age—*i.e.*, first upon the face—and with the tenth and eleventh days, the acme of the process, the stage of *suppuration* begins. The vesicles are converted into pustules, they increase to the size of a pea, the umbilication is effaced, and they are full and tense. The base is surrounded by a red zone, often by a large inflammatory halo.

With the beginning suppuration the fever again increases (suppurative fever), together with the subjective annoyances due to the changes in the buccal and pharyngeal mucous membrane, to the number of pus foci in the skin, and to the accompanying inflammatory symptoms (a feeling of pain and tension in the skin, difficulty in swallowing, thirst, insomnia, dull feeling in the head, etc.). In this stage febrile delirium not infrequently drives the patients to attempts at suicide, so that they must be constantly watched.

The face of the patient, when the pustules are abundant and uniform, is swollen, the eyelids œdematous and closed, the nose and lips

thickened, the lower lip pendulous from the weight of the pustules, the mouth open and drooling, the nostrils closed by pustules and crusts, the ears thick, so that the features become unrecognizable. The arms and hands are thickened and flaccid ; the fingers are kept semi-flexed. The feeling of pain and tension is most annoying upon the palms of the hands and soles of the feet, where the pustules are flattened, not prominent, on account of the thickness of the layer of epidermis ; also upon the scalp.

As a general thing the pustules are distributed uniformly, but are most scanty upon the abdomen. In places they approach one another more closely (variola corymbesa), while they are usually absent in parts which have been the site of erythema (the femoral triangle) and in certain parts, such as the buttocks, etc., which constitute intermediate zones according to Voigt's distribution of the cutaneous nerves. Upon the trunk and shoulder region they are arranged in parallel rows which correspond on the one hand to Langer's cleavage lines, on the other hand to the course of the nerves.

Parts of the skin which had been irritated prior to the disease—for example, by sinapisms or the prolonged pressure of trusses, etc.—are covered, as a rule, by an unusually dense efflorescence of pustules, evidently because the cutaneous vessels are more predisposed to hyperæmia and stasis.

Pocks also appear upon the mucous membranes, where their development is much more rapid. Even toward the end of the prodromal stage the hard and soft palate, tongue, cheeks and lips, tonsils and pharynx often contain red papules, which are soon covered by a grayish layer due to the maceration of the epithelium by the warmth and saliva. In a few days this layer falls off and we see, in the centre of the efflorescence, a little pit with a red base, which is denuded or covered with a thin layer of epithelium. On the twelfth to fifteenth day (later in severe cases) the remains of the gray layer are exfoliated and the site of efflorescence is covered with newly formed epithelium. The number of pocks is usually proportionate to that on the general integument. The dorsum of the tongue is often densely covered with them, and these give rise to pain on swallowing, an abundant flow of saliva, and dryness of the throat. In severe cases they are also abundant upon the epiglottis and interior of the larynx, and are found, on autopsy, upon the mucous membrane of the trachea and the larger bronchi. It is only in severe cases that the latter may cause aphonia, œdema of the glottis, gangrene, and perichondritis. In children and infants variola of the buccal mucous membrane is a serious element on account of the interference with nutrition. Otherwise the efflorescences in these regions run a very rapid course and are unattended with serious local complications.

Large numbers of pocks are often found in the œsophagus.

They appear in scanty numbers and very slowly on the vulva and vagina and the lower part of the rectum.

The external auditory canal is covered as far as the bony portion. The deeper parts and the drum membrane always escape. Hearing is very little impaired during this time.

Efflorescences are found upon the integument of the eyelids and also upon the edges of the lids, corresponding to the Meibomian glands. They rarely appear on the mucous membrane of the lids, where they are soon macerated. Pocks do not form upon the conjunctiva of the ball; at the most a rapidly disappearing little pustule at the limbus, especially in children who had already suffered from conjunctivitis pustulosa (herpes corneæ, Von Stellwag). The grave affections of the eye in small-pox depend upon complications and sequelæ.

The stage of *desiccation* begins in moderate cases about the thirteenth day, in more severe cases one or two days later. In the former it runs its course in eight to ten days, in the latter in ten to fourteen days. Its onset is shown by diminution of the suppurative fever. The pulse, formerly 112 to 120, falls in one to two and a half days to 96 to 80, later may fall below the normal. The patient is able to sleep and the appetite returns. Here and there upon the face the pustules have ruptured and are covered with yellow crusts. The others, which are usually full of a thick, purulent matter, sink in at the apex (secondary umbilication) and form brown crusts, the product of the desiccation of their covering and contents. At the same time the swelling of the skin subsides and the face resumes its normal contours. The drying of the pustules proceeds rapidly within the next few days. After the sixteenth day the crust may fall off, leaving a shallow, white, glistening depression. The dark-brown, lenticular bodies which are encapsulated in the epidermis of the palms of the hands and soles of the feet, and are due to desiccation of the pustules in those localities, persist for three to four weeks. With this exception decrustation is completed during the fourth week. The patient, who was emaciated at the beginning of desiccation, now takes nourishment and increases steadily in weight.

The marks of the pocks, in part white and shining (cicatricial), in part slightly depressed, brownish or bluish-red patches, are always visible, at least for many months. The former persist during life, the latter gradually disappear.

There are numerous deviations from this type of variola. There is not a single epidemic in which all possible forms are not observed, and, fortunately, the most numerous deviations are favorable in character. I will first consider the favorable varieties.

The prodromal stage may be free from any confusing symptoms. The patient exhibits variola efflorescences and hardly remembers

that he has been ill. Some of the patients call at the dispensary on account of a supposed acne. Numbers of pustules are not present in such cases, although the face may contain not a few. Considerable suppurative fever may develop later, and even disagreeable sequelæ may be observed.

In other cases the prodromal symptoms are extremely violent, there is considerable prodromal eruption, on the fourth day the fever subsides and ten to twenty papules appear and rapidly develop into large vesicles. The majority desiccate ; only a few become pustular, without suppurative fever, and desiccate on the tenth to twelfth day. This condition constitutes *variola apyretica*, *varicella*, appearing for the most part in children, and vaccinated individuals in youth and middle age, and is probably often mistaken for acute pemphigus.

Again, the prodromal and eruptive stages run a typical course, but an enormous number of papules form and develop into vesicles with or without umbilication. On the ninth or tenth day there is moderate suppurative fever, then there suddenly occurs a general desiccation of the little pustules and cessation of decrustation on the fourteenth or fifteenth day. It is evident that this can only happen when the pustules are superficial.

LECTURE XIV.

SMALL-POX (*continued*).

UNFAVORABLE ATYPICAL FORMS—VARIOLA HÆMORRHAGICA—VARIOLA CONFLUENS—COMPLICATIONS AND SEQUELÆ—ANATOMY.

THE unfavorable atypical forms of variola are much more varied in their course. The chief importance attaches to

VARIOLA HÆMORRHAGICA,

known to the laity as black small-pox, but this term may not be applied properly to every case in which hæmorrhages appear during the course of the variola process. It is, however, impossible to draw a sharp boundary between cases of true hæmorrhagic small-pox which run an unfavorable course and those in which the hæmorrhages are merely a slight symptom. There are numerous transitions between the two forms.

The significance of hæmorrhages in variola depends upon the stage of the disease in which they occur and their localization. The number of the hæmorrhagic foci is the most important feature, then the circumstance of their development—whether at one time or in succession. The larger the number of hæmorrhages and the more continuously they develop, the greater the degree of malignancy which attaches to the disease. But the mere loss of blood *per se* is not the cause of the severity of the course, and hence is not the most important symptom of variola hæmorrhagica. In the really malignant cases the hæmorrhages are merely the result of the destructive general disease.

Variola hæmorrhagica, in this sense, may be divided into two forms:

First Form, or Purpura Variolosa.—For two or three days the patient suffers from general malaise, headache, anorexia, pains in the small of the back. On the fourth day violent fever sets in, with great restlessness and an eruption. The latter consists of a dark purple redness which is uniformly distributed over almost the entire integument—the face, neck, trunk, abdomen, and limbs—and disappears under the pressure of the finger. The skin is hot, dry, and turgescent, and resembles a severe case of scarlatina. This mistake

is prevented by the uniform staining, which looks as if ink were diffused through the skin, and by the distribution throughout the face. The other symptoms are still more significant. The fever and the frequency of the pulse are considerable; the cornea is shining, the pupils contracted; the patient throws himself restlessly about in bed; the pains in the back have increased to such an extent as to provoke outcries. Some patients also complain of severe pains in the epigastrium and of dyspnœa without any objectively demonstrable cause.

Even at this time—*i.e.*, on the first day of the eruption—consciousness is impaired. The patient still answers questions promptly, but he ignores everything else that is going on around him. He appears to be lost in himself and his sufferings.

Hæmorrhages develop in a short time—at first in the conjunctiva, where they occupy an internal or external angle in the shape of a triangular ecchymosis. Next they appear upon the general integument, especially upon the trunk and abdomen. Here they appear as blackish-blue patches, from the size of a pin's head to that of a lentil, which do not disappear under the pressure of the finger. The integument over them is smooth and elastic. At first they develop singly and here and there, without any definite arrangement, upon the trunk, the face, and the limbs. The individual patches spread very rapidly (within a few hours) at the periphery, as if they became diffused like drops of fat on filtering paper. Thus a patch as large as a lentil may within a few hours grow to the size of the palm of the hand. In this way, and also by the coalescence of adjacent patches, extensive blackish-blue discolorations develop resembling *livor mortis*. The number of new hæmorrhages also increases rapidly.

The other tissues also become the site of extravasations in the course of a few hours. The bulbar conjunctiva, which is ecchymotic at the canthi at the onset of the scarlet redness, becomes infiltrated within a few hours into a dark bluish-red ridge which projects like a wall above the shining cornea. The epithelium of the mucous membrane of the lips and tongue dries into a dirty brownish-red crust which interferes with their mobility. Fissures are produced, with free extravasations of blood, hæmorrhagic suffusions beneath the crust, and also hæmorrhages into the mucous membrane itself. The mouth emits a fœtid smell; the mucous membrane of the palate and pharynx is brownish red, dry, and fissured; the voice is aphonic; the occasional cough is attended by expectoration of sputa which contain bright-red streaks or black clots of blood. Bloody evacuations from the bowels are occasionally observed, and, in women, uterine hæmorrhages. There is usually retention of urine, and the bladder extends above the symphysis. Bloody urine is evacuated with the catheter.

In some patients consciousness is unclouded nearly to the end, but in the majority it is impaired at the very onset and their attention

is attracted only by direct questions. With the spread of the process consciousness is lost entirely. The respirations become feeble and irregular, the pulse small and thready, bloody froth escapes from the mouth, and death ensues.

All these symptoms run their course in twenty-four to thirty-six hours. The diagnosis of this form of variola hæmorrhagica is possible from the first appearance of the diffuse redness, and hence the prognosis of an absolutely fatal and rapid course may also be made. The hæmorrhages and the impairment of consciousness increase from hour to hour. The symptoms constantly change for the worse, and in hardly any other general disease does death result so quickly. In rare cases the process lasts more than two days, but death takes place at the latest on the third day of the eruption.

Upon autopsy small or extensive extravasations are found in almost all the tissues and internal organs—the serous membranes, muscles, periosteum, the parenchymatous organs, the liver and kidneys, occasionally in the meninges, the renal capsules, etc. The free blood in the heart, veins, and parenchymata is blackish-red and thin like prune juice.

In view of the symptomatology it is easy to understand that it is sometimes doubtful whether we really have to deal with variola, especially as not the slightest sign of a small-pox efflorescence is found upon the skin.

It is to be remembered, however, that the etiological relation of this disease to ordinary forms of small-pox has been observed in not a few cases. A person who has nursed a patient suffering from ordinary variola or variola modificata is attacked, after a corresponding interval, by purpura variolosa. A third person, who has come in contact with the latter, is attacked by ordinary variola. In not a few cases the clinical identity of the different forms is also shown. In the protracted cases of purpura variolosa, which may last a week, we sometimes find upon various parts of the skin, particularly the lower limbs, small, flabby, flat eruptions, recognizable as small pox, and which may either be hæmorrhagic or free from hæmorrhages. In the ordinary, extremely rapid course of purpura variolosa there is no indication of true variola eruptions. It may be assumed, however, that their development has been prevented by the rapid fatal termination. In the words of the older writers, this is a "variola sine variolis" in the true sense. The identity of the disease is also shown by the more frequent occurrence of purpura variolosa during extensive epidemics of variola.

On the whole, purpura variolosa is rare, and years may elapse before a case is seen. It may even be absent in extensive epidemics. From 1866 to 1871 the epidemic of small-pox in Vienna never disappeared entirely. Among the 4,088 cases treated during this time in

the General Hospital I observed this form only once in its complete development. During this time two exquisite cases were observed in my private practice. In 1874, 10 cases of this kind occurred in the hospital among 209 cases of small-pox.

Apart from the malignancy of certain epidemics, no cause can be found for the occurrence of purpura variolosa.

Vaccination does not appear to offer the slightest protection against this form of the disease. If we consider the absolutely small number of cases of purpura variolosa, it occurs in a surprisingly large proportion among vaccinated and revaccinated persons and those who have already had an attack of small-pox. The victims of purpura variolosa are not old, decrepit, or cachectic individuals among the poorer classes, but are chiefly young, blooming individuals, twenty to thirty years old, who often live in the best of surroundings. Here, as in the attempt to explain the malignant course of other zymotic diseases, such as typhus, we are compelled to conceal our ignorance by assuming a peculiar individual predisposition.

Second Form of Variola Hæmorrhagica.—The prodromal symptoms resemble those of the first form and of every severe variola. On the fourth day of the disease the symptoms become violent. A prodromal eruption may be present or absent. The pains in the back are extremely severe. Great restlessness is present, with hot, dry skin and rapid pulse. Upon the same or following day the patient complains of violent pains in the lower limbs. The integument of the latter, sometimes that of the abdomen and forearms, appears swollen, as hard as a board, scarcely pits on pressure, is very tender. The finger passed across the skin feels little nodules.

By the aid of palpation and a good illumination the swelling and hardness of the skin are seen to be due to small, hard, round, slightly acuminated nodules which are situated deep in the corium. These are uniformly aggregated in immense numbers throughout the skin. On the first or second day of the eruption punctate, blackish-blue patches (hæmorrhages) appear at the apices of the individual nodules; these patches are deep-seated and shine through the epidermis. Their number increases from hour to hour, and at the same time they spread peripherally and may coalesce into extensive patches. The majority, however, remain isolated within the individual nodules. In places the integument becomes necrotic over areas of variable extent, either after a hæmorrhage or from the very start, and is converted into a discolored, dry, blackish-green mass.

The integument of the trunk and face may exhibit various appearances. Sometimes it contains a moderate number of efflorescences, which either develop normally or are hæmorrhagic from the start; at other times hæmorrhagic, rapidly increasing patches

develop on an erythematous base, either associated with the variola efflorescence or independently.

In the meantime the febrile symptoms have increased in intensity, the pulse has become very frequent, the tongue dry and fissured. The faculties become clouded, delirium and restlessness give place to coma, which gradually ends in death. Curschmann's variola hæmorrhagica pustulosa appears to refer to this form. Although it does not run so rapid a course as the first form, it is usually fatal in two to three days, occasionally in four days. In the latter event the above-mentioned efflorescences always develop distinctly upon the face and trunk, and a few flat, usually hæmorrhagic pocks also appear upon the infiltrated parts of the lower limbs. These cases always terminate fatally.

The most striking symptom is the large number of efflorescences closely aggregated upon the lower limbs and abdomen. The hæmorrhages which take place here appear to be an effect of the local circulatory disturbance following upon the dense inflammatory infiltration rather than an expression of a general decomposition of the blood.

This form approaches the typical varieties of small-pox by the production of the initial forms of variola efflorescences, and on the other hand approaches purpura variolosa in those cases in which the diffuse hæmorrhages develop on the trunk, on parts free from eruption.

According to my experience, this form of variola hæmorrhagica is even rarer than purpura variolosa. In one direction its etiology is clearer than that of purpura variolosa. It occurs only in the non-vaccinated, or in older people who were vaccinated many years before.

Apart from these two forms of variola, which I call variola hæmorrhagica on account of the early appearance and severity of the hæmorrhages and the absolutely fatal course, hæmorrhages are observed in variola under very manifold circumstances.

In some cases the hæmorrhages are confined chiefly to the individual variola efflorescences and their immediate vicinity. They appear between the fifth and the eleventh days of the disease, on the average, as hæmorrhagic contents of an already existing eruption, and as an extravasation into the papillary layer and corium of the base and surroundings of the individual efflorescences. The hæmorrhagic efflorescences are dark-red to black throughout, or only at the edges and in the immediate vicinity, are usually flabby and flat, are never tensely filled, and dry very rapidly.

Many hæmorrhages occur into the papules, and, if the latter correspond very often to the follicles, appearances like those of acne cachecticorum are exhibited in places. Such papules do not develop any further, but dry into a black mass which falls out after exfolia-

tion of the superjacent epidermis. The hæmorrhagic patches are therefore disseminated, and differ in number, not in severity or extent. Such hæmorrhages are found, in almost every general confluent variola, chiefly in the face, back, and legs.

Their origin is extremely variable. Just as confluent small-pox occurs usually among the non-vaccinated, they are found chiefly among this class. Some malignant epidemics are characterized by unusual frequency, not alone of the typical forms of variola hæmorrhagica, but also of variola confluens with or without hæmorrhages. They also appear almost constantly in hard drinkers, and on the legs of individuals who suffer from varicose veins or stand a good deal, and in whom all inflammatory processes and eruptions are attended by pigmentation and hæmorrhages. In all these cases it is better to speak of hæmorrhagic eruptions in variola rather than of variola hæmorrhagica.

The course of the disease does not depend upon the hæmorrhages, but upon the causal conditions. Life is endangered because confluent small-pox is a dangerous disease *per se*, and because any febrile disease—for example, a pneumonia—is apt to run a fatal course in hard drinkers.

The slighter the general cause to which the hæmorrhage is due, the less is the danger. A hard drinker is jeopardized to a greater extent by variola without hæmorrhages than a baker who does not drink but who exhibits hæmorrhagic variola on account of varicose veins. Likewise a confluent variola without hæmorrhagic eruption is more dangerous than a moderate variola modificata in a tuberculous individual in which the eruption is flabby and partly hæmorrhagic.

In general, however, the greater the number and intensity of the hæmorrhages the more serious is the symptom, whether as an expression of the malignancy of the contagion or of some factor inherent in the individuality of the patient.

The slightest significance attaches to those hæmorrhages which occur as the result of local increase of the hyperæmia in the prodromal erythema of variola. There is usually very slight or no development of variola eruptions upon the area which is the site of such hæmorrhages.

The attempt has been made, from a histological standpoint, to place variola hæmorrhagica in a special category (Erismann). If pieces of the skin from variola hæmorrhagica in its various forms and stages are examined, we can convince ourselves, as has been shown by E. Wagner, O. Wyss, and Zuelzer, that the development of the eruption takes place in the same way as in ordinary variola. The extravasation of red blood globules and serum is not confined to the efflorescence, but occurs superficially, into the papillæ, and deeper in the corium along the vessels. It interferes with the development

of the efflorescences only in so far as it occurs within the latter or appears at a relatively early period.

In regard to the absence or incomplete development of the pocks and the early cessation of the disease, these forms of variola hæmorrhagica constitute, in a measure, abortive forms of small-pox. In the form which will next be considered there is an excessive development of eruption.

VARIOLA CONFLUENS.

This begins, as a rule, with a severe prodromal stage. During the papular stage the fever hardly diminishes, continues high during the florid stage, and is often intensified to a typhoid condition (variola typhosa), with delirium or stupor and coma, during the suppurative stage. The papules are firmer than usual, because their inflammatory base is situated deep in the papillary body and corium, and they appear in such large numbers that they are in close proximity even during their development into vesicles. The pustules are aggregated still more closely. In places, especially on the face and hands, they form a confluent, firm projection above the skin, which is made nodular by the apices of the pustules. The skin is also enormously swollen *in toto* by inflammation of its deeper layers and by œdema. There is extreme swelling and tension of the face, lids, scalp, and hands. In these places and on the trunk the pustules may be so dense that they unite into a continuous pustular covering which may involve large areas. The corium is then exposed, and is soon covered by a yellow diphtheritic layer. Again, the skin becomes gangrenous and is converted in places into a discolored pulp on account of the dense inflammatory infiltration. Many of these pustules are hæmorrhagic.

There is always a very considerable number of pocks upon the mucous membrane of the mouth, pharynx, and larynx. The tongue, sometimes greatly swollen (glossitis variolosa), is pressed against the teeth and exhibits diphtheritic points of contact with the latter. Aphonia comes on, respiration and deglutition are labored. Curschmann has observed obstinate vomiting. The mucous membrane of the pharynx, epiglottis, and larynx is dry and brownish red, as if varnished, or diphtheritic ulcers develop. Laryngeal perichondritis usually does not occur until the stage of decrustation. There is considerable bronchial catarrh.

The general and febrile symptoms are extremely severe, corresponding to the presence of many hundred deep pustules and, in places, of diphtheritic inflammation or gangrene. During the suppurative stage—twelfth to fifteenth day—the fever is continuous ; the patients are delirious or lie in a condition of stupor from which they do not awaken until desiccation begins. They often die at an earlier

period from pulmonary œdema, pleuro-pneumonia, paralysis of the heart, suffocation from laryngeal and tracheal croup. Even during the stage of decrustation death may result from metastatic inflammations of the skin and other organs, or from exhaustion, or permanent sequelæ may be left over, such as laryngeal stricture, ocular defects, blindness, ankyloses, etc.

Variola vera and confluens are also attended by numerous *complications*. Among these I may mention the rare occurrence of aphasia, paralysis of various groups of muscles, paraplegia (Westphal and myself). When the variola runs a favorable course these conditions may recover with the subsidence of the febrile and meningeal symptoms, or during convalescence, or they may remain permanent. Albuminuria and diarrhœa are less frequent and of slight importance.

The greatest importance attaches to the metastases which occur during the stage of suppuration and decrustation. They are most frequent in the skin and subcutaneous cellular tissue. Larger and smaller abscesses (fifty to one hundred or more), furuncles, circumscribed inflammations over which hæmorrhagic vesicles form, develop one after another. Individual pock crusts are sometimes surrounded by an inflammatory zone, above which the integument is raised into a vesicle, surrounding the central crust like a wall (*pemphigus variolosus, rupia variolosa*). At other times simple pustules and furuncles (*impetigo variolosa*) develop between the drying pocks upon erysipelatous or inflamed integument. Hardly has one set been relieved by operation when a chill heralds another metastatic localization. Despite careful examination nothing can be seen, but at the end of twelve or twenty-four hours moderate redness of the skin with subjacent fluctuation is observed upon some part of the body, especially over the knees. Upon incision an enormous amount of ichorous fluid mixed with necrotic shreds of tissue is evacuated. Subsequent necrosis of the bone is rare. These abscesses and phlegmons usually heal very rapidly. They may be complicated with lymphangitis and erysipelas, adenitis with or without suppuration. The attendant vital losses, the fever, pain, insomnia, and anorexia, may cause death by exhaustion or protract convalescence for six to eight weeks. Death may also be due directly to metastasis of internal organs (pleurisy, pericarditis).

The ocular affections to which variola may give rise occasion the greatest anxiety to the attending physician. Sight is often destroyed in variola vera and confluens. I have often observed xerosis of the cornea, keratomalacia, abscesses and diffuse suppurative keratitis, pustules terminating in ulceration, hypopyon, iridocyclitis, perforation of the cornea and prolapse of the iris, panophthalmitis. All these affections occur as metastatic processes during the period of suppuration or later, and therefore constitute complications and

sequelæ. Hans Adler has published an excellent work on this subject which contains the most recent bibliography.

When the patient has already suffered from acute or chronic diseases of the skin, variola usually causes their partial disappearance. Thus, the acari of scabies are destroyed, as a rule, and the eczema symptoms also subside, but during the period of convalescence the ova which have been left behind develop and the scabies symptoms again begin. Diseases of the skin. such as eczema, psoriasis, and lupus, which are attended by thickening of the epidermis and chronic congestive conditions of the corium, favor a more copious development of pocks upon the affected parts. After the crusts have fallen off the skin disease has also disappeared or become less severe. Usually, however, it returns at a later period. The symptoms of prurigo, ichthyosis, and early syphilis also diminish or disappear during small-pox, but are not permanently cured.

I have often observed the development of small-pox during the convalescent stage of typhoid fever. I have already discussed its coincidence with scarlatina and measles. The majority of cases occur in such a way that the eruption of scarlatina or measles appears after the variola has already reached or passed its height. But even such cases involve the coincident presence of both viruses in the organism, if we take into consideration the duration of the period of incubation.

We may apply the term sequelæ to all those affections which I have described as complications and metastatic processes in variola, if they persist for a long time after the variola process ; for example, certain affections of the eyes and joints, also tuberculosis in predisposed individuals. Among the common sequelæ we may mention seborrhœa, pigmented patches and cicatrices.

The seborrhœa affects chiefly the face, particularly the nose and scalp. Its symptoms and its special characteristics, as seborrhœa congestiva (Hebra), with a possible termination in lupus erythematosus, have already been described. Seborrhœa of the scalp is accompanied by rapid falling out of the hair, but this is soon replaced by a new growth. Permanent loss of hair affects only those parts in which the hair follicles have been destroyed by deep suppuration.

In those who have suffered from variola vera and confluens of the face the nose is traversed by deep furrows, above which project warty remains of the integument. Other warty projections are due to an accumulation of sebum in the sebaceous glands, or to acini which have been occluded by cicatrices. Still others result from closely aggregated variola papules which have not gone on to suppuration, but persist for some time as papillary and epithelioidal hyperplasiæ. All of them are known as warty pocks (variola verrucosa).

The traces of small-pox constantly remain over as pigment patches and cicatrices. The former are as large as a lentil, yellowish brown, the centre white and somewhat depressed, paling very slightly under the pressure of the finger. They are found in all sites of superficial (*i.e.*, within the epidermis) pocks, whether the vesicular ones of varicella or the pustular ones of varicella and variola. Their color is due to an accumulation of pigment in the rete and to the continued congestion of the papillary vessels. For this reason they look bluish red in the cold. After the lapse of months the normal color returns.

The cicatrices have the same shape and size as the pigment patches, at first bluish red, later glistening white and slightly depressed. They develop only in those places where the pustules have extended to the papillary body and a part of the latter has been destroyed by suppuration. When they are closely aggregated the intervening unchanged integument forms islets and bridges which make the part look like a network. There are no cicatrices, however, which are characteristic of small-pox; they resemble those which are due to similar efflorescences, such as syphilis, acne, etc. Their source can only be inferred from their uniform arrangement and distribution.

The *anatomy* of variola exhibits many peculiarities which distinguish its efflorescences from the analogous stages of non-variolous inflammation—for example, the papules, vesicles, and pustules of eczema. In my opinion this does not depend upon nutritive processes which are foreign to the other inflammatory conditions, but is the mere result and expression of the *typical* local process, which develops and terminates within a certain definite period. The type itself, however, is a mystery, as is its causation. Those who think they have found the bacteria, the micrococci and microspores of small-pox in the contents of the pocks and in the underlying corium are prepared (Weigert) to give a direct explanation of the finer anatomical processes from the presence of these contagious particles. But, apart from the fact that this relation is not recognizable by other observers, there is also a great difference of opinion in regard to the other actual findings and their significance.

The conditions are exhibited most clearly in the typical variola eruption, which is situated within the epidermis, and whose development does not extend beyond it.

The eruption begins with a swelling of the papillæ in the region, upon which the papule is to form; this is due to hyperæmic, serous, and moderate cellular infiltration. This finding in the initial stage, which Auspitz and Basch first described, I have even observed in specimens taken from purpura variolosa. But the greater part of the papule, which is elevated above the level of the skin, is formed by

proliferation of the Malpighian cells. It begins with cloudy swelling of the rete cells. Rindfleisch gives an excellent description of this change in the epithelial cells. They become either simply finely granular, or they undergo a dark granulation which entirely conceals the nucleus, enlarge, and are converted into shapeless clumps. The epithelial cells also undergo this change in other processes. Weigert believes that this conversion of the rete cells into non-nucleated clumps is directly due to contact with the bacteria which pass from the corium into the epidermis, and constitutes a "diphtheroid" destruction. Renaut, however, who describes pustulation in conformity, in other respects, with the views of the older writers, speaks of micro-organisms in the upper prickle cells which are about to undergo proliferation.

Rindfleisch describes the middle layer of the rete Malpighii as the starting point of the proliferation. Unna, on the other hand, indicates the stratum lucidum—*i.e.*, the lowermost horny cell layer —as the one which, by proliferation and further conversion of its cells, forms the subsequent true pock. There appears to me to be no doubt that the upper rete layers, whether with or without the stratum lucidum, take part in all typically developing pocks.

While these cells are passing from cloudy swelling to degeneration, a proliferation also begins in the surrounding cells, whose increased masses form a sort of shell for the middle layers. But in the centre, where Weigert's diphtheroid focus and lower umbilication are situated, the connection between the rete cells is loosened by the serous exudation which arises from the papillæ. This exudation, penetrating the epidermic layers, pushes the uppermost horny layers before it as a covering. This results in a vesicle with transparent contents

The exudate separates the uppermost cell layers of the mucous layer, but mainly the lower horny cell layers. These cells, which are in part swollen, form the walls and ridges of a network in the interior of the vesicle (Fig. 19). Ebstein divides the meshwork into a superficial one formed of horny cells, and a deeper one of rete cells. The latter are not involved until the pock is further developed and extends more deeply.

The meshes are filled with clear fluid containing a moderate number of exudate cells, débris of epidermis, amorphous masses, and small shining corpuscles supposed to be specific in character (micrococcus, Haller, Klebs, Cohn).

The production of umbilication in the first stage of some pocks (not all are umbilicated) is explained by Auspitz and Basch on the theory that the distention of the central part of the pock does not proceed uniformly with the enlargement of the peripheral portions of the papule, the latter being due to epithelial proliferation. Weigert

believes that the diphtheroid epithelial ridges in the centre of the pock resist stretching by the exudation for a longer time than the peripheral cells, and that the apex of the pock adheres firmly until it finally ruptures. Renaut attributes umbilication to compression of the blood capillaries exercised by the distended lymph capillaries. I agree with Hebra and Rindfleisch, who attribute the umbilication to the horny portion of the opening of a follicle or sweat gland which traverses the pock (Fig. 19, *a*) ; this acts by retaining in place the surrounding parts. Umbilication is not peculiar to the pock, but is found under the same anatomical conditions in simple inflammatory eruptions. It always disappears at the beginning of suppuration.

The conversion of the vesicle into a pustule is due to the accumulation of pus cells within the bleb and clouding of its contents. The pus cells are derived in part from the vessels of the papillæ (wander-

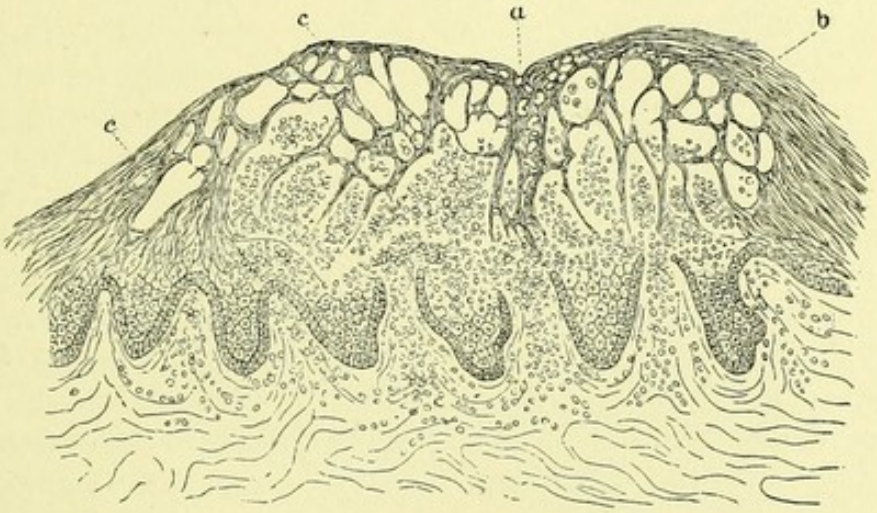

FIG. 19.—SECTION THROUGH A POCK IN THE STAGE OF PUS FORMATION (AFTER RINDFLEISCH).

a, umbilication with duct of sweat gland ; *b*, meshwork in the epidermis ; *c*, smaller meshwork, containing lymph and pus cells.

ing cells), and in part they are derivatives of the proliferating rete cells. The trabeculæ of the meshwork disintegrate, partly from purulent degeneration, partly from the pressure of the increasing amount of fluid. The middle and lower parts of the pustule are occupied by an irregular pus cavity, into which project epidermic shreds of the superficial walls and of the rete cells situated on the sides. The pustule has now reached the height of its development.

In the following stage of desiccation the inflammatory congestion of the papillæ and the resulting exudation diminish. The rete cells which surround the pus focus from the sides and below give birth to young cells, which are not disturbed in their physiological conversion into horny cells. In this way a horny cell layer forms on the sides and below, and unites with those of the top of the pustule to form a complete capsule (Fig. 20, *e*) around the pus focus, which is thus entirely excluded from the nutrient strata of the skin.

On account of the drying (evaporation or absorption) of the fluid contents, the pustule and its covering form a crust. This crumbles mechanically after the lapse of days or weeks, and the thicker the overlying horny layer the later the disintegration, as in the palm of the hand and the sole of the foot.

The papillæ at the base of the pock are swollen and prominent at the beginning of papulation, but later they become compressed and flattened by the proliferating rete cells of the base of the vesicle, or, according to Unna, of the stratum lucidum. They are seen compressed in Fig. 20, in comparison with the normal papillæ, *g*, at the periphery. In these typical forms the removal of the crusts will be followed only by a depression, which is colored brown from increased pigmentation of the rete (below *e*, Fig. 20) ; but no cicatrix will result, inasmuch as the papillæ remain intact.

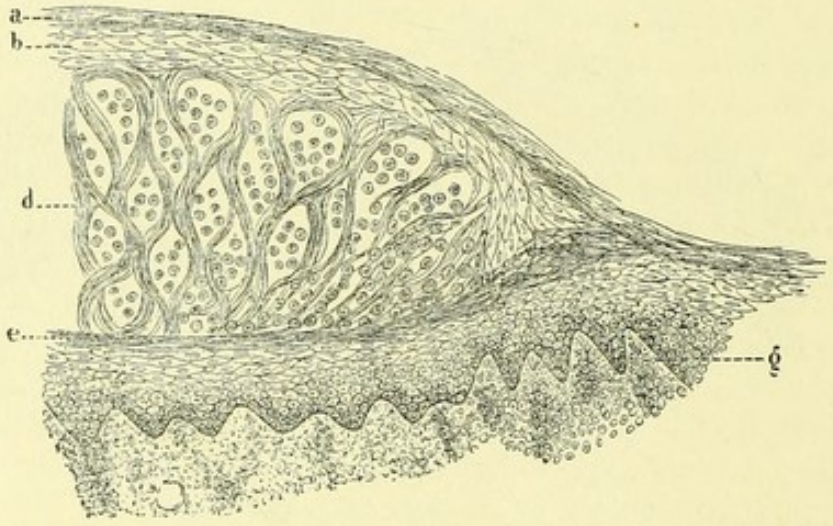

FIG. 20.—SECTION THROUGH AN ENCAPSULATED PUSTULE (ONE-HALF) (AFTER AUSPITZ AND BASCH).

a, outer epidermis ; *b*, rete cells overlying the meshwork ; *d*, meshwork containing pus cells; *e*, new formed epidermis cells ; *g*, papillæ, infiltrated with cells, forming the base of the pustule.

This is the anatomical course of all, or at least the majority, of pocks in varicella and varioloid, and also of many in variola vera.

At other times the inflammatory infiltration of the papillæ which precedes the development of all pocks reaches such a degree that some of them, or even the adjacent part of the corium, undergo purulent and necrotic destruction. On transverse section such a part has a uniform white color (diphtheritic pock), because, as Rindfleisch's injection preparations show, even the afferent vessels are compressed by the exudation and cellular infiltration. This part of the papillæ and corium thus undergoes necrosis, and the products of degeneration, together with the pus cells and the cells derived from the overlying rete, increase the contents of the pock. In such forms the pus cavity extends into the papillæ and even deeper. This must result in a scar because a connective-tissue portion of the integument has been

destroyed. This demonstrates our inability to prevent cicatrices by the application of ointments, etc., at the beginning of suppuration, because they depend upon the deep situation and the intensity of the first inflammatory processes.

The eruption of variola hæmorrhagica develops in the same way as in variola pustulosa, except that, in addition to white blood corpuscles and serous exudation, the pocks also contain red blood corpuscles. Hæmorrhagic foci are also found in the papillæ and corium.

In purpura variolosa we find, in addition to cellular infiltration of the papillæ (evidently the beginning of the development of papules), merely scattered extravasations in the corium and subcutaneous tissue.

Accurate investigations have not been made with regard to the finer changes occurring in the pocks which are located on the mucous membrane of the mouth, pharynx, larynx, bronchi, and œsophagus.

The other anatomo-pathological changes which are found upon autopsy correspond to the clinical complications—for example, pneumonia, pulmonary œdema. In many fatal cases the anatomical basis of the immediate cause of death remains unexplained. The post-mortem findings in the internal organs are so contradictory that they do not warrant any general statements. In purpura variolosa Hebra has seen the liver, heart, lungs, and spleen also infiltrated with hæmorrhages, and the spleen often appeared to be converted into a fibrinous clot of blood. Curschmann and Ponfick, on the other hand, state that in purpura variolosa the spleen is small and firm, and that the abdominal organs generally exhibit an appearance different from that found in variola pustulosa. Perhaps these differences of opinion will be extinguished when the number of observations increases.

In variola hæmorrhagica extravasations of blood are sometimes found in the nerve sheaths and meninges (Neumann, Zuelzer, myself), and importance is also attached by recent observers to questionable bacteria and micrococcus colonies. One author states that they penetrate from the surface of the epidermis and produce every pock, forgetting that there would then be no reason for the preceding fever. Zuelzer also calls attention to the rigidity and friability of the walls of the vessels as the result of granular degeneration, especially of the muscular coat.

Papules and nodules have also been found in the lungs, beneath the pleura, and in the liver, partly in human beings who have died of small-pox, partly in animals that have died after injection of blood from individuals dying of variola hæmorrhagica. These are probably metastatic inflammatory foci (also colonies of bacteria according to Weigert), and are not true pocks.

LECTURE XV.

SMALL-POX (*concluded*).

DIAGNOSIS—PROGNOSIS—INFLUENCE OF VACCINATION ON THE SEVERITY OF THE DISEASE—ETIOLOGY—TREATMENT—PROPHYLAXIS—VACCINATION: ORIGINAL AND HUMANIZED LYMPH—VARIOLA VACCINA—NORMAL AND ABNORMAL COURSE.

THE *diagnosis* of well-developed variola is not difficult. Under certain circumstances, however, it is possible that disastrous and disagreeable mistakes may be made. During the prodromal eruption, or even on the first day of the eruption when the first papules appear, no one can be blamed for being in doubt whether he has to deal with erythema papulatum or, taking into consideration the febrile and catarrhal symptoms, with morbilli papulosi. On the following day the symptoms become clearer, because in variola the papules are considerably enlarged and are undergoing vesiculation, particularly in the face. The predominant development of papules in the face also speaks in favor of variola. Vesicular variola (varicella) must sometimes be distinguished from impetigo faciei or beginning acute pemphigus. Variola in the pustular stage will seldom be mistaken. More frequently a pustular syphilide, or rarely the pustules occurring in glanders, will be regarded as variola. The resemblance to syphilis is shown by the term big-pox, applied to the latter. In the syphilide (varicella syphilitica, syphilis pustulans varioloides) we always find efflorescences in different stages of development, large nodules from the size of a lentil to that of a pea, efflorescences which have reached their height and are undergoing necrosis in the centre, and a distinct, firm wall of nodules at the periphery of the latter. Such a mistake can last only for a few days, because even the novice will be struck by the uniform persistence of the syphilitic eruption compared with the rapid evolution and involution of small-pox. In glanders there are always large, furuncle-like nodules and abscesses in addition to the superficial pustules. Decided fever may be present in both affections, though it is usually very slight in moderate variola or varicella. Differential signs of variola will almost always be discovered on examining the mouth and throat.

Moderate cases of variola in which pointed pustules, usually cor-

responding distinctly to the follicles, develop rapidly upon the face, while late, scattered, and abortive efflorescences appear upon the trunk, are apt to be regarded as acne pustulosa. In such cases the rapid and coincident appearance of pustules favors the diagnosis of variola, while the lack of uniformity in the development of the symptoms, the chronic course, the presence of comedones, inflammatory nodules and abscesses, are characteristic of acne. Medicinal acne, due to the internal use of iodine and bromine, develops acutely and is therefore difficult to distinguish at the onset.

The *prognosis* of variola has already been considered in part in the discussion of the symptoms. Violent prodromal symptoms, if they remain within the typical limits, do not warrant an inference concerning the severity of the disease, but in purpura variolosa the fatal termination is evident from the start. Apart from the latter process, the number of pustules is an important prognostic feature in all forms, in addition to age, the condition as regards vaccination, the character of the prevailing epidemic, and (in women) pregnancy and the puerperal condition.

Varicella always runs a favorable course; varioloid is milder in the vaccinated than in the non-vaccinated, while variola confluens is extremely dangerous even in the vaccinated, and its termination is greatly influenced by the surroundings; variola hæmorrhagica is always fatal.

As regards age, infants almost always die when attacked by variola, on account of the interference with nursing, while older, vigorous children often survive severe attacks. In old age there is a predisposition to flabby and hæmorrhagic small-pox, and the prognosis is always doubtful, as it is in hard drinkers, who usually die from the severity of the disease or from pulmonary œdema during delirium tremens.

Pregnant and puerperal women are more endangered by severe small-pox than other women are. In 1866–1867 I observed one hundred and twenty pregnant and puerperal women among seven hundred cases of variola in women. Of the former, one in five died; of the latter, one in twenty-two. The greater mortality resulted from complicating puerperal processes to which abortion and premature delivery give rise. The majority of the latter occurred in the seventh and ninth months of pregnancy.

Successful vaccination is an important factor in prognosis. Vaccinated individuals suffer usually from the milder forms, but the chances are better even when they are attacked by variola vera. It must not be forgotten, however, that, under similar external conditions, the vaccinated may suffer from severe variola, the unvaccinated from mild forms. I cannot enter in detail into the statistics of thousands upon thousands of cases, collected by individual physicians

and commissions, which afford the most undoubted proof that variola is weakened by vaccination and that the latter offers a relatively great protection against the disease. The expert, on entering a small-pox hospital, will at once recognize the non-vaccinated ; they suffer from severe forms of the disease and the pustules are large and numerous. In countries in which vaccination is not general, the ravages of an epidemic are as disastrous as in former centuries. In our city the average mortality among the non-vaccinated is thirteen. twenty to forty-five per cent ; among the vaccinated two, five to fifteen per cent. according to the malignancy of the epidemic and the number of cases observed.

Hebra and Oppolzer have emphasized the fact that the previous occurrence of variola has a bad prognostic significance, as they have repeatedly seen patients die in a second or third attack, although they exhibited the scars of previous attacks.

Finally, due importance in prognosis must be attached to complications and sequelæ, such as phlegmons, diseases of the joints, heart, and lungs, etc.

The *etiology* of small-pox has hardly been advanced further than that of the other infectious diseases. We merely know that it is due to a specific virus which emanates from the patients and is carried through the air ; that it is contained in the contents of the efflorescences, and, whether fluid or dried in crusts, can be conveyed to other individuals by subepidermal inoculation ; that, after an incubation of twelve to fourteen days, it produces a general disease and reproduces itself. It is probable, though not proven, that it is also contained in the blood of the patient, but not in the other secretions. When inoculated in animals (sheep, horse, ass, goat, cow) it produces an analogous if not identical disease, either local or general. It is certain that when this is reinoculated in man it gives rise mainly to a local process (vaccinia), not to a constitutional affection.

The view that the virus is organized is constantly gaining ground. Since Keber many look upon small (0.001 millimetre) granules in the pock lymph as the carriers of the virus, if not the virus itself. Ferd. Cohn supports this view, and has shown that the corpuscles are capable of proliferation and are a sphærobacterium peculiar to variola. But despite this great authority and the analogous discoveries by Luginbühl, Klebs, Weigert, Zuelzer, De Toma, Alvaro, etc., and despite experiments of all kinds and apparently successful pure cultures, injections into animals, etc., it cannot be said that the specific micrococcus of variola has been discovered beyond a doubt. In the last few years an entire series of cocci and bacteria have been found normally in the epidermis, and as a matter of course these may be present in the eruptions of variola and vaccinia.

The virus is usually absorbed by the respiratory tract. Vario-

lation, which was formerly practised, proved that infection through injured parts of the skin is also possible. I may here remark that, as the result of the contamination of slight wounds by variola products, violent lymphangitis, erysipelas, phlegmons, pyæmia, icterus, even death, may occur. I have seen a mild case, a colleague has observed a very complicated case, of this kind.

In exceptional cases direct inoculation of the small-pox virus by accidental contact or rubbing the skin of a healthy individual against that of a small-pox patient gives rise to true inoculation variola. This was formerly aimed at by inoculating with the contents of pocks (*vide* Variolation). I have seen four cases of this kind in the last few years. The first occurred in a woman of thirty-three years who had nursed a child suffering from small-pox. The dorsum of each hand contained fifteen to twenty vaccinia-like, large, umbilicated pocks. At the end of ten days erythema variolosum, fever, and general moderate variola set in. The second case occurred in a woman of thirty-two years who had also nursed a child sick with variola. The third case occurred in a woman of twenty-two years, the fourth in a child of three and a half years, both of whom had been in direct contact with variola. In the last three cases the variolation eruption was very profuse, but confined to the face and neck, so that it closely resembled impetigo contagiosa. A moderate general eruption occurred only in the child at the end of ten days. All four individuals had been vaccinated.

There appears to be no doubt that the virus may be conveyed by intermediate persons and utensils. It is destroyed much more readily by very high than by very low temperatures.

Variola spreads chiefly through the medium of the sick, more rarely of intermediate persons. In some localities, as Rendu demonstrated in regard to the city of Lyons, the spread of the disease may often be traced from case to case. These individual cases produce localized endemics, and finally epidemics which may traverse entire countries. In Vienna, as in all large cities, sporadic cases are never absent. From 1866 to 1876 an almost uninterrupted epidemic prevailed, and in 1870 to 1872 it had developed into the most extensive epidemic of the century as regards extent, malignancy, and mortality. Among 25,000 deaths in Vienna in 1872, 3,800 were due to small-pox. In 1864, among 21,000 deaths, only 137 were due to small-pox. At the height of an epidemic severe and fatal cases are more frequent, but different epidemics vary in many respects; in the epidemic of 1870, for example, the frequency of variola hæmorrhagica was astounding. In Vienna the largest number of cases occur in the winter months, from December to the end of February.

It is a matter of constant observation that, in a population which is thoroughly vaccinated, an epidemic of small-pox does not produce

such ravages as in a non-vaccinated population. I have already stated that vaccination diminishes, permanently or for a long time, the receptivity to variola. This is also true of individuals who have already had an attack of small-pox. But two or three attacks in one person have often been observed, and such individuals are more endangered in the later attacks. This simply shows that they possess a special predisposition. I have reported one case in which, two weeks after an attack of varioloid, another attack developed, and Kramer has reported a similar case.

The individual predisposition varies greatly, being slightest in infancy and old age. The fœtus in utero may be attacked, and is then almost always aborted, either dead or alive. It is doubtful whether this is always preceded by disease of the mother. I cannot say that pregnant and puerperal women exhibit a greater predisposition to the disease, but they are certainly more endangered. This is also true of negroes, in whom the disease is always very malignant, probably because they are generally unvaccinated. It is an interesting fact that in the thirty years since which time the small-pox ward has formed part of the dermatological service of our hospital, not a single one of the regular physicians and attendants has suffered from small-pox. On the other hand, several physicians and students who merely attended lectures have been attacked every year. An individual may be temporarily immune against small-pox, may escape at one time and be infected on another occasion when the danger is apparently less.

A wide field is open to *treatment* in a process which is so rich in symptoms as small-pox, but the results attained are by no means as satisfactory as might be desired. Some believe that the fine granules found in the pock contents or the blood of variola cases are bacteria or micrococci and constitute the variola virus, and that by the administration of salicylate of soda or xylol (Burkart, Zuelzer) these schizomycetes are destroyed and the morbid process ameliorated. I doubt the value of the supposed results. Neither is much to be expected from large doses of quinine, tartar emetic, vaccination at the onset of small-pox, or the subcutaneous injection of vaccine. In fact, we must rely solely on symptomatic treatment.

As the symptoms in small-pox of moderate grade run a typical course to a favorable termination, very little treatment is necessary. Moderate temperature of the room, free ventilation, even in cold weather (in the interest of those who come in contact with the patient), cooling drinks (water is very refreshing when the buccal cavity contains pocks), suitable diet—these are all the necessary measures.

The interference with deglutition, and the inflammation due to variola of the mucous membrane, may be treated with gargles (potass. chlorat. or alum. 5 : 300 aquæ or infus. tiliæ with tinct. laudan.

croc. 2.50 and mel. rosæ 10). In severe cases the patients are hardly able to swallow, and it is best to give fresh water and pieces of ice.

As soon as decrustation begins the patient should take a warm bath daily or every other day and should be well washed with soap.

More vigorous treatment is required in variola vera and confluens. In variola hæmorrhagica all remedies (xylol, sesquichloride of iron, ergotin internally and subcutaneously, etc.) are useless, as it terminates fatally in the prodromal and first eruptive stage. Not much can be done against the often violent symptoms of these periods—the restlessness, fever, vomiting, pains in the back, cardialgia, oppression. I warn you particularly against chloral hydrate, bromide of potassium, opiates, and subcutaneous injections of morphine, in order that the vital forces may not be depressed. I can recommend opiates only when a fatal termination is certain, as in purpura variolosa, or when the patient attempts suicide or is violent, or in delirium tremens, or when he is not tractable to other means. Curschmann recommends chloral hydrate per enema (chloral hydr. 6.0–8.0, aq. destil., mucil. g. arab. āā 25.0).

Various drugs may be used at pleasure to relieve auxiliary symptoms, such as aqua lauroceras. in nausea, cold compresses in case of a feeling of heat in the head, brandy, camphor, etc., in collapse.

In the further course of the disease the large number of pustules and the concomitant dermatitis occupy the most prominent place among the symptoms. With them are intimately associated the fever, insomnia, delirium, coma, or sudden death from paralysis of the heart or lung involvement. Hence these morbid factors cannot be affected directly.

The more numerous and deep the pustules the greater the subjective symptoms, pain and tension, and the greater the danger of metastases in the stage of decrustation. For these reasons, and also in order to prevent the formation of cicatrices, attempts have always been made to combat the formation of pustules, and to accelerate their involution and desiccation by coagulation of their fluid contents.

An old plan is that of pricking the pustules or cauterizing them with bluestone (ectrotic treatment). When the pocks are few this is unnecessary ; if numerous, it is impracticable or useless, or may even be injurious.

To diminish the painful tension of the face, hands, and feet we may recommend the application of linen smeared with simple ointments, inunctions, etc., or preferably cool wet compresses—compresses of water-glycerin or rubber cloth.

More importance attaches to those measures which are intended to secure abortive desiccation of the vesicles (before suppuration) and

of the pustules. It must not be forgotten that deeply situated pocks are attended by suppuration of the papillary body, and therefore must be followed by cicatrices, while superficial pustules (in the rete) will heal without cicatrices. We may accordingly judge of the value of various applications (ointments, tinctures) and of shutting out the light, etc., in preventing pock marks. In addition to the above-mentioned cold compresses and simple salves, unguent. cinereum, empl. hydrargyri, applications of tincture of iodine, corrosive sublimate (sublimate 0.20, aquæ destil. 100 or collodium elasticum 50.0). Sublimate baths (5.00 to 300.0 water, to be poured into the bath) have also been recommended ; the danger of salivation should not be forgotten. Lister's liniment (acid. carbol. 1.0, olei olivar. 8, cretæ alb. pulv. 2.00), which we employed in the sixties, has also been warmly recommended (Schwimmer), but we have seen no brilliant results Nevertheless I may recommend such applications (particularly compresses of liquor Burowii, ten per cent) in so far as they prevent tension and the retention of pus, and thus avoid, in part, the danger of erysipelas and metastases.

In severe cases of variola I can warmly recommend the continuous baths, which were first advocated by Hebra. From the ninth day, the beginning of suppuration, the patient may be placed daily in a lukewarm bath for two to four hours, the water being kept at an agreeable temperature. When removed from the bath the entire body is dusted with powder. This is not troublesome, inasmuch as very sick patients can enter and leave the bath alone, and if necessary can be carried into it. The most striking effects are the rapid sinking-in of the pocks and cessation of tension, the acceleration of the desiccation and decrustation, so that cases in which the latter might be expected toward the end of the fourth week have finished decrustation on the fifteenth or sixteenth day. Hebra has kept the most severe cases, with pleuro-pneumonia, in the bath for days and nights, and seen recovery, and I have treated numerous cases in this way. The great advantage of this plan is the undoubted prevention or diminution of metastatic inflammations of the skin, abscesses, and gangrene. According to our experience such metastatic processes also run a more favorable course in the continuous bath Otherwise they must be treated according to general surgical principles. Abscesses must be opened as soon as they are recognized by redness or pain and slight fluctuation.

Among the ocular affections, metastatic keratitis, iritis, and hypopyon require prompt treatment. Puncture of the cornea, instillation of atropine, compress and bandage, inunctions of belladonna (extract. belladonnæ 0.50, unguent. cinereum 10.0) above the eyebrows, etc., are indicated.

In intense variola of the larynx, with aphonia and incrustation of

the mucous membrane, I have never saved life, even by performing tracheotomy.

Seborrhœa, left over after variola, is to be treated according to the recommendations made on page 127. Warty cicatrices, islets and bridges of skin on the nose and forehead, should be removed with scissors.

The *prophylactic* measures are, in part, the same as in other infectious diseases, viz., strict isolation of the patients and disinfection of their living rooms, of their clothing, utensils, etc. In addition to free ventilation, chloride of lime should be placed in the sick-room or carbolic spray used. But the most important means of prophylaxis is inoculation with vaccine, or

VACCINATION.

The outbreak of pocks has been observed occasionally in many domestic animals—the cow, pig, horse, goat, dog, and monkey. In the cow they are situated on the udder and teats. They constitute a purely local affection and probably develop only by direct infection. The virus is not volatile, and hence is conveyed to other animals and to man only by direct contact with a wounded part of the integument.

This infection is sometimes observed in men. I have twice seen an eruption of vaccinia in cowherds. It appeared in the shape of flat vesicles, surrounded by a red zone, filled with clear lymph and partly umbilicated ; these were scattered or arranged in groups upon the hands and arms. They healed in two weeks after becoming cloudy and undergoing decrustation.

In sheep the pock (ovine) also occurs as a local affection, but occasionally as a general disease, which is contagious and may engender extensive ovine epizootics to which large flocks fall victims. It has been recommended that the sheep be vaccinated with human or animal pock. This is not done generally, both on account of the attendant practical difficulties, and especially because, in such inoculations, general ovine occasionally develops instead of the local pocks and gives rise to an ovine epizootic.

Such a condition is not to be feared in inoculating vaccine into the human subject. This causes only a local eruption, and the lymph from the vaccinated individual can only be conveyed by direct contact to man or other animals. The inoculation of variola contents, on the other hand, not alone produces local variolation pustules, but general variola sometimes develops after a corresponding period of incubation.

Immediately after Jenner's publication (1798) cow-pox alone was directly inoculated in man (primary lymph), but this method was gradually abandoned on account of the cost of the material, its fre-

quent inefficiency, and the occasional occurrence of violent inflammatory symptoms. Then humanized vaccine, obtained from human pocks produced by primary cow pocks, was universally employed for vaccination.

The protection afforded was directly proven by inoculating, without effect, vaccinated children with small-pox, as is shown by Peter Franck's reports (1801), published by Auspitz. But the sanguine expectations of the first years of vaccination were not fulfilled, as you all know that even vaccinated individuals may suffer from small-pox. Variola itself does not afford absolute protection against a second or third attack. An enormous mass of material, however, proves the relative, considerable protective power of vaccination, and in the face of this opposing arguments are useless.

The opinion has gradually gained ground that in some individuals this protective power lasts for life, but as a general thing it is gradually weakened, and in many does not last more than ten to twelve years. Hence revaccination is necessary.

The infection of some vaccinated individuals by variola has been attributed to the fact that the humanized lymph has passed through so many generations since its derivation from the original cow pock that it has lost its protective power, and it is therefore advisable to freshen it occasionally by retrovaccination of the cow. But in some of the wards in the chief Vienna institute for vaccination the lymph has been propagated from the original lymph sent by Jenner (1802), and this has apparently not lost its protective power. But, although the regeneration of the cow pock by retrovaccination does not appear necessary, it has been repeatedly recommended and often successfully performed.

Finally, it has been held that vaccination with humanized lymph may convey various constitutional diseases—scrofula, rickets, tuberculosis, and especially syphilis—from one child to another. This reproach is only justified in the case of syphilis (vaccination syphilis). But the number of these unfortunate accidents is extremely small in comparison with the millions of innocuous vaccinations; and, furthermore, careful examination of the cases has shown many errors—in part the children had been syphilitic prior to vaccination, in part the accident could have been avoided by the exercise of proper care on the part of the physician. A few cases remain unexplained, and this is also true of the mode of infection. Viennois' belief, that the only cause of the occasional communication of syphilis is the admixture of blood with the lymph, is undoubtedly incorrect, because even the purest lymph contains a few blood corpuscles.

The objections to humanized virus have been sufficiently strong to lead, in some quarters, to the use of original vaccine for vaccination. In Naples an institute for cow-pock vaccination has existed

for more than fifty years, and since 1864 they have also been erected in France, Belgium, Germany, and recently in Austria-Hungary. The calves are vaccinated on the abdomen, and the lymph. obtained by pricking or squeezing the base of the pock, is employed directly or is preserved in a dry or fluid condition. Although the reports are very favorable, it appears to take with greater difficulty and there is greater danger of complicating inflammations. erysipelas and gangrene, even with a fatal course. As a matter of principle, however, we can only favor vaccination with primary lymph, because in the hands of skilled physicians it will be free from deleterious effects and will furnish protection to those who have an antipathy to humanized lymph.

The cocci found in the lymph are supposed to be the active agent. Neither the most recent investigations (M. Bauer-Weichselbaum) nor the assumed successful pure cultures (Quist, Lawrence, Hamilton, L. Voigt, Bareggi) have been able to demonstrate conclusively the specific organism of vaccine. The experiments of Voigt, who produced a local eruption and immunity against vaccine in calves by pure cultures, appear to have come nearest to a solution of the question.

Vaccination is done from arm to arm, or with fluid lymph kept in glass tubes, or with lymph dried upon bone and other substances. If necessary the fluid lymph may be diluted with glycerin and water, of each two parts (Müller). The dried lymph is dissolved, before application, in a drop of water or in the serum which exudes from the point of vaccination. In late years the lymph has been made aseptic by admixture with thymol, corrosive sublimate, etc. At all events the site of vaccination and the instruments should first receive an antiseptic cleansing. In direct vaccination we take the exuded lymph from superficial pricking of vaccination pustules on the seventh or eighth day. By means of the vaccination lancet and subepidermal pricking it is conveyed to the skin, which has been scarified superficially with scalpel, lancet, etc. This is done on the extensor aspect of the arm ; in girls the location should be so high that the scar will not be seen on wearing short sleeves. Two places on each arm will suffice Healthy children cannot be vaccinated too early, especially during an epidemic or if the danger of small-pox is imminent—for example, among the children of physicians. I vaccinated my own children during the first week of life, and the process was unattended with fever.

If vaccination runs a normal course, small red papules appear on the third or fourth day, develop in five to seven days into vesicles, and on the seventh or eighth day may be as large as a penny, tense, often umbilicated and transparent. They are surrounded by a moderately red zone. After the ninth day the contents become

cloudy and dry into a crust, which falls off in ten to fourteen days, leaving a scar.

A "good" scar is not always a criterion of successful vaccination. During this time it is well not to bathe the children, in order that the development of the pocks should not be disturbed by maceration or mechanical injury. The acme of the process is generally accompanied by moderate fever.

In anomalous cases the vaccination eruption remains in the papular stage without developing into vesicles (variola vaccina atrophica). Occasionally the site of vaccination and its vicinity are occupied by itching papules and vesicles, which are soon scratched off by the child (eczema pocks, variola vaccina herpetica); or there are large vesicles, which dry without leaving a scar (vesicular pocks, variola vaccina pemphigoides); or furuncles may develop. After the removal of the pock crust a sore spot sometimes remains and secretes serum and pus for weeks and months; it enlarges peripherally, the base is hard and infiltrated, covered with proliferating granulations and, if not cared for, with thick crusts. This may give rise to swelling of the axillary glands and simulate a syphilitic ulcer. It heals after cauterization with the solid stick, or scraping with a sharp spoon and the application of a mild astringent dressing (kali caust. 0.05, aq. destil. 100.0, or ung. simpl. 25.0, argent. nitrat. 0.04).

We sometimes notice the development of auxiliary pocks (vaccinolæ)—*i.e.*, efflorescences similar to vaccination pocks, which appear at the same time or somewhat later upon non-vaccinated parts, chiefly the arms, shoulders, and thorax. They may number twenty to thirty, and are either separate or aggregated in clumps.

The most frequent complication is roseola vaccina, a redness which starts from the arms during the course of the vaccination pocks and extends over a large part of the integument. It appears here and there in isolated patches, attended with moderate fever, but without any bad effects.

A dangerous complication is erysipelas (variola vaccina erysipelatosa), which, starting from the point of vaccination, may attain large dimensions, become phlegmonous, and even lead to gangrene. It may also occur in adults (particularly in revaccinations), and may terminate fatally; it is more frequent in children, particularly after vaccination with primary lymph. In children a fatal termination is not uncommon. It is more frequent during the prevalence of erysipelas in the surgical wards of a hospital. It is sometimes due to impure virus, but much more often to other circumstances which are generally regarded as productive of erysipelas. Cases are reported in which, among several children vaccinated with the same lymph, only one was attacked by erysipelas.

Vaccinia and variola, although their identity has been proven

quite conclusively by experimental methods, may run their course alongside one another. Thus, a child who exhibits vaccination pocks may be attacked by small-pox whose virus has been communicated prior to the vaccination. Furthermore, vaccination may take in an individual suffering from small-pox.

If the vaccination is not successful it should be repeated in two to three months. Some persons are immune only at times and to a certain lymph, but few are absolutely immune. Whether this is true in regard to small-pox cannot be determined, as a matter of course, because inoculation with small-pox is not permissible.

LECTURE XVI.

II. ACUTE, NON-CONTAGIOUS, INFLAMMATORY DERMATOSES.

THE ANATOMICAL CHANGES IN THE ERYTHEMATA IDENTICAL BUT VARYING IN DEGREE—ERYTHEMA MULTIFORME AND HERPES IRIS ET CIRCINATUS—ERYTHEMA NODOSUM—PURPURA RHEUMATICA.

We now come to the consideration of a large class of skin diseases which are characterized by the phenomena of inflammation. They also run an acute, in part typically limited course, but they are not contagious.

The numerous dermatoses in this category fall naturally into three principal groups.

In the first group the inflammatory process begins with a peculiar vaso-motor disturbance of vessel tonus and fulness. These present the angioneurotic type in one stage (the beginning), and they might be called typical angioneuroses, but with the proviso that this does not constitute their entire character. To this group belong (*a*) *the typical forms of erythema* and (*b*) *urticaria*.

The second group is characterized by the typical development of vesicles (*phlyctænoses*), due to an excess of exudation within the papillary layer and the rete (*herpes*).

In the third group the symptoms of inflammation are most pronounced (*dermatitis* proper).

I. TYPICAL ANGIONEUROSES.

The sharply defined symptoms of this group of diseases warrant the inference that they are due to disorders of vascular innervation. Since the investigations of Eulenberg and Landois they have been known as angioneuroses.

The chief clinical symptom in the integument is an acutely manifested condition of instability in the tonus of the finest vessels and capillaries. Within a circumscribed region the vessels first undergo irritation and contract as a result of stimulation of the vaso-constrictors (or of the living matter of the walls), upon which appears an active hyperæmia. This is followed by paralysis of the

vaso-constrictors or the walls of the vessels, or by irritation of the vaso-dilators, giving rise to passive or relaxation hyperæmia with its well-known characteristics.

In the first stage of active hyperæmia the bright-red color disappears under the pressure of the finger. In the second stage the enlargement of the lumen of the vessels causes a slowing of the current of blood. When the blood remains in the skin for a longer period than usual, more oxygen is withdrawn from it by the tissue elements and more carbonic acid is carried to it, so that its color becomes darker. In this stage the hyperæmia is dark to bluish red (the color of asphyxia), and the dark shade is intensified by the coincident escape of the blood-coloring matter into the tissues.

The disturbance of vascular tonus may not only produce hyperæmia, but, as the result of further relaxation of the walls of the vessels. it may cause fluid exudation, producing vesicles and wheals. Indeed, it may even result in hæmorrhages.

We will now consider in detail the manner in which such symptoms are produced.

It is well known that the entire spinal cord contains vaso-motor centres. Any one of the latter may send out a stimulus, which appears at the periphery as an angioneurotic disorder, and it is to be expected *a priori* that this disorder will appear in various and symmetrical portions of the body. The circulation of a virus in the blood may act upon the centres and may thus produce angioneurotic erythema.

Such poisons include the blood of fever (pyrogenic substance), the virus of small-pox, measles, scarlatina, typhoid fever, drugs (quinine, opium, etc.). Hence roseola febrilis and typhosa erythema variolosum, the acute exanthemata in general, the drug eruptions (page 95), are angioneuroses, and are all due to direct irritation of the vaso-motor centres. This does not exclude the assumption that some of the cutaneous symptoms due to the same poisons result from direct irritation of the skin by local deposit or excretion of poisonous matters. For example, erythema variolosum is to be interpreted as an angioneurosis, an effect of central vaso-motor irritation, while the variola pustule and roseola syphilitica are due to the local excretion of the variola or syphilitic virus.

The vaso-motor centres may also be irritated in an indirect, reflex manner, and the same symptoms will then appear at the periphery. The stimulus may start from the cerebral cortex—for example, in the flushing of shame, anger, and confusion. It may also start from the peripheral terminations of the sensory nerves. A part of the integument is irritated, the stimulus is conveyed to the spinal cord, there the vaso-motor centres are excited, and the irritation is then projected externally, usually to some other part of the skin (reflex

vascular irritation). This process plays a great part in diseases of the skin. For example, a caterpillar falls upon one hand, and a burning, itching wheal forms as the result of direct irritation of the peripheral vaso-motor and sensory nerves. But in a few seconds other wheals develop in remote parts of the body, and these continue to increase in numbers, because the mechanical irritation (scratching) furnishes the source of other reflex irritations. A similar condition obtains in eczema, psoriasis, pemphigus, lichen ruber, etc.

It must not be forgotten, however, that the blood vessels of the papillary layer may be thrown into a similar condition of unstable equilibrium (as shown by active, later by passive fluxion) by mechanical, chemical, and dynamic agencies which affect the skin directly from the outside. We make a streak upon the skin with the finger nail. A corresponding white line (contraction and anæmia of the capillaries affected by the pressure of the nail) develops forthwith, in a few seconds becomes bright red (irritative fluxion), often raised in wheals (exudation), in a few minutes grows dark, bluish red (relaxation hyperæmia), and remains in this condition for a variable length of time. These symptoms usually extend over a vascular territory which is wider than the line made by the stroke of the nail. These in turn are changes of an angioneurotic character.

All these forms, which are identical as regards the local process and the physiology, but vary as regards course, history, and causation, have been described by some writers as a single clinical group, but this appears to us to be a forced and unnatural classification.

No argument is necessary to prove that there is no clinical relationship between the prodromal eruption of variola and an erythema solare, between an urticaria starting from a uterine or gastric affection and the blush of shame, between acne rosacea and purpura rheumatica, although they all coincide very closely in the physiologico-anatomical conditions.

Hence we include among the angioneuroses only those conditions in which the general clinical character, the causes, and the course of the process exhibit a great similarity.

A. THE TYPICAL FORMS OF ERYTHEMA.

The anatomical character of erythematous processes with a typical course consists in the development of red patches, due to hyperæmia and indicative of beginning inflammation, with moderate serous imbibition (exudation) of the uppermost layers of the skin.

If there is a slight increase of the hyperæmia and serous swelling, papules, nodules, or wheals will result. Indeed, the amount of exuded serum may be so great that the epidermis is lifted into vesicles. According to their supposed or demonstrated causation, the

typical forms of erythema may be subdivided into α, idiopathic, and β, toxic erythemata.

α. Idiopathic (Essential, Auspitz) Erythema.

This is again subdivided into 1, erythema exsudativum multiforme ; 2, erythema nodosum ; 3, purpura rheumatica.

I. Erythema exsudativum multiforme (*Hebra*).
Erythema polymorphe (author).

As its name indicates, this process presents a manifold appearance, while the exudation distinguishes it from the congestive erythema (page 92) due to simple hyperæmia.

Erythema multiforme begins symmetrically upon the dorsum of both hands and feet, and upon the adjacent parts of the forearms and legs, with the development of sharply defined, disseminated patches (erythema læve), as large as a pin's head and rapidly increasing to the size of a lentil. The patches are bright red, become pale under the pressure of the finger ; they are flat or moderately raised, and are either normal to the touch or are firm and œdematous.

These patches enlarge within a few hours by peripheral growth, while new efflorescences appear between them. At the end of a few hours the oldest points of eruption (*i.e.*, the centres of the larger patches) appear depressed and cyanotic, while the more recent, peripheral parts form a bright-red border.

The patches soon attain the size of a kreutzer or dollar, and these finally coalesce into still larger patches, so that on the second or third day the dorsum of the hands is cyanotic, diffuse, bluish red in color, and feels cold. On pressure the bluish redness disappears and yellowish-brown pigmentation is left over. Patches which pass through the same changes as regards size and color next appear upon the arms, the throat and neck, the chest, the knees and thighs, especially on their extensor surface.

The rapid occurrence of cyanotic injection after the bright-red hyperæmia indicates stasis in the venous capillaries. Hence it is easily understood that, in addition to occasional œdema of the deeper layers of the skin and the subcutaneous cellular tissue (for example, in the eyelids), the coloring matter of the blood soon exudes and even actual hæmorrhages may take place. During the next few days such patches pass from blue to yellow, green, yellow, and brown, the changes occurring from the centre toward the periphery.

In the meantime the originally affected dark-colored patches on the dorsum of the hands and feet become the site of new and scanty primary patches and papules. These have a brick-red to cinnabar color, probably from the admixture with the oxidation color of the hæmatin which has already been exuded.

Erythema annulare results when the patches fade rapidly in the centre while the red border extends centrifugally. The coalescence of several circles, which are effaced at the points of contact, produces sinuous lines and all sorts of delicate figures (erythema gyratum, figuratum).

In *erythema iris* a red patch again forms in the centre of the growing lesion.

The term *erythema papulatum* is employed when the original patch has developed into a somewhat more elevated, firmer papule as a result of increase of the exudative process. The term *erythema urticatum* or *lichen urticatus* is employed when the size corresponds to that of an urticaria wheal. In the latter cases the papules usually itch violently, are soon scratched and covered with a black crust of blood. As the process of development is the same as in the simple patches, this results in characteristic figures. The patches vary from the size of a lentil to that of a penny ; the centre contains a blood crust, then follows a bluish-red and depressed area enclosed in a red, elevated border.

A still further increase of exudation results in the formation of vesicles. As a rule these are very firm and contain a large amount of serous fluid. As a matter of course, they develop according to the type just described. In a few hours the centre of the nodule sinks in and becomes bluish red in color as the fluid is absorbed and the cyanosis of the base becomes visible. At the periphery a red, elevated, sharply defined border continues to form, and upon it is situated a ring of vesicles (*herpes circinatus*). We find occasionally in the centre an old or newly developed vesicle ; at the periphery a ring of vesicles ; occasionally a central ring adjoining the latter (*herpes iris*). Finally, the epidermis in one or more places, in the centre or at the periphery, may be lifted up into a large vesicle (*erythema bullosum*). It is evident that all these various forms constitute one and the same process.

I will here give an illustration (Fig. 21) of a section of a papule from a case of erythema papulatum, the lesions of which are undergoing conversion into vesicles. At the base of two papillæ are seen extravasated red blood globules (hæmorrhage). Above the middle papilla the rete Malpighii is partly swollen and loosened, and its upper layers are separated into the septa of a network. This is filled with serum and a few exudate corpuscles and wandering cells—*i.e.*, it is a vesicle with a thick epidermic covering.

The individual patches terminate in brown pigmentation without desquamation. It is only when there has been greater superficial exudation, as in the vesicular forms, that the desiccation of such efflorescences gives rise to corresponding, often considerable, formation of crusts and desquamation.

As a rule there is no fever and notable subjective symptoms are wanting. Sometimes there is slight burning in the papular form, violent itching in lichen urticatus, and occasionally notable pains in the phalangeal joints, wrists, and ankles.

Erythema multiforme runs a typical course in two to four weeks, at the most six weeks. It lasts longer when the eruption, as in rarer cases, gradually attacks the entire trunk and new crops appear upon places already attacked. Each new patch requires eight to ten days for its complete disappearance. As a general thing an erythema combined with extensive serous exudation (erythema urticatum, herpeticum) will run a longer course than erythema læve.

In exceptional cases the erythema lasts months and months, but

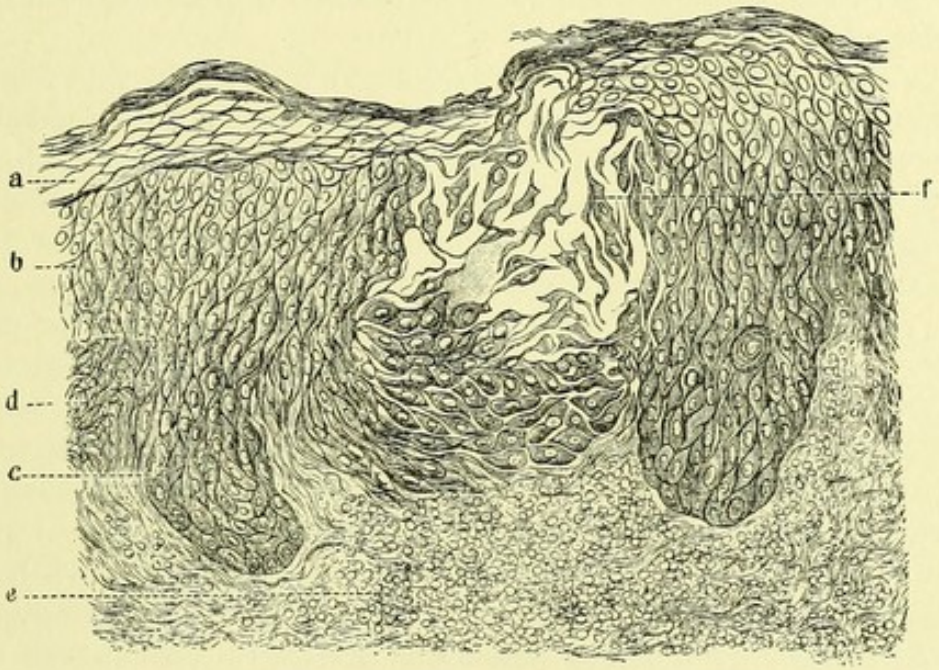

FIG. 21.—SECTION THROUGH A PAPULE OF ERYTHEMA PAPULO-VESICULOSUM.

a, horny layer; *b*, rete; *c*, rete pegs; *d*, papilla whose elements are separated by serous exudation; *e*, hæmorrhage into subepidermal connective tissue; *f*, beginning vesicle, with contents of serum and exudation cells.

only on account of the repetition of relapses. Each eruption then runs an acute course. I have seen several such cases in which, for one, two, or more years, there were constant relapses of erythema, herpes iris and circinatus. Under such conditions the central pigmentation of the patches is so intense and their circumvallation is usually so firm that they may be mistaken for syphilitic papules which are undergoing involution in the centre.

There are also notable deviations from the type described in regard to localization, the intensity of the concomitant symptoms, and the complications. Thus, the dorsal surfaces of the hands and

feet may not be affected or only at a late period, and the eruption may appear chiefly on the face and trunk, or the lower limbs may escape entirely ; it may be confined to a small region of the skin, or it may be universal, or the palms of the hands and the soles of the feet may be chiefly involved.

The cutaneous symptoms may also be attended by an entirely analogous eruption of reddened patches and vesicles upon the buccal and pharyngeal mucous membrane. The overlying epithelium rapidly grows gray and cloudy and is detached, so that the patches appear raw and painful. In rarer cases they also appear on the epiglottis, the larynx, and also on the vulvo-vaginal mucous membrane and in the urethra. In the latter event there is a profuse secretion of mucus. In infants such affections of the mouth and pharynx may give rise to dangerous complications (interference with the ingestion of food, suffocation).

As regards the intensity of the symptoms, we may find firm infiltration extending into the subcutaneous tissues and attended with hæmorrhage. There may be severe gastric symptoms, chill, high fever, intense inflammation, ulceration, hæmorrhagic disintegration and gangrene of the pharyngeal mucous membrane with a fatal termination, renal hæmorrhages, severe affections of the joints, symptoms of mental depression (Lewin). In one of my cases the appearance of the erythema was always preceded (every two weeks) by hæmorrhages from the kidneys.

Among the complications and sequelæ we may mention endocarditis, pericarditis, meningitis, tuberculosis, valvular disease of the heart, pneumonia, and death has occurred in a considerable number of such cases (in ten cases among seventy collated by Lewin). It is evident that in all these cases the erythema is merely symptomatic, like so many roseolæ, and hence the fatal termination may not be ascribed to the erythema. For this reason I do not favor the classification into mild and malignant forms of erythema multiforme (Lewin). It is sufficient to recognize the fact that in some cases the general disease, of which the erythema is a reflex effect or symptom, may run an unfavorable course.

Apart from such rare cases the *prognosis* is favorable and the disease runs a benign course.

The *diagnosis* is quite easy. The appearance of disseminated patches, of which the larger ones exhibit a central bluish-red depression, is so peculiar that it cannot be mistaken for any other disease. Those forms which are associated with papules and vesicles are also very characteristic.

The patches are found to develop from individual centres, the process begins symmetrically on the dorsal surfaces of the hands and feet (the patches there appear to be older, as shown by the yellow

color after employing pressure with the finger), and there is a rapid change of symptoms (within a few hours). All these data render a mistake impossible.

From herpes tonsurans and psoriasis, which also appear in circular forms, erythema is easily distinguished by the absence of scaling; from syphilis annularis by the absence of the infiltration of the cutis, which does not disappear under the pressure of the finger, in addition to the other positive signs.

We are entirely in the dark with regard to the *cause* of this remarkable process. There is no doubt, however, that we have to deal with a capillary hyperæmia and subsequent paresis of the finest papillary vessels. Since Landois' article it has been regarded as an angioneurosis, which merely signifies that the process begins with a sudden change in the innervation of certain vascular districts. This physiological category, however, also includes other processes which have no clinical resemblance to erythema, so that the term angioneurosis does not correspond clinically to erythema multiforme. We merely know that the disease is more frequent in certain seasons (March, April, October, November), that some individuals are attacked at about the same time for two or three years, and that young people exhibit a greater predisposition than adults and old people.

In view of the fact that the disease is usually so frequent at certain seasons as to form a little epidemic, that it often returns every year, and that it is not infrequently complicated with articular pains (this is especially true of erythema nodosum and purpura rheumatica), a relationship between articular rheumatism and erythema multiforme is not improbable, and the cases which occur so often in certain seasons might be regarded as miasmatic—infectious erythemata. But as the etiology of articular rheumatism is itself obscure, this assumption does not explain anything. The hypothesis of a bacterial origin is no less hypothetical.

The mode of life, general constitution, food and drink appear to exert no effect on the development of the disease.

As Hebra showed, local causes do not produce erythema multiforme. My observations on the action of mercury have shown, however, that a local irritant occasionally produces an erythema of this kind. Lewin states that the erythema developed in a few cases from irritation of the urethra by erosions, and I am acquainted with one patient in whom an erythema annulare appears upon the backs of both hands whenever he experiences an exacerbation of his chronic clap. Such cases have the same significance as those in which the erythema is a reflex symptom starting from the female sexual sphere or from the kidneys.

Still, such isolated cases are insufficient to explain the etiology

and particularly the typical localization on the hands and feet. It is possible that the latter fact depends upon other conditions, as the result of which capillary stases are most apt to develop in the peripheral parts of the body.

I may here add that in certain women who suffer from amenorrhœa (infantile uterus), dysmenorrhœa, chlorosis, or sterility, periodical erythemata may develop, for a number of years in succession, upon the hands, and particularly the forehead, in the shape of erythema urticatum et iris. In these cases the patches on the forehead sometimes run a very slow course, have a very firm border and a dark-brown depressed centre, so that the diagnosis of corona veneris is made by inexperienced physicians.

Lewin was the first to report several cases of erythema in which blowing cardiac murmurs simulated valvular disease. They are probably a symptom of chlorosis, not of erythema, unless fever and local auscultatory signs indicate endocarditis.

Urticaria and roseola may also occur in circles and may thus simulate the analogous forms of erythema multiforme. As the former may be undoubtedly produced by certain foods and drugs or by local injuries—for example, by caterpillars—it is easily understood that these causes have been supposed to be productive of erythema, when the latter diagnosis has been erroneously made. This includes cases like Mader's, in which the cutaneous eruption was repeatedly attended with severe colic (reflex from the intestinal mucous membrane), or like that of Arnold Pick, in which the eruption occurred repeatedly at menstruation. Hebra attributes an entire series of analogous forms to functional disorders of the uterus.

If we combine all the cases which warrant a conclusion in regard to the causation of erythema multiforme (erythema nodosum and purpura rheumatica), we think ourselves justified in claiming that :

1. The typical cases, which occur annually in large numbers at certain seasons of the year (like epidemic zoster), lead us to infer a miasmatic infectious, bacterial origin.

2. The sporadic cases are generally a reflex effect (pure angioneuroses) of anomalous conditions of the internal organs (chiefly of the female sexual organs—amenorrhœa, dysmenorrhœa, uterine displacements, etc.).

3. Some may be due to autoinfection with toxic substances which have entered the blood as the result of internal diseases (tuberculosis, nephritis) which are associated with inflammation, suppuration, and malassimilation.

4. Others may be due to a primary (constitutional) instability of the vaso-motor centres.

As a matter of fact all these processes—erythema, roseola, herpes, and urticaria—have so much in common and exhibit so many transi-

tional forms that it is sometimes difficult to maintain a sharp clinical differentiation.

II. Erythema Nodosum.

This disease, also known as *dermatitis contusiformis* or *urticaria tuberosa,* is allied to erythema multiforme. It appears in the shape of nodules and tumor-like lumps, from the size of a hazelnut to that of a hickorynut, of firm consistence, usually on both legs and the dorsum of both feet, less often upon the forearms, thighs, and nates, very rarely also on other parts of the body.

The nodules of erythema nodosum project moderately above the level of the integument, or lie at its level and exhibit a rose-red periphery with a bluish-red centre. These patches are distinctly felt by the finger as firm nodules, which are painful of themselves and are also extremely tender on pressure. They are usually isolated, but sometimes a larger number (fifteen to twenty) are found on each limb. Their development is extremely acute, and is sometimes attended by febrile symptoms, general malaise, gastric symptoms, pain and swelling of the joints, especially of the affected limb. The individual nodules last several (eight to fourteen) days. The fresh nodules remain unchanged apparently for two to three days, then the bright redness of the overlying skin changes to bluish red and yellowish green, at first in the centre, then extending to the periphery. At the same time the mass of infiltration diminishes until, after a lapse of one and a half to two weeks, every trace has disappeared with the exception of some brown pigmentation. A moderate hæmorrhage sometimes takes place into the infiltrated part of the skin, and the changes of color incident to every hæmorrhage then occupy a somewhat longer time. Some nodules feel soft, elastic, so that they appear to be abscesses. In very exceptional cases suppuration of a nodule does occur, according to the statements of some writers (Hardy).

During the first two weeks new nodules appear, as a rule, and pass through the changes described above, so that the disease lasts three to six weeks, and in exceptional cases even months. The febrile symptoms, which are rarely absent in severe cases, run parallel with the eruption ; they exhibit a quotidian or quartan type, and subside with the beginning of general involution. Apart from the joint affections, the concomitant symptoms are dyspepsia, more rarely colic and diarrhœa, painful nodules in the tongue and the mucous membrane of the mouth and pharynx. The latter often terminate in ulceration and necrosis (Pospelow, Rasumow, etc.).

Erythema nodosum is much more distressing than the other forms of erythema. Indeed, if it runs a febrile course of several months the patients may be very much reduced, and life may even be imperilled by dangerous complications. Nevertheless these ery-

themata are analogous processes. The nodules of erythema nodosum may be mixed with the patches of erythema multiforme, or pure erythema nodosum may appear on the lower limbs and erythema multiforme upon the upper limbs, face, and trunk. Both processes exhibit the same localization of the eruption, run an acute typical course at the same season of the year and under the same circumstances. I am therefore unable to maintain, as some do, that erythema nodosum is a process *sui generis*. In my opinion it should be clinically distinguished as a special type, but it is essentially identical with erythema multiforme and peliosis rheumatica.

This opinion is supported by the fact that all the *causal* factors, demonstrable and hypothetical, which we have mentioned with regard to erythema multiforme, also obtain with regard to erythema nodosum. It is true, however, that, in accordance with the greater intensity of the local process in some cases, in erythema nodosum there is also more marked implication of the general organism, such as chronic and acute tuberculosis, nephritis, etc. In the majority of cases, especially those of the annual type, the causation is as obscure as in erythema multiforme and purpura rheumatica. Erythema nodosum is most frequent in young people, infants and children, and during the spring and autumn.

Anatomically the nodules consist mainly of serous infiltration of all layers of the tissues as far as the subcutaneous connective tissue, together with capillary stasis (at first arterial, later venous), increased fulness of the lymph spaces and capillaries, moderate accumulation of cells in the tissues, and localized accumulations of emigrated red blood globules. With regard to the suddenness of its development, its complete involution, and the anatomical findings, the erythema nodule simply forms a more fully developed and stable urticaria wheal.

The *prognosis* is good, but it should not be forgotten that the disease may interfere with daily work and may even cause a stay in bed. In little children the disease is more serious, although, as a rule, it runs a favorable course. In some cases, however, the constant impairment of nutrition, the frequent fever and accidental complications, such as renal hæmorrhage, may give rise to a fatal termination. It must be remembered with regard to all these processes that they are the more serious the greater their tendency to the development of hæmorrhages.

If solitary nodules develop upon the legs or some other part of the body—for example, the arm or eyelids—are enormously swollen and show the well-known colors due to a hæmorrhage, the condition is apt to be regarded as traumatic (subcutaneous injections of morphine-eaters, blows, contusions, etc.), and hence the origin of the term erythema contusiforme. As a matter of course a wrong dia-

gnosis will not be made in cases of typical localization upon the legs when the nodules vary in age, and the recent ones are rosy red at the periphery and bluish red in the centre.

Non-ulcerated gummata of the legs may be mistaken for erythema nodosum. The former are always sharply defined; the latter are gradually lost at the periphery.

III. Purpura Rheumatica.

In this condition (also called *peliosis rheumatica*) bluish-red patches, which change to brown (hæmorrhages) and do not disappear on pressure, appear in a typical localization. They develop within a period of three to six weeks, and are usually accompanied by a more pronounced inflammatory and painful affection of the joints, especially of the knees, hands, and feet.

Strictly speaking, this process belongs to the category of hæmorrhages, but your comprehension of the peculiar character of the exudative processes already described will be increased when you learn that the extravasations in question first occur typically on the legs and forearms, and that the entire process presents the same course, complications, etiology, and prognosis as the first-mentioned forms. As a rule purpura rheumatica runs an acute, cyclical course of six to eight to twelve weeks, terminates in recovery, occurs in little epidemics at certain seasons of the year, and, apart from the rare sporadic cases, at the same time as the other two forms.

In not a few cases we find all the morbid forms in this group occurring at one time in the same individual. For example, erythema annulare, læve, diffusum, iris, upon the upper limbs; herpes iris and circinatus on the backs of the hands and on the face; urticaria papulosa and erythema nodosum, in addition to herpes and erythema bullosum iris with hæmorrhagic contents, on the lower limbs; and scattered superficial hæmorrhages, the size of a lentil or larger, complicated with swelling of the joints. There is no doubt, therefore, of the physiological relationship and clinical unity of all these forms.

The mucous membrane of the mouth and pharynx is often the site of the same affection. Punctate and larger hæmorrhages are observed, the remainder of the mucous membrane being intact.

The hæmorrhages are repeated by fits and starts during the course of the disease, and are accompanied by malaise, pains in the joints, fever, etc.

The escape of blood by diapedesis or through solutions of continuity indicates more profound relaxation of the vascular walls and a more serious ailment, and the complications on the part of other organs are often found to be more serious than in erythema multiforme and nodosum. Thus there may be paroxysmal hæmaturia

for a couple of hours every second or third day. Acute endocarditis, valvulitis with temporary or permanent insufficiency, are also observed more frequently. In two of my cases aortic insufficiency gradually developed without fever (reported by A. Schwarz, 1883). We have also observed danger to life, and in one case a fatal termination, from hæmorrhagic suffusion and destruction of the palatal mucous membrane.

Apart from these exceptional cases, the course of purpura rheumatica is entirely analogous to that of erythema multiforme and nodosum, and warrants a favorable prognosis. Children are put in greater danger on account of the successive losses of blood. In some patients the disease occurs in relapses for years, and is then associated, in my experience, with chronic nephritis, which may or may not be complicated with cardiac disease.

With regard to the *treatment* of all these forms of disease, we are unable to antagonize the nervous influence which gives rise to the production of macules, papules, wheals, vesicles, and hæmorrhage with the typical tendency to peripheral progression. We are unable to prevent the first eruption or subsequent relapses, or to accelerate their involution, so that treatment is really unnecessary.

Cases of ordinary erythema polymorphe may be left to themselves. In cases of pruritus which is annoying, in lichen urticatus, erythema urticatum and papulatum, good effects are obtained from applications of alcohol or brandy, with or without the addition of 1.0 acid. carbol. or acid. salicyl. to 200 grammes fluid, and then dusting with starch, or the use of cold compresses. If there is pain or considerable swelling of the joint and the febrile symptoms continue, it is better to keep the patient in bed and to administer salicylate of soda, antipyrin, quinine, ergotin, iron, etc. Relief is also obtained from compresses of ice, cold water, lead wash, or liquor Burowii. General symptoms, such as fever, gastric distress, etc., must be treated by suitable internal medication, such as phosphoric acid, antipyretics, dietetic measures, etc.

When frequent relapses occur the individual conditions must be carefully considered and corresponding therapeutic measures adopted. In some of these cases good results will be obtained from mineral waters (Carlsbad) and nerve tonics, such as quinine, iron, ergotin, hydrotherapy, electricity.

β. Toxic Erythemata.

These forms of erythema are in part morphologically identical with those already described, but differ clinically in view of their atypical character, their localization and course, and also because they are due to infectious or toxic causes.

The first to be mentioned is *roseola*, which has already been discussed (page 94). Its macules may be converted into papules by increase of the underlying hyperæmia and exudation, and would then be included among the exudative erythemata already described, such as roseola autumnalis, vernalis (Willan), or roseola cholerica, variolosa, vaccinica. In the latter, as well as in roseola typhosa, it is probably the specific disease germs which affect the vaso-motor nerves in such a way that the eruptions appear upon the skin, and hence, from an etiological standpoint, they may be called toxic.

The prodromal eruption of small-pox has already been described (page 178), also roseola cholerica (page 94). An eruption in the shape of macules and papules also occurs in typhus and typhoid fever. A peculiar form is observed in certain epidemics of typhoid fever. It appears in the shape of roseolæ or erythema papules, from the size of a lentil to that of a finger nail, and situated mainly on the trunk, abdomen, and inner surfaces of the limbs. It is more stationary than typical erythema multiforme. Dietel's typhoid eruption occurs in the shape of elongated, shining papules (resembling wheat grains) upon the chest and abdomen.

The eruption of typhus fever is more extensive, polymorphous and changeable, often petechial, and, like the typical erythemata, is situated particularly on the extensor surfaces of the limbs in addition to the trunk. Similar eruptions have been reported during the course of influenza and also in Weil's disease.

Syphilitic roseola also appears in the shape of patches, whose shape and size continue unchanged until they disappear without desquamation. Their unchanged shape and histological character distinguish them from the roseolæ of the erythemata. Anatomically they form a forerunner of the syphilitic papule and are the product of direct irritation by the locally deposited syphilitic virus. Hence they are not angioneurotic.

In view of their general etiological relations and their physiological significance, the majority of the so-called drug eruptions must be included among the toxic erythemata. Formerly it was known that erythema, roseola, and urticaria may develop after the ingestion of a few drugs—for example, copaiba; but in recent years it has been learned that such action on the vaso-motor nerves is produced by numerous drugs, whether administered by the mouth, enema, subcutaneous injection, inhalation, or direct contact with the skin. These include quinine, opium, morphine, chloral hydrate, chloroform, turpentine, digitalis, antipyrin, phenacetin, rhubarb, mercury, tar, iodine, bromine, etc. Apart from the rarer papular, vesicular, nodular, and pustular forms which are characteristic of certain drugs, the drug eruptions usually consist of an erythema, a scarlatina-like or measles-like eruption, an erythema papulatum et urticatum. It

rarely exhibits the typical localization of erythema multiforme, and passes through its various stages much more rapidly than the latter. The quinine eruption generally appears as a dark-scarlet redness, and sometimes extends over the entire body. In one woman I have seen this, on several occasions, as a diffuse, dark scarlet redness, which in the course of a few hours changed to a sepia-brown pigmentation on the affected parts of the skin; in another case there was universal purple redness with fever, followed by general desquamation. In other individuals the smallest dose of quinine, antipyrin, phenacetin is followed by an eruption of disc-shaped patches, of a bright-red, later bluish-red to brownish-red color, and varying from the size of a dollar to that of the palm of the hand; not infrequently there is an acute development of vesicles, sometimes upon definite parts of the skin, upon the inner surface of one thigh, the left forearm, one side of the buttocks, on the mucous membrane of the female genitalia, the arms, the mouth.

As has been shown by Behrend, Unna, and others, the physiological process in toxic and drug exanthemata is not always identical. The toxic substances which enter the blood from the intestinal tract, etc., have a reflex or direct effect upon the vaso-motor centres and thus give rise to the multiform toxic erythemata. In other cases the toxic substances, being excreted through the cutaneous capillaries, have a direct irritant and paralytic action upon the peripheral vessels and adjacent tissues, so that the erythema and the more intense inflammations (iodine and bromine acne) develop only at these points. When local applications are made (naphthol, mercury) both modes of action may result, so that, in addition to the direct inflammation, there is also a typical progressive erythema (local intoxication), and later a more remote and general erythema due to the absorption of the remedy, from general poisoning of the blood and irritation of the vaso-motor centres. Behrend expressed this difference in the action of drugs by dividing the toxic exanthemata into the specific, excretory, and dynamic forms.

Finally, the group of toxic erythemata also includes those which form an initial or part symptom of pellagra and acrodynia.

Pellagra.

Pellagra (*risipola lombarda, mal rosso, mal del sole, Lombardy leprosy*) is an endemic disease, found chiefly among the poorer classes inhabiting the Lombardy plains, Venice, and Southern France. In the last few years a large number of cases have also been reported from Roumania (Scheiber) and from Spain. Despite the extensive literature of the subject which has accumulated since the middle of the last century, no uniform and convincing description of the symp-

toms, causes, and significance of the disease has been furnished, and doubts have arisen with regard to the real existence of the disease. In 1876 Winternitz, as the result of personal observations and studies, came to the conclusion that there is no nosological entity corresponding to the term pellagra, and that this diagnosis has been made improperly in all sorts of cases.

Nevertheless there are a very large number of careful observers who have studied this disease and characterize it as a specific process. Hebra states that he has observed a large number of pellagra patients and that the symptomatology exhibits the greatest resemblance to other toxæmias produced by spoiled vegetables. I have also observed a few cases which could not possibly be placed in any other group but that of the pellagrous affections.

The symptoms of pellagra have been described in various ways. I believe that this is owing to the fact that the disease does really occur in various forms, may run a more acute or an extremely slow course, and may exhibit very few or very many symptoms.

There are generally several stages of the malady. The first stage is characterized by erythema. The skin has a dark brownish-red color upon the backs of the hands, the face, neck, and chest, so far as they are uncovered and exposed to the rays of the sun. This occurs during the spring and summer, disappears with slight desquamation in the autumn and winter, and returns for several years in warmer weather. After frequent relapses the epidermis of the erythematous places becomes dark olive-brown in color and peels in thick callosities. Muscular weakness and mental depression are superadded. The disease may then undergo recovery, or it may enter the second stage, which is characterized by increased muscular weakness, formication, constant feeling of coldness, and further changes in the skin. The entire integument, especially in the face and on the hands and feet, then has a bluish-red or bronze-brown color, is shining and extremely tender; the epidermis is thin and looks like silk. There is numbness or tingling in the fingers, which are kept flexed. Contact with the floor is painful to the feet. Disorders of sensation, spasms, diarrhœa, delirium, stupor, melancholia, and dementia gradually develop. The patients die of marasmus, colliquative diarrhœa, or acute and chronic affections of the lungs, kidneys, and heart.

Apart from the anatomical changes which are due to the complications just mentioned, post-mortem examinations (Labus, Scheiber, etc.) have shown pachymeningitis, cerebral and spinal sclerosis, and an anæmic or atrophic condition of the viscera, such as is found after chronic inanition.

Among the manifold assumed causes of pellagra (bad surroundings, telluric and climatic conditions, the sun's rays, heredity, etc.), an exclusive diet of maize is especially emphasized, and attention

has been called to the fact that the disease is found only among the poor of certain regions who live on such food. Since the predominance of the fungus theory in the etiology of disease, pellegra has been attributed to a fungus, the *sporisorium maidis*, especially as the disease is supposed to follow the ingestion of spoiled maize. Lombroso produced the symptoms of pellagra experimentally (1860) by the administration of a tincture derived from spoiled maize. He regards the real cause of the disease as some special substance which develops during the spoiling of the grain, and excludes the maize fungus and the ordinary mould fungi as causes.

In recent years greater attention has been paid to this subject by various governments (Roumania, Hungary, Italy, Spain, Austria). Neusser, who studied the disease in Friaul (1887) at the instance of the Austrian government, states that the symptoms develop in the following chronological order: 1, functional psychical affections; 2, amyotropic lateral sclerosis; 3, tetany; 4, meningitis; 5, chronic gastro-enteritis; 6, chronic degeneration of the organs with secondary atrophy; 7, essential cachexia or anæmia; 8, Addison's disease; 9, the pure dermatosis; 10, pellagra sine pellagra, in which the cutaneous symptoms occur only in the latest stage. In regard to the etiology Neusser occupies an intermediate position. He does not ascribe it to the bacteridium maidis, because Paltauf obtained negative results with pure cultures and inoculations. He believes that a poisonous principle develops in spoiled maize under the influence of the bacteridium maidis, and that this gives rise to pellagra only after the system has been weakened by other causes (insolation, gastric disorders).

Scheiber, I, and others have also seen cases of pellagra in individuals who had never eaten maize but had lived like well-to-do city people. As some of these patients had never worked in the sun, and as one of my cases came from Bohemia, where pellagra is not endemic, it is evident that there are many points in regard to the disease which are still obscure.

Therapeutic results can only be expected in the early stages of the disease from improvement in the surroundings, good food, cold water cures, iron, etc. Some cases recover spontaneously. Those forms which are more fully developed, and particularly those which are complicated with insanity, always terminate fatally.

Acrodynia.

ACRODYNIA, or endemic erythema, exhibits many analogies with pellagra. It was epidemic in Paris in 1828 according to Alibert, and in 1829 and 1830 according to Hirsch. The hands and feet of the patients were the site of an erythema with subsequent desqua-

mation, or there was a development of vesicles and detachment of the thickened epidermis in callosities, as in pellagra. The integument of the chest and abdomen appeared almost black. Tingling and numbness of the fingers and toes, violent pains in the same parts, vomiting, diarrhœa, and ischuria developed later, and often ended in death. The disease was generally attributed (Chomel, Recamier, etc.) to the use of spoiled grains and was regarded as analogous to pellagra.

LECTURE XVII.

B. URTICARIA, NETTLE-RASH.

FORMS AND SIGNIFICANCE OF URTICARIA, IDIOPATHIC AND SYMPTOMATIC, ACUTE AND CHRONIC.

URTICARIA (*cnidosis, nettle-rash*) is a disease which manifests itself, as the name suggests, in the production of wheals. These are round or irregularly shaped efflorescences, the size of a finger nail or larger, developing suddenly, rosy red or white in color, and surrounded by a red zone. They itch and burn intensely, and completely disappear within a few minutes, without desquamation and usually without leaving a trace.

The individual wheal spreads rapidly over the surface, its red border advancing peripherally until it attains the size of a dollar or more. The top has a white, shining appearance (*urticaria porcellanea*), and is either flat or somewhat depressed in the centre. The wheal disappears without a trace or leaves slight brown pigmentation. The involution sometimes begins in the centre, while the periphery spreads. This causes wheal rings (*urticaria annularis*), and, by the union of several of these rings, gyri (*urticaria gyrata s. figurata*). Sometimes there are several concentric and eccentric circles, which rapidly change their shape. Inasmuch as the skin of the patients is sensitive everywhere, every contact of the finger nail with the skin will produce urticaria. We are thus enabled to produce lines, or even drawings and letters, upon the integument by outlining them with the finger (*urticaria factitia*). At first a white streak is produced ; this at once becomes red, then white and shining, and projects like a wheal. This persists for a variable length of time, and may even spread.

This phenomenon has long been known, so that it was unnecessary to attach so much importance to Dujardin-Beaumetz's " femme autographique," upon whom any figures and letters could be produced by the stroke of the finger. This does not take place in hysterical women alone. There are individuals in whom the local pressure of the shoe or the corset will produce wheals and even vesicles (*urticaria factitia bullosa*), and Campana has published the

photograph of an old, non-hysterical man upon whose back a name and date appear in urticaria writing.

In my opinion the cases described by Goldscheider, Valentin, and Köbner as hereditary predisposition to the formation of vesicles (*epidermolysis bullosa hereditaria*, Köbner), and in which the vesicles were due to mechanical pressure, belong to this category.

In some places the wheals are converted into vesicles by an accumulation of serum in the epidermis (*urticaria vesiculosa et bullosa*), and after their rupture crusts form ; or we find merely papules with serous infiltration (*urticaria papulosa*).

The finer *anatomical* changes in urticaria are essentially the same as in erythema papulatum (page 214). We find dilatation of the papillary capillaries, serous infiltration of the papillæ and occasionally of the rete, dilatation of the interstitial tissue spaces and the lymph spaces of the papillæ and vessels, a few lymphoid and wandering cells, occasionally microscopic extravasations of blood (Pick). In urticaria bullosa the histological changes peculiar to the formation of vesicles are also observed.

The morbid process, then, which consists in the formation of such wheals, is known as urticaria. As the result of various causes there is an outbreak of urticaria wheals, which appear either at the same time or successively in different parts of the body, so that all stages of development and involution are present at once. Upon the eyelids and prepuce the wheals give rise to considerable œdema, so that the eye, for example, may be closed for a short time.

The mucous membrane of the mouth, pharynx, and epiglottis occasionally exhibits temporary spots of redness and œdematous swellings corresponding to wheals. The uvula may thus be enormously enlarged and the epiglottis swollen until there is danger of suffocation. Such phenomena are exceedingly rare and have been seen particularly in giant urticaria (Milton), in which the skin also contains enormous, firm œdematous tumors. These are recognized as wheals by their sudden development, painlessness, and brief duration. The affection described by Quincke as "acute circumscribed œdema of the skin," and which is attended with gastric symptoms and relapses, is probably an urticaria recidiva, in which analogous processes occur on the intestinal mucous membrane and give rise to colic and diarrhœa (Riehl).

Like the other angioneuroses, urticaria is often associated with general malaise and dull pains in the limbs.

Each wheal lasts only a very short time, and the entire process is very acute and does not last, as a rule, more than one day, or at the most a few days (*urticaria acuta, evanida*). Under certain circumstances, however, the eruption may continue many weeks, months, or years, either uniformly or as the result of exacerbations and remis-

sions (*urticaria recidiva, chronica*, or *urticatio*). The significance of the affection is thus increased, and also the number and variety of the complicating symptoms. These include excoriations, pigmentations, papules and pustules, extremely annoying itching and burning at the sites of eruption, formication, a feeling of numbness in the fingers, pains in the joints and bones. The general symptoms include dyspepsia, nausea, vomiting, diarrhœa, fever, and many others which will be considered later.

The importance of this disease becomes evident only when we become acquainted with its various *causes*. At one time it is an insignificant malady, at another time it is a very serious affection.

Idiopathic urticaria is due to external injuries, to direct irritation of the skin, as in the nettle-rash due to the stinging nettle. Practically the most frequent causes of urticaria are the flea (pulex irritans), bedbug (cimex lectularius), lice (pediculi), and other insects, caterpillars, and flies.

At the point which is penetrated by the insect—for example, the bedbug—wheal-like elevations form, so that there is serous infiltration and swelling of the rete Malpighii over a certain circumference. As the wheal itches the individual scratches, and two or three of the finger nails raise the loosened layer of epidermis. This produces two or three parallel stripes of excoriation, which usually unite in a round blood crust at the bitten point. Wheals are produced, not alone in those parts which are directly irritated by attacks of fleas and bedbugs, but also in many other parts of the body over which they merely crawl or jump, and likewise in parts with which they do not come in contact, as the result of reflex irritation of the vaso-motor nerves.

Itching which is confined to one part of the skin furnishes an irritation which, being conveyed along the sensory nerves, causes a reflex development of urticaria wheals upon an entirely different part. The integument which is already attacked by urticaria is especially irritable in this regard, so that new wheals are produced by the mere contact of the fingers, still more by scratching, the rubbing of stiff linen, the pressure of garters, etc. Hence urticaria is met with in all forms of disease which are attended with itching. Many days after the original etiological factor has been removed, new wheals will be produced as the result of reflex irritation by the already existing wheals.

After a single flea-bite a child's body may be found covered immediately with a large number of wheals. Bedbugs, when present in large numbers, may give rise to exquisite chronic urticaria. When the skin is attacked under such circumstances we will find scattered excoriations, partly fresh, partly in the shape of brown stripes, which form intersecting lines. From this appearance alone the diagnosis

of chronic urticaria, due probably to bedbugs, may be made, even if not a single wheal is present at the time of examination. This diagnosis will be correct in almost every case if the patient states that the itching is present only at night.

We have already said (page 228) that mechanical pressure or mere contact, in persons already suffering from urticaria or who have a special predisposition, may produce an eruption (urticaria factitia). Such individuals suffer greatly, because the condition is not alone annoying, but may also interfere with work. Urticarial œdema is sometimes produced on the hands and in the face from washing with cold water or exposure to cold air.

Symptomatic urticaria is either acute or chronic, and is a reflex symptom due to disease of some other organ or system, or it is a concomitant symptom of other skin diseases.

It occurs most frequently from irritation of the gustatory nerves and the gastro-intestinal tract; the ingesta may have produced pronounced gastric and intestinal catarrh, with nausea, vomiting, diarrhœa, cholera-like conditions, coated tongue, fever, etc., or all such symptoms may be absent. In all these cases we must assume a special idiosyncrasy against certain articles of food or drink. It is inadmissible to assume, at least in some cases, that the urticaria is produced by the absorption of some substance from the gastro-intestinal tract, which enters the blood and acts upon the vaso-motor centres. It is a well-understood fact that the eruption is very often produced almost immediately after the offending substance or drug is placed upon the buccal mucous membrane. As a matter of course, this can only be explained as a reflex from the gustatory nerves.

The following list includes many of the substances which occasionally or constantly produce an outbreak of urticaria in certain individuals: strawberries, raspberries, currants; all kinds of fish, especially salt-water fish, lobsters, oysters, crabs; snails, sausage, ham, champagne, mayonnaise; pork, smoked, broiled, or boiled; certain kinds of cheese, ices. The list of drugs includes copaiba, turpentine, various mineral waters, quinine, etc., which also give rise to the so-called drug eruptions; urticaria may also result from the mere inhalation of balsams, turpentine, and a number of other substances.

Some writers believe that in these cases the eruption is due in great part to disgust or to imagination, inasmuch as an individual who has experienced such a disagreeable condition once or twice after eating a certain article of food will entertain a certain degree of dread when such an article is again placed before him. This is not true. I am acquainted with several cases in which, although

the individual was deceived concerning the character of the food, he was again attacked by diarrhœa and vomiting.

Gastric disturbances in general (in one patient Pringle observed urticaria with every attack of hæmatemesis) exhibit a striking predisposition to urticaria, and, inasmuch as the gastric affection may last for months, the secondary urticaria may become chronic.

Such an individual suffers very serious distress. He is constantly tormented by itching, cannot go into society, and in time emaciates because sleep is disturbed and nutrition is greatly impaired. For days, and sometimes for many weeks, with brief intermissions, the patient may be able to take only bland fluids, tea or water, or a little soup, or only warm or cold food, because urticaria breaks out over the entire body as soon as one or the other article is placed in the mouth.

In young children special attention should be paid to such conditions, because urticaria which lasts for weeks and months is often merely the reflex effect of a chronic gastric catarrh due to improper feeding, poor milk, or fatty food. Urticaria may also be produced by sudden emotional excitement, such as shame, embarrassment, anger. Among the urticarias which are due to the absorption of various substances by the blood are included those which occur in conjunction with scarlatina or measles or in the prodromal stage of variola. Urticaria has been observed repeatedly in intermittent fever or attended by an intermittent fever (febris urticata intermittens). It may also occur as a prodrome and accompaniment of the vesicular eruptions constituting pemphigus. Numerous patches of erythema appear upon the body in combination with urticaria, and pemphigus vesicles develop upon some of the wheals while the greater part of the erythema disappears, or a few urticaria wheals alone develop and the vesicles form only in such places. The latter course is observed particularly in pemphigus pruriginosus.

As a rule prurigo also begins with the symptoms of urticaria. For several months wheals alone will develop in an infant, and it is not until the second year of life that the characteristic, localized prurigo papules make their appearance.

There are also chronic and symptomatic urticarias which are the expression of a partly demonstrable disease of some internal organ or of ill-defined general conditions. These include functional disorders of the female sexual organs—dysmenorrhœa, amenorrhœa, chronic albuminuria (Leube also observed albuminuria in acute urticaria)—chronic gastric disorders, intestinal worms, intestinal catarrh, congestion of the liver, diabetes. Urticaria also occurs as the expression of a general marasmus, especially in old age and in connection with senile pruritus; also after prolonged depressing emotions, such as grief over the loss of relatives, loss of fortune,

etc. As a rule such urticarias are chronic because their causes are also of long standing.

Urticaria pigmentosa is the term applied by Sangster to another chronic form, which was first described in 1869 by Nettleship. Since then about thirty cases have been reported, mainly in England, by Tilbury Fox, C. Fox, Baker, Cavafy, Crocker, Morrow, Lewinski, Pick, and myself. In all the cases the disease had begun in the first few days or months of life. The symptoms consist of the appearance of a few wheals, which persist as such for many days or even months, gradually assume a reddish-brown color, and often swell again afresh. After their final disappearance a brown pigmentation continues for many years (in Lewinski's case, until the age of nineteen). The pigmentation either corresponds to the shape of the wheal, or it is confined to its borders and is then annular in shape, usually smooth or prominent and firm (xanthelasmoidea of T. Fox). According to the number of wheals which develop, the distribution of these patches varies; they are found on the trunk and limbs, rarely on the palms of the hands and soles of the feet. In some cases the malady stops toward the ninth or tenth year, but the pigmentation does not disappear. With the microscope Pick found hæmorrhagic foci at the site of the wheals and thus explains the abnormal pigmentation. But this does not explain the slow absorption of the pigment or the accompanying enlargement of the lymphatic glands, which can only be due to the persistent local exudations, the wheals.

It is difficult to decide whether the *prognosis* of a case of urticaria will be favorable or unfavorable. If it is due to bedbugs or to the ingestion of sausage, the condition is hardly worthy of mention and disappears spontaneously in a few days. An urticaria which is dependent on amenorrhœa or on some obscure cause, or which relapses after partaking of any article of food or drink, is a very severe malady, which annoys the patient and those around him, interferes with his occupation, reduces the health physically and mentally, and may lead to an attempt at suicide.

Hence the prognosis depends materially on the etiological factors —*i.e.*, upon the special diagnosis. In order to arrive at a conclusion it is necessary above all to decide whether the urticaria is acute or chronic. The chief reliance must be placed upon the patient's statements. If the urticaria is chronic, numerous pigment streaks will be found in addition to fresh excoriations. These have a special localization in pediculosis vestimentorum (the back of the neck, shoulders, small of the back); when due to other causes they are scattered irregularly over the body.

Urticaria which is due to the bites of insects, etc., is, as a rule, acute and temporary. Bedbugs, however, may produce a chronic

affection. Here the diagnosis is aided by the knowledge that the cause is acting only at night. Usually the children awake with urticaria, but this disappears during the course of the day, to reappear the next night and morning. The single ingestion of one of the foods in the list mentioned above will also give rise to temporary urticaria, and the prognosis is favorable.

The special diagnosis is more difficult in cases of chronic urticaria. Here we must proceed by way of exclusion, and consider all the various causes which may occasion a chronic urticaria, so that the case in question is finally narrowed down, for example, to a chronic urticaria from Bright's disease or from hysteria.

The *treatment* also depends on the etiological conditions. An acute, temporary urticaria does not require treatment, although it may be desirable to relieve the itching. We must first endeavor to remove the cause of the disease. This is particularly true of the urticaria due to bedbugs. In urticaria ab ingestis a laxative will facilitate the removal of the injurious substance from the bowels and will perhaps shorten the attack. Urticaria due to chronic gastric catarrh must be treated by suitable diet, by soda, magnesia, rhubarb, bitters, iron, baths (Marienbad, Carlsbad, Franzensbad), etc.

With regard to the urticaria due to emotional causes, experience has shown that it suddenly develops after unexpected misfortunes ; and after lasting for a long time, often for years, it either improves gradually and finally disappears, like the psychical condition itself, or it may disappear suddenly with an abrupt change in the mood or in the external conditions surrounding the patient. The urticaria sometimes ceases suddenly when the patient changes his place of abode. As soon as he leaves the scene of his sufferings he may eat and drink everything or become excited, but the malady remains quiescent. If he return at the end of three or four months he may also escape a relapse, but in some cases the urticaria reappears in a few weeks.

In very few cases are we able to remove the cause of the disease, and the symptomatic treatment is still less satisfactory. Our object is to relieve the itching as much as possible.

The patients are relieved, in general, by those measures which abstract heat from the skin, such as washing with cold water to which aromatic volatile substances have been added (acetum vini, acetum aromaticum, spiritus vini gallici, spirit. mindereri, æther sulphuricus), cold packs and douches (in rare cases lukewarm baths are preferable), fresh-water and salt-water baths.

The patient should be kept as cool as possible, sleep in a cool room, and have light bed-covering. Close, warm rooms, theatres, etc., are to be avoided. Not alone do the warmth and gaslight produce urticaria in predisposed individuals, but the fear of an outbreak

may be a psychical excitant of the eruption, if the individual is seated in a theatre, for example, and cannot follow his inclination to scratch.

The urticaria outbreaks generally occur two or three times a day, usually a couple of hours after eating, upon going to bed, or a couple of hours after falling asleep. As soon as the eruption begins the body may be moistened with one of the solutions mentioned, such as spir. vin. gallici 200.0, æth. petrol. 5.0, glycerini 2.5; or spir. lavand. 100.0, spir. vin. gall. 150.0, æth. sulph. 2.5, aconitini 1.0 (or menthol, ac. salicyl., etc.). Dusting powder is dusted over the moistened parts of the skin.

If involution of the first wheals is rapidly produced, scratching is prevented and a general violent attack is usually avoided. The application of ammonia is serviceable when a few wheals have been produced by the bites of insects, flies, or bees. In desperate cases we may try baths, to which are added soda ½–1 kgm., alum 500 gm., corrosive sublimate 5–10 gm. Temporary improvement is sometimes obtained from the internal administration of arsenic, atropine (atropin. sulph. 0.01, aquæ destill., glycerini āā 2 0, pulv. tragacanth q. s. ut f. pil. no. x. S. One pill twice a day), or ergotin.

The treatment of the urticaria which is symptomatically associated with other skin diseases, such as pemphigus, prurigo, scabies, etc., is similar to that of the latter affections.

LECTURE XVIII.

2. VESICULAR ERUPTIONS.

THE acute, non-contagious dermatitides belonging to this group are characterized by typical vesicles, which develop with the symptoms of inflammation. In addition to the common characteristics of a similar local lesion and an acute cyclical course, there is a third reason for grouping these diseases in one class, viz., the largest number of them stand in direct causal relation to certain diseases of the nerves, although in others this relation is only probable and in still others is not probable.

Hence we distinguish, 1, neuritic phlyctænoses with (*a*) typical course (herpes zoster, labialis, progenitalis), (*b*) atypical course (neuritic inflammation with formation of vesicles); 2, idiopathic phlyctænoses, miliaria rubra, pemphigus acutus.

1. Neuritic vesicular dermatoses (phlyctænoses).
   a. With typical course.

HERPES.

General Character—Herpes, Definition—Herpes Zoster.

There is hardly a single dermatological term which has been applied to more diseases than the term herpes. Etymologically it means to crawl, and hence the older writers applied it to diseases which spread slowly from one place to another, partly in a superficial manner, partly into the depths of the tissues. In the former sense it is still employed by many physicians, who speak of an herpetic eruption in the case of every chronic, usually "dry," disease of the skin. In the latter sense the terms herpes esthyomenus, exedens, rodens have been employed by Alibert and other writers of the forties for an eroding ulcer which, according to our terminology of the present day, is called serpiginous cancer or lupus. According to our notions, it is not permissible to speak of an herpetic eruption.

Since the time of Willan we understand by the term herpes an acute, benign disease of the skin, which runs its course in a brief period; it is characterized by the formation of groups of vesicles, filled with clear fluid, and usually corresponding in location to the course of certain cutaneous nerves.

Typical herpes usually exhibits the following clinical history. In a certain region of the skin, usually corresponding to the course of a nerve, one or more groups of papules, little epidermal elevations, develop in an acute manner. These are rapidly converted into vesicles by the accumulation of serum, and the acme of the process is then reached. The vesicles last from a few hours to one or two days, and dry into crusts by absorption of the serum. Normal epidermis forms beneath the crusts in consequence of the resolution of the inflammation and the cessation of the exudation, the crusts fall off, and the herpes is at an end. We may distinguish four special types :

1. Herpes zoster.
2. Herpes præputialis s. progenitalis.
3. Herpes labialis s. facialis.
4. Herpes iris et circinatus.

HERPES ZOSTER, ZOSTER (ZONA), SHINGLES.

In herpes zoster the groups of vesicles form upon one half, rarely upon both halves, of the trunk, the head, or the limbs, and usually follow the anatomical direction of the nerves.

Even at a time when the disease was known as *ignis sacer*, on account of the violent burning which attended the eruption, its unilateral occurrence was regarded as the most striking symptom. This should have aroused at an early period the notion that the eruption was connected with the cerebro-spinal system, or at least the spinal nerve ; but it was not until 1818 that Mehlis, and later Rayer, Romberg, Hebra, and Häusinger, called attention to such a relation. Bärensprung was the first to give this relation a concrete expression. He demonstrated, as the result of theoretical considerations and post-mortem findings, that the location of the zoster always corresponds to that of a spinal nerve, and is due to disease of the corresponding intervertebral ganglion upon the sensory roots of the nerves. As the trigeminus is the only cerebral nerve in whose distribution zoster appears, and as it also possesses a ganglion, Bärensprung attributed zoster of the face to disease of the Gasserian ganglion. He believed that the ganglion sends trophic fibres to the nerve trunks, and that these preside over the nutrition of the tissue elements and at the same time supply vaso-motors to the finest blood vessels of the upper cutaneous and papillary layers. Hence a change in these ganglia may also produce inflammation and exudation, which run their course as herpes.

Bärensprung recognized the following varieties, according to the nerve involved :

1. Zoster facialis ; *a*, labialis. 2. Zoster occipito-collaris. 3. Zoster cervico-subclavicularis. 4. Zoster cervico-brachialis; *a*, brachialis.

5. Zoster dorso-pectoralis. 6. Zoster dorso-abdominalis. 7. Zoster lumbo-inguinalis. 8. Zoster lumbo-femoralis. 9. Zoster sacro-ischiadicus ; *a*, genitalis.

The interesting findings of Bärensprung were confirmed by Rayer, Danielssen, Weidner, Charcot and Cotard, E. Wagner, O. Wyss, Sattler, myself, Lesser, H. Hebra, and others.

In a case of zoster frontalis (death from pneumonia) I found hæmorrhages into and destruction of the Gasserian ganglion; and in a case of zoster lumbo-inguinalis (death from urinary infiltration in the perineum) I found the affection of the corresponding spinal ganglia, illustrated in Figs. 22, 23, and 24.

Fig. 22 shows the section of an intervertebral ganglion ; the vessels are seen to be distended with blood. Fig. 23 shows a hæmorrhagic focus in the ganglion, in which a number of cells have been altered or destroyed by the extravasation into their capsule. In Fig.

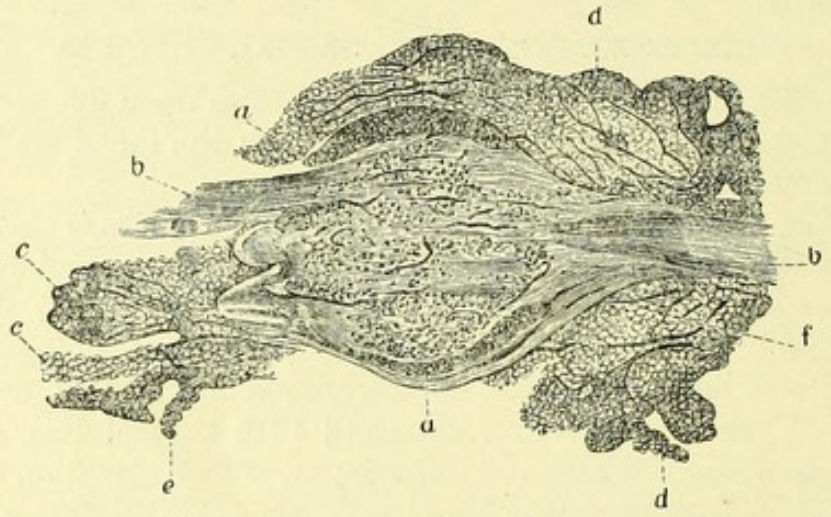

Fig. 22.—Longitudinal Section through the Third Intervertebral Spinal Ganglion of the Right Side in an Attack of Zoster lumbo-inguinalis.

aa, ganglion—the black points within it representing darkly pigmented ganglion cells, the dark streaks the congested vessels ; *a*, *b*, *c*, *d*, *e*, fat surrounding ganglion ; *f*, fat cells ; *d*, dark mottling of hæmorrhage and congested vessels ; *b b*, nerve bundle in longitudinal section ; *c c*, same in cross section.

24 red blood corpuscles are found on the inside of a ganglion cell whose protoplasm and nucleus are still intact.

Despite these positive findings, I have expressed the opinion that disease of the spinal ganglia is not in all cases the cause of zoster. It may undoubtedly develop as the result of diseases of the spinal cord, perhaps also of the brain if the vaso-motor centres are involved. This opinion is favored by the occasional bilateral occurrence of zoster, and by those cases which have been observed after poisoning with carbonic oxide ; by the coincidence with myelitis, hemiplegia, tetany, although, as shown by Charcot's case of zoster femoralis in cerebral abscess, this coincidence does not always indicate a causal relation.

There is also no doubt that zoster may be due to an affection of the peripheral nerves in any part of their course. This is shown by

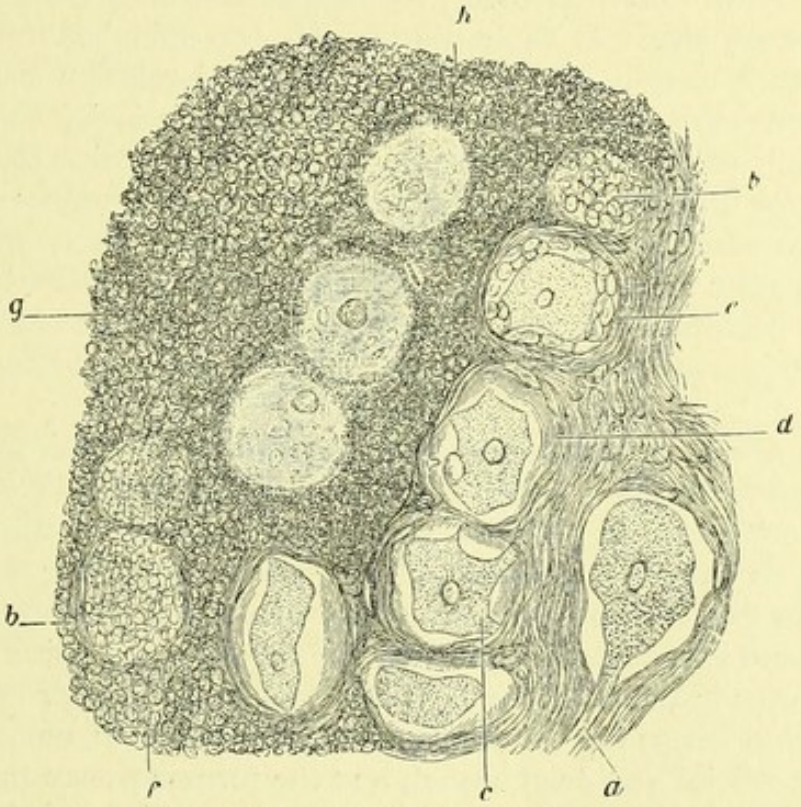

FIG. 23.—HÆMORRHAGIC FOCUS IN GANGLION.

g, ganglion cells destroyed in infiltration (*h*) ; *b b*, *f*, capsules of ganglion cells filled with red corpuscles : *e*, corpuscles lying between cell and capsule ; *d*, normal cells ; *a*, same with axis-cylinder process.

the clinical experience that it often occurs, not in the distribution of the entire nerve, but only in the distribution of a small twig. Furthermore, zoster occurs in the course of nerves which have been injured by traumata, tumors, or abscesses (Oppolzer, Dubler, Schwimmer). Finally, in a series of cases the corresponding nerve trunks have been found surrounded by perineuritic inflammatory nodules (Curschmann and Eisenlohr, Pitrés and Vaillard) or the site of interstitial and parenchymatous neuritis (Dubler).

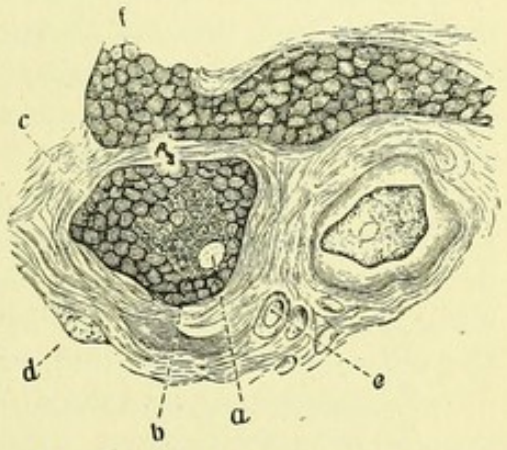

FIG. 24.—*f*, blood stasis within a ganglion; *b*, red corpuscles, the cell body and nucleus (*a*), inside the capsule of a ganglion cell; *c*, connective tissue ; *e*, exudate cells.

Whether a case reported by M. Weiss as zoster cerebralis, in which vesicular eruptions upon the fingers, associated with paræsthesiæ and hyperidrosis, repeatedly

occurred as the result of mental excitement, should really be interpreted in this way I am unable to decide.

There is no doubt, however, that zoster is always due to a disease of the nerves, either at their origin, or in the spinal ganglion, or in their further course. But as the anatomical relation between the eruption and the affected nerve is not always marked with equal distinctness, it will be sufficient, in practice, to recognize the types of zoster according to the coarser topographico-anatomical regions in which it occurs most frequently. These types are : 1, zoster capillitii ; 2, zoster faciei ; 3, zoster nuchæ et colli ; 4, zoster brachialis ; 5, zoster pectoralis ; 6, zoster abdominalis ; 7, zoster femoralis.

Symptomatology of Zoster.

The outbreak of zoster may be preceded for several days, sometimes even for three to six weeks, by neuralgic pains. These extend over the entire domain of the subsequent eruption, or they are confined chiefly to a few well-defined points, corresponding to points of division or exit of the nerve branches. In zoster pectoralis, for example, there is a painful point near the spine, at the point of exit of the posterior branches of the spinal nerves, and another point in the axillary line where the anterior branch of the spinal nerves divides into a superficial and deep branch and the former passes through the muscles to the skin. More rarely there is a third painful point at the anterior median line. The neuralgic pains are sometimes very severe, and when situated in the thorax may interfere with respiration and may simulate pleurisy. In many cases these prodromal neuralgias are entirely absent.

The outbreak of the zoster is extremely acute, whether prodromata have been present or not. Attended by a feeling of burning, very red papules, of the size of a millet seed or somewhat larger, appear in groups upon a previously reddened base. Within a few hours, or in one or two days, they develop into vesicles from the size of a pin's head to that of a pea. The period of outbreak may last from four to eight days, as all the groups may not appear upon the first day. One group may have attained its highest development while another is just beginning. The vesicles of the different groups are either entirely isolated, or, when they become larger, they are closely aggregated, and may even coalesce into a large vesicle which is nodular on the surface.

For three or four days the contents of the vesicles remain tolerably clear, but then they become cloudy and purulent, and dry, together with the tops of the vesicles, into yellowish-brown crusts. This entire period occupies from eight to ten days in each well-developed group, and, inasmuch as relapses often occur within the first week, the entire affection may last, on the average, from two to four

weeks. After the crusts have fallen off the skin is completely covered with epidermis, but exhibits a brown pigmentation for some time.

The number of vesicular groups is extremely variable. In the mildest cases there is only a single group, at the exit, the peripheral extremity, or along the course of the nerve. In moderate cases six to eight groups are uniformly distributed along the course of the nerve. In very severe cases, not alone are the vesicles of the different groups closely aggregated, but the latter are also in close proximity. The entire territory is almost uniformly covered with large vesicles, and it is only at the periphery that we can recognize, from the configuration, the development from individual groups. In such cases the pains are much more severe, the febrile symptoms more marked

There are various deviations from these normal cases of zoster. For example, the prodromal neuralgia may not cease with the appearance of the eruption, but may continue unchanged or even persist after the cessation of the zoster. Furthermore, the eruption may be aborted, inasmuch as the papules do not develop into vesicles. A few imperfectly developed groups are found in almost every case; sometimes they are the late arrivals.

A very noteworthy abnormality in the course of zoster is due to the occurrence of hæmorrhages into the contents of the vesicles and into the papillary layer. In every severe zoster some of the efflorescences, or all of them in certain groups, will exhibit hæmorrhagic instead of clear contents. These may dry up, together with the hæmorrhagic contents. In so-called *zoster hæmorrhagicus*, however, the majority of the vesicles are hæmorrhagic. The pain is then extremely violent, and the vesicles do not dry, but the covering bursts and is shed, leaving losses of substance of varying depths. These solutions of continuity are remarkably tender, and must pass through a suppurative process before the tissues, which have been destroyed by the extravasation, are exfoliated and the new skin is formed. As a matter of course, such places heal with the formation of scars because a part of the connective-tissue papillary body has been destroyed. Zoster of this variety may last six weeks to three months.

In *zoster gangrænosus* the local changes and sequelæ are still more pronounced. Even while the contents of the vesicles are still intact, a blackish-green discoloration (soon becoming blackish-brown) appears in the uppermost layer of the cutis which forms the base of the vesicle, and the necrotic change shines through the top of the vesicle. Later the vesicle dries up upon the slough or the gangrene appears from the start, as if the part had been cauterized with nitric acid, without any previous development of vesicles. Hence the sloughs may either be as large as the individual vesicles, and like them collected into groups; or as large as an entire group of vesicles,

and like them have a rounded, jagged border, corresponding to the contour of the marginal efflorescences.

In every severe zoster, especially in the hæmorrhagic forms, the base of some vesicles is found necrotic. In zoster gangrænosus the gangrene affects entire groups or even all the groups ; the pain and fever are considerable.

The sloughs are cast off as the result of desiccation and suppuration at their base, and leave large, often keloid-like cicatrices. This is almost always followed by protracted neuralgia.

An atypical zoster of peculiar characteristics is the one described by me as *zoster gangrœnosus hystericus*. It always occurs in women who have more or less distinct symptoms of hysteria. For a period of ten to twenty years a cyclical eruption of zoster recurs at intervals of weeks and months. It may be unilateral, bilateral, or distributed over numerous nerve tracts on one or both sides of the body. The vesicles in certain groups are often hardly as large as poppy seeds. Black, dry gangrene of the cutis occurs forthwith at the base of the small vesicles, sometimes of the larger ones, or even of whole groups of vesicles. Keloid-like cicatrices remain after their exfoliation.

Zoster may also run an abnormal course with regard to its sequelæ. In some cases neuralgia, paralytic symptoms, atrophy of the muscles, trophic disorders, falling of the hair or teeth (M. Singer), or anomalies of secretion are left over for months or even permanently in the territory which had been attacked by the zoster.

Paralysis and atrophy of the muscles have been observed most frequently in zoster facialis—*i.e.*, in the distribution of the trigeminus and upper cervical nerves (Tryde, Greenough, Verneuil, Letulle, etc.). Vernon reports partial paralysis of the motor oculi in ophthalmic zoster. In traumatic brachial zoster Schwimmer and Broadbent observed paralysis of the arm muscles ; Joffroy, atrophy of the deltoid. The latter author also reports atrophy of the muscles supplied by the ulnar nerve.

After zoster facialis I have repeatedly seen neuralgias, particularly in the maxillary nerve. The patients were markedly run down, because a fresh attack of tic douloureux was produced at every attempt to chew or to speak.

In a woman, four months after the cessation of a zoster collaris dexter, I observed that, following mental excitement, sweat suddenly appeared in drops over the region mentioned. Donders made the same observation in zoster facialis. Diminution of sensibility or of individual forms of sensation, even anæsthesia, have been observed in the region of zoster, despite the existence of tenderness (anæsthesia dolorosa).

Apart from such rare abnormal cases, zoster is a benign disease

which usually terminates in complete recovery and without leaving any permanent change in the skin. Cicatrices are only left over after zoster hæmorrhagicus.

It is an astonishing fact that, as a rule, zoster attacks an individual only once. Very few cases of a second attack have been reported (Wyss, Fabre, Stern, Skabell), and these were not always observed by the same physician. I have been told by two persons (one of them a physician) that they had suffered repeatedly from zoster in the distribution of the crural and genital nerves. I have already reported a ninth relape of zoster in the same patient, and since then have observed a tenth and eleventh abortive attack. But this case was such a remarkable exception in other respects (zoster gangræ-nosus punctatus et striatus) that it does not disprove the rule, and should rather be included in the category of zoster hystericus.

Quite a number of exceptions to the typical one-sidedness of the eruption have been observed, particularly in cases of zoster facialis and cervico-brachialis. I have also observed a case of bilateral zoster sacro-femoralis and ischiadicus. A case described by Tomaso de Amicis as zoster bilateralis universalis should probably be regarded as herpes iris.

The disease occurs in youth as well as in adult life, and even in old age ; it is much rarer in children. It is more frequent at certain seasons of the year, usually at times when pneumonia and the various forms of erythema prevail. In some months there are no cases or they occur only sporadically.

This quasi-epidemic occurrence of zoster might lead us to suspect a miasmatic origin. But this would still leave unexplained the fact that such a virus attacks only a single ganglion and that the individual is affected only once. An analogue might be found, however, in the observation, which has been repeatedly made, that zoster (generally frontal) occurs after poisoning with carbonic oxide. Here the entire amount of blood contains a poisonous substance, and yet the disease appears only in the distribution of one spinal nerve or of a branch of the trigeminus (in Sattler's case the first branch was involved, and degeneration of the nerve was found as far as the Gasserian ganglion).

The difficulty in explaining the development and distribution of the disease led Pfeiffer to make statistical investigations (one hundred and seventeen cases). He found that the distribution coincided with the ramifications of the blood vessels, and believed that he had thus found another argument for the miasmatic infectious theory of the origin of zoster. This theory has been disproven by the observations of myself and others (Pick-Weiss).

Like every other form of neuritis, zoster is perhaps due occasionally to catching cold. It has also been observed repeatedly in those who

were undergoing arsenical treatment, but I agree with White, Faber, and Juliusburger that this coincidence is accidental.

We may disregard all these accidental factors, inasmuch as we are possessed of positive facts in regard to the *etiology* of zoster. We refer to the hæmorrhages and inflammatory irritation of the spinal ganglia and the Gasserian ganglion (to which attention has already been called), and to the neuritides which have been found on microscopical examination.

Among the exciting causes may be mentioned neoplasm, tuberculosis, abscesses, periostitis, pleurisy (in so far as it irritates adjacent nerve trunks), poisoning with carbonic oxide, traumata (gunshot wound, contusion, blow of a whip), which may be immediately followed by an eruption of zoster—for example, in the distribution of the frontal, the brachial, ulnar nerves, or of one of the spinal nerves.

I exclude from this category the diffuse erythemata (glossy skin), with painful inflammation and the formation of vesicles, which occur in a chronic manner in the distribution of injured nerves (Mitchel, Morehouse and Keen, Mougeot, Schieferdecker, etc.), and are often reported as zoster. They do not run the typical and cyclical course of the latter disease.

Localization of Zoster.

In order to refer the zoster to the corresponding nerve tracts, it is necessary to be acquainted with the peripheral distribution of every sensory nerve.

Voigt has done excellent work in this field by carefully dissecting the cutaneous nerves as far as their most peripheral terminations, and thus determining the boundaries of the individual nerves. It was found that in the median line of the body, as well as in other regions, the cutaneous nerve passes into adjoining tracts, so that strict boundaries can hardly be said to exist. In addition the spinal nerves, near their exit from the cord, are connected by anastomotic fibres with upper and lower nerves, and also with nerves from the other side of the body. Hence disease of one nerve may result in irritation and inflammation along the anastomotic fibres.

Zoster facialis is the most variable in this regard, on account of the numerous anastomoses between the branches of the trigeminus, facial, and upper cervical nerves. It occurs very often as zoster frontalis, corresponding to the distribution of the frontal twig of the first branch. Closely aggregated groups of vesicles appear upon one-half of the forehead, separated sharply from the median line, corresponding to the distribution of the supraorbital nerve which emerges from the supraorbital foramen; the vesicles also appear on the upper lid and extend to the angle of the eye, corresponding to the supratrochlear nerve. This zoster is very often hæmorrhagic or gangrenous.

As a result of the implication of ethmoidal and infratrochlear branches of the nasal nerve, there is usually swelling of the nasal mucous membrane and an eruption on the corresponding half of the nose as far as the lip. When the zygomatic and lachrymal branches are also involved the eruption will appear on the adjacent part of the temple. Such an extensive distribution constitutes zoster ophthalmicus.

This is an extremely painful disease and may even terminate in death. As a result of implication of the ciliary nerve and the long root of the ciliary ganglion, we find injection of the ciliary vessels, papules, vesicles, and ulcers in the cornea, iritis and xerosis of the cornea; conjunctivitis is found in involvement of the lachrymal branch. In this affection, known as "herpes ciliaris," the connection of the eruption with one of the ciliary nerve twigs running to the cornea has been demonstrated by Iwanoff. Von Stellwag not only regards this disease as identical with zoster, but he also attributes the corneal phlyctænulæ which are so frequent in scrofulous and feeble children to direct irritation of the ciliary nerves and to the diminished power of resistance of the vessels in such individuals. The neuralgic pains and photophobia in these cases are sometimes extremely violent. They may finally result, as in Wyss' case, in phlebitis (around and within the globe), panophthalmitis, and, from extension of the phlebitis to the skull, in pyæmia, meningitis, and death.

A second form of zoster facialis is located mainly on the cheek, corresponding to the ramifications of the superior maxillary nerve, with groups running toward the ala nasi and the lower eyelid, supplied by the infraorbital branch. Upon the corresponding mucous membrane of the gums and the pharynx there may be painful red patches and groups of efflorescences of brief duration, due to implication of the palatine and pharyngeal nerves. Notable difficulty in deglutition and severe toothache may accompany the disease, and paralysis of the palate on the same side may be left over permanently or for a long time. Protracted neuralgic toothache, falling out of the teeth, and atrophy of the alveolar process have also been observed as the result of the disease in the distribution of the posterior alveolar nerve (M. Singer).

Zoster in the distribution of the third branch of the trigeminus corresponds in the main to the inferior branch, which is composed chiefly of sensory fibres. Groups of vesicles appear on the anterior part of the concha and the adjacent temple, in the external auditory canal as far as the membrane (anterior auricular nerve), upon the angle of the chin (mental nerve); occasionally there are irritative conditions and epithelial losses on the same side of the tongue (lingual nerve).

A few groups of vesicles may also appear on the posterior surface of the concha (post-auricular branch of the facial nerve), and on the temple, the ethmoidal region, the cheek, lower jaw, and the upper anterior part of the neck (temporal, zygomatic, buccal branches, and the maxillary and superior subcutanei colli nerves which form a plexus with the mental nerve).

The extent of facial zoster may be still further increased by including those tracts which are supplied by the upper cervical nerves, particularly the occipitalis major. This arises from the third cervical and supplies the back of the neck and posterior surface of the concha.

Sometimes only a few groups of zoster are present. At other times the entire facial region may be occupied, either with almost confluent, closely aggregated groups of hæmorrhagic efflorescences, or with disseminated, normally developed vesicles, including many groups of abortive papules. In rarer cases there is a coincident eruption on the occiput and back of the neck, from implication of the branches of the first three cervical nerves (zoster occipito-collaris). Finally, in very rare cases zoster facialis is bilateral, as first described by Hebra and illustrated in his Atlas.

Neuralgias, muscular paralyses, and trophic disorders (shedding of the hair and teeth, atrophy of the muscles) are most frequent after zoster facialis of all forms. Inasmuch as the zoster is due to disease of the trigeminus, and muscular paralysis presupposes an affection of the facial nerve, it is not easy to explain this relation. Strübing believes that the inflammation extends from the peripheral trigeminus branches to the adjacent and anastomosing branches of the facial, and thus gives rise to a neuritis which extends to the trunk of the facial. Eulenburg calls attention to the vaso motor and trophic fibres which the facial nerve contains according to Samuel and Schiff (from the pneumogastric), so that an inflammation of the facial nerve can produce zoster of the facial and cervical regions by implication of such accompanying fibres, and also trophic and paralytic conditions. In certain cases a disease within the Fallopian canal, before the chorda tympani is given off, has been assumed, so that the facial nerve appears to have been affected at the start. E. Remak believes that it is more plausible to assume that the same agent (a cold) which affected the branches of the trigeminus also attacked the facial nerve and caused a disturbance of its function.

In zoster occipito-collaris we find, in addition to groups of vesicles upon the occiput (occipitalis major and minor), others upon the posterior surface of the concha and the lobe of the ear (auricularis magnus) and extending toward the median line of the neck and beneath the chin (subcutaneous colli from the upper cervical nerves).

In zoster cervico-subclavicularis the eruption begins on the neck, at the border of the scalp, extends downward and outward on the side of the neck to the shoulder, then anteriorly between the clavicle and the nipple, and a part of the neck above the clavicle. The distribution corresponds to that of the fourth cervical, subclavicular, and the ascending cervical nerves.

Zoster cervico-brachialis is due to disease of the brachial plexus, which is formed by the union of the anterior branches of the four inferior cervical nerves and the first and second dorsal nerves.

The plexus sends cutaneous branches to the neck and shoulder; the first and second dorsal nerves send cutaneous branches to the posterior and inner part of the arm and to the anterior part of the thorax over the first and second ribs. Such an attack of zoster may involve the flexor and extensor aspects of the arm to a variable extent, sometimes spreading over the forearm and even to the little finger; there may also be groups of vesicles over the first and second ribs as far as the sternum. Coincident diseases of the associated middle cervical nerves may also produce an eruption on the back of the neck as high as the occiput and in the region of the shoulder (the latter corresponding to the superior brachial cutaneous nerve.

I have seen one case of bilateral zoster occipito-collaro-brachialis in which the vesicles extended to the tips of the fingers and the palms of the hands.

In zoster pectoralis the nerve type of the disease is shown most clearly. Every spinal nerve divides, immediately after its exit, into an anterior and a posterior branch. The posterior branch perforates the muscles of the back, partly supplying them, and sends cutaneous branches to the neighborhood of the median line. The anterior branch runs forward as the intercostal nerve and divides into an external and an internal branch. The former perforates the intercostal muscles, supplies the integument of the lateral region of the back, and runs forward to the median line as a cutaneous branch, forming in the thoracic region the pectoral cutaneous nerves, in the abdomen the abdominal cutaneous nerves.

Zoster pectoralis occurs at times as a row of groups of vesicles which extend from the spine to the anterior median line, and ranging from the width of one to three intercostal spaces. The groups of vesicles not infrequently coalesce. They are often hæmorrhagic in part or entirely, and are then extremely painful; this is attended with suppuration and cicatrization, and in many cases the disease lasts three months. In other cases there are very few groups of vesicles—for example, one near the spine, a lateral group at the exit of the external ramus, and one at the peripheral extremity in the anterior median line. There may only be a single group. As a rule the

terminal groups on the back and in the anterior median line pass a little beyond the middle.

Zoster pectoralis very often begins with a prodromal neuralgia. Cases have been reported in which intercostal neuralgia preceded the zoster for years. The disease has often been observed as a complication of pleuritic irritation, pleurisy, or vertebral caries or cancer. Stitches in the side and difficulty in breathing are usually present during the zoster, and neuralgia is often left over after the disease.

Concerning zoster dorso-abdominalis and lumbo-inguinalis not much more need be said than the name signifies. It is to be remembered that the posterior branches of the lumbar nerves send branches to the integument of the buttocks, and of the outer side of the thigh as far as the trochanter. Hence groups of vesicles are found from the sacrum to the trochanter over the glutæi muscles, and also upon the mons veneris, groins, and on the scrotum, corresponding to the ileo-inguinal and scrotal nerves.

Zoster lumbo-femoralis corresponds to disease of the second and fourth lumbar nerves. The eruption appears over the lumbar and sacral portions of the spine, the buttocks, anterior surface of the thigh on the outer and inner aspects as far as the knee and along the calf, also upon the scrotum and the labium majus, corresponding to the anterior external femoral cutaneous, genito-crural, sensory branch of the obturator, and the middle cutaneous and saphenous branches of the crural nerves.

In zoster sacro-ischiadicus and sacro-genitalis the eruption appears upon the buttocks, sacrum, perineum, posterior surface of the scrotum, anal region, labia, and introitus vaginæ. The latter localizations, and also an eruption on the dorsum penis, correspond to the pudendal nerve, eruptions over the trochanter and the tuber ischii to the posterior cutaneous magnus nerve. The sciatic nerve does not give cutaneous branches to the thigh, and on the lower extremity only supplies the integument of the dorsum and sole of the foot.

With regard to affections of the pudendal nerve, I may state as an interesting fact that I have repeatedly seen zoster of the penis and scrotum which was confined strictly to the median line of the penis.

The *anatomical* changes in zoster refer to the affected nerves and to the vesicular eruption. The former have already been considered (page 238, Figs. 20 to 22).

Haight's finding of inflammatory cell infiltration around a nerve fibre of the deeper layers of the skin may be seen in every inflammation of the skin and is not peculiar to zoster.

The changes in the integument are the same as those known with regard to the formation of inflammatory vesicles, and which I described apropos of erythema vesiculosum (Fig. 21). It is character-

istic of all forms of herpes and also of zoster that the vesicles develop in the deeper layers of the rete, so that the cells of the latter appear separated into a network whose meshes are filled with clots of fibrin, serum, and exudate cells (wandering cells). The tissue of the papillæ and of the corium is also infiltrated with exudate cells and serum, the vessels are distended, the meshes of the connective tissue are enlarged. Lesser believes that the proliferating and degenerative processes in the epidermis precede the nutritive changes in the papillary body. Weigert and Neisser entertain the opinion that the necrosis of peripheral parts as the result of changed nerve influence is the primary and essential feature in the zoster eruption, and that suppuration of the eruption only occurs after the subsequent entrance of infectious matters. This view is contradicted by the actual facts.

The more intense the local inflammation the deeper the cellular infiltration and exudation will extend along the vessels, the larger the vesicles, and the more developed the network. In the hæmorrhagic forms the extravasation of blood into the papillæ and upper layers of the corium mechanically destroys a part of the connective tissue, and produces a loss of substance which heals only after suppuration and cicatrization. In the ordinary vesicles only a part of the rete is lifted up. Normal epidermis forms over the intact papillæ (which are partly covered with perfect cells); this raises the drying vesicular mass, the crust, and recovery occurs without cicatrization.

A clear idea may be formed of the relationship between the disease of the ganglia and nerves and the skin disease in the shape of zoster. It must be remembered that every spinal nerve is a mixed nerve and contains sensory, motor, vaso-motor, and secretory (perhaps trophic) fibres. If the disease irritates the sensory and vaso-motor fibres, the peripheral area of distribution will undergo inflammation in the shape of papules and vesicles (or hæmorrhages). The latter then undergo development and involution in the manner peculiar to all such forms. In view of the histological findings, it is peculiar that the nerve lesion is a sudden and transitory one; the irritation does not continue, and hence the peripheral inflammation of the skin is not repeated. Even the larger hæmorrhages in the ganglia are evidently absorbed rapidly, and the ganglia and nerve cells are undoubtedly regenerated.

If the processes in question entirely destroy certain nerve fibres, the trophic influence of the latter will be entirely abolished and tissue necrosis (gangrene) will set in at once in their area of distribution.

In the same way the occasional paralyses, changes in secretion, and neuralgias are explained by irritation or destruction of the corresponding motor, secretory, or sensory fibres. The permanence of

such symptoms after the termination of the zoster is explained by the permanence of the nerve changes: the neuralgias by irritation of perineuritic processes, the paralyses by lasting destruction of motor fibres, etc.

With regard to *diagnosis*, I refer you to the symptomatology, which readily enables us to recognize even an abortive or rudimentary zoster. This also furnishes the data for prognosis.

Treatment is unable to shorten the typical morbid process or to influence it in any way. We are merely called upon to relieve annoying symptoms.

Zoster produces the least annoyance when the vesicles remain intact and dry up. Hence cold or warm compresses should not always be applied in order to relieve the feeling of burning, because they macerate the epidermal covering. The spots which are deprived of covering then pain intensely, because the papillary body is exposed or is covered only with a thin layer of epithelium. The pain is best relieved by dusting with starch, with or without a little powdered opium. This facilitates desiccation and also prevents the rubbing of the underclothing against the vesicles and their destruction. But if the vesicles burst as the result of excessive distention, and extensive raw surfaces are exposed in this way; or if, as in hæmorrhagic zoster, suppurating wound surfaces are presented, it is best to cover the wounds with bland oils and ointments—not with unguentum diachyli, which burns very severely, but with unguentum simplex, ceratum simplex, or an ointment of cera flava and oleum olivarum 1 : 3, to which extractum belladonnæ or extractum opii aquosum (0.5 : 50.0) or cocaine has been added.

Violent neuralgias, either during the prodromal period or later, intense diffuse pains in the area of eruption, and the frequent insomnia of the eruptive period are best relieved by subcutaneous injections of morphine, the internal administration of chloral hydrate or opiates, or the local application of plasters containing opium. In many cases, however, all these measures are useless and relief is not obtained until the beginning of the stage of desiccation.

A difficult problem in therapeutics is presented by the neuralgia which is occasionally left over after zoster. Apart from the subcutaneous administration of narcotics which is resorted to in such affections in general, relief is sometimes obtained from the use of quinine or the methodical administration of Fowler's solution. We begin with two drops t. i. d. in an ounce of aqua fœniculi or aqua anisi, and increase the dose every third day by two drops until thirty to forty drops are taken daily. When the improvement is pronounced, or if gastric distress and diarrhœa supervene, the dose is gradually diminished to fifteen to twelve drops.

LECTURE XIX.

HERPES LABIALIS—HERPES PROGENITALIS—HERPES IRIS ET CIRCINATUS —MILIARIA RUBRA, ALBA ET CRYSTALLINA—PEMPHIGUS ACUTUS.

We have entered so extensively into the description of herpes zoster that a briefer consideration of the other forms of herpes and the acute phlyctænoses is permissible.

HERPES LABIALIS.

The term herpes labialis, or, according to Hebra, herpes facialis, is applied to that form of disease in which one or more groups of vesicles develop acutely upon the lips, alæ nasi, and around the mouth.

Their development is attended with a burning sensation. The vesicles last one to three days, then desiccate and the crusts fall off. An analogous eruption is sometimes found on the mucous membrane of the cheek, the hard and soft palate, tongue, and inner surface of the alæ nasi. The epithelium in such places becomes gray and cloudy, is exfoliated, and the spots are then red and remain tender for a few days. Difficulty in deglutition and annoyance in speaking and chewing are attendant symptoms. It is well known that this herpes appears in the course of ephemeral affections and in acute febrile diseases generally, in very trifling as well as in severe ailments (coryza, pneumonia, typhoid fever, etc.). The appearance of herpes labialis or facialis is not by any means to be considered a favorable omen regarding the course of the process with which it is associated, since it may appear in typhoid with a fatal termination.

In any case we are not in a position to assert anything definitely as to the cause of this remarkable process. It is true, Bärensprung has expressed the opinion that herpes facialis represents a zoster which is restricted, as it were, to the most peripheral nerve twigs of the trigeminus, and whose cause possibly lies in the irritation of a ganglion in a peripheral location, as. for instance, the ganglion incisivum; but even he does not maintain this opinion with regard to all the eruptions appearing in the form of herpes labialis. The latter, moreover, differs from zoster in that its groups are usually situated irregularly on both sides of the median line and not corresponding to a single nerve twig; that it may repeatedly attack one and the same

individual—namely, as often as a febrile disease gives rise to it. Gerhardt, on the other hand, thinks that it may be caused perhaps by the irritation of the branches of the trigeminus which run in osseous canals, through pressure exerted by concomitant blood vessels which are overfilled in states of fever.

HERPES PREPUTIALIS S. PROGENITALIS.

The term herpes preputialis s. progenitalis (better, herpes genitalium, according to a suggestion made by Besnier) is applied to an acute eruption of groups of vesicles on the male or female genitals. Its location is on the prepuce, the coronal sulcus, and the adjoining integument of the penis ; in the female, on the prepuce of the clitoris, the labia minora, and possibly the adjoining portions of the inner surface of the labia majora.

One or several groups of vesicles appear upon the localities named, associated with burning and itching sensations ; they are usually miliary in size, though they may be as large as the head of a pin or slightly larger, and are seated on a reddened or swollen base. The accompanying œdema is considerable and extends very far, so that, for instance, the prepuce forms a thick wall or the labia minora become tense from serous infiltration. There is not uncommonly superadded, after the epithelium ruptures, a transudation of serum with catarrhal secretion of the mucous membrane of the urethra and the vagina. An analogous inflammatory patch may form in the anterior part of the male urethra, associated with a sero-purulent discharge and burning during micturition.

In two or three days the vesicles dry into crusts, and after the lapse of a like number of days the crusts have fallen and the seat of the eruption has healed. As in zoster, so in herpes progenitalis, some or all of the vesicles may enclose hæmorrhagic contents. In that case, after the vesicles rupture suppuration will occur owing to the hæmorrhagic destruction of the uppermost papillary layer, last from ten days to two weeks, and lead to cicatrization after the destroyed tissue is cast off.

The *diagnosis* of this affection is generally rather easy, since the groups of vesicles, even when the several efflorescences have coalesced to a patch the size of a cent or nickel, will show by the scalloped appearance of the margins that they are composed of single vesicles and thus disclose their herpetic character. It is only when the external covering has been removed by mechanical influences, such as scratching, agglutination of the linen, or great exudation and hæmorrhage, exposing a layer of tissue with a yellow or bloody coating, or even in the crust-forming stage when some purulent secretion has been encapsulated, that it would not be easy at first sight to distinguish the disease from a commencing chancroid or a specific primary affec-

tion. Especially when it has been ascertained that the person within a corresponding space of time—that is, at most a week—has been exposed to an infection by coition, judgment must be suspended. For even if the existence of herpes is beyond doubt, infection might have occurred at the same time, and its effect would be manifest only in the further course by the formation of a chancrous ulcer or an induration. Aside from this possibility, herpes progenitalis always runs an acute course and invariably admits of a favorable prognosis.

The frequent recurrence of herpes progenitalis is peculiar. There are persons, especially males, who are attacked by it several times during the year. Many patients positively assert that they must be prepared for an eruption of herpes after every coition. It is difficult to say whether this statement is founded in fact.

This would presuppose a mechanical *cause* for the occurrence of herpes. I have found some persons, who were liable to recurrent herpes progenitalis, suffering from digestive disturbances (as did Plumbe), anæmia, cold hands and feet, and hyperidrosis, hence of "nervous" habit. Others (Diday and Doyon) believe herpes progenitalis to be related to specific genital affections. At any rate we are not in a position to assign any cause for it. Bärensprung looks upon this herpes, too, as a kind of peripheral zoster genitalis. It should be noted that, contrary to zoster, the groups of vesicles in herpes progenitalis are by no means confined to one half of the affected part, but are situated quite irregularly, just as the frequent recurrence of herpes progenitalis is opposed to the invariably single appearance of zoster.

As to *treatment*, in the typical acute course obviously no active interference is called for. We restrict ourselves to dusting with starch, and especially the insertion of charpie or cotton coated with starch between the prepuce and glans and in the labial furrows, in order to moderate the burning sensations, prevent the maceration of the vesicles, and favor their drying up. Where the seat of the eruption is laid bare, and in the case of suppuration, indifferent dressings which will prevent the formation of crusts will be employed, such as simple cerate, etc.

HERPES IRIS ET CIRCINATUS.

Among the acute vesicular eruptions herpes iris et circinatus is usually enumerated as a separate variety. The term is applied to an acute vesicular eruption in the well-known form of the iris, *i.e.*, concentric circles, or in a single circle (circinatus).

This iris form arises in this way: A single vesicle appears, and while this in a day or two is beginning to sink down a new circle of vesicles, followed immediately by a second, appears round about it on the peripherally advancing redness of the skin. After the central

vesicle has undergone complete involution, only the external circle of vesicles remains and includes a reddened or even pigmented portion of skin—herpes circinatus.

The vesicles of herpes iris et circinatus are from a pin head to a pea in size; vesicles belonging to the same group being usually of equal size. Sometimes the central vesicles coalesce with the peripheral in an unbroken ring, whose scalloped appearance alone indicates its origin from separate vesicles. The vesicles, as a rule, are very firm to the touch, being formed by a serous exudation in the papillary layer and swelling of the deep layer of the rete. For this reason they break very rarely, and hence weeping and crusting will hardly ever be observed. On the contrary, they undergo involution, as a rule, in from eight to ten days, by absorption of their contents, leaving some pigmentation and rarely slight desquamation.

If I were called upon to make a definite statement as to the importance of herpes iris et circinatus, I would feel much embarrassed. It will be remembered that, in fact, it has been mentioned before in connection with erythema iris and circinatum. Indeed, we have every reason to identify these two processes (Hebra, Köbner). In the first place, the disease appears in the above-described combination (page 214) with erythema iris and circinatum, and, second, it appears in the pure herpetic form, in the same typical localization on the dorsum of the hand and foot, with a like cyclical course within two to three weeks, an annular type—in short, with the identical character of erythema multiforme.

In the same manner as erythema multiforme, but more frequently, I have also observed herpes iris recidivus for years, in continuous or interrupted relapses, on the mucous membrane of the lips, cheeks, tongue, palate, almost exclusively in anæmic hysterical women, and more rarely in anæmic men suffering from digestive disturbances. The affection is quite troublesome, owing to the painfulness of the several patches deprived of epithelium on the oral mucous membrane; and the sharp limitation of the several lesions and their gray coating with macerated epithelium may readily cause them to be mistaken for syphilitic patches.

There was a time when herpes circinatus was looked upon as a fungous disease, the herpes tonsurans of our day. This was especially the case with Bateman, who enumerated and figured it as porrigo scutulata, and even later among the French, particularly Cazenave. I have repeatedly seen cases—as in a tailor's apprentice of sixteen and a girl of twelve—where there was on the dorsum of the left hand, or, in another case, on the forearm of a child, a double circle as large as a dollar and one-sixth of an inch high, consisting of very firm vesicles coalesced into a dentated ring, while other isolated miliary vesicles were scattered over the dorsum of the hand. Micro-

scopical examination showed an abundant network of mycelia within the rete cells, so that in all these cases there was no doubt of the fungous nature of the affection which presented the picture of herpes circinatus.

It will be seen, therefore, how hard it is to decide this question, since it requires a careful microscopical examination. I should advise solving the problem in the following manner: When herpes iris et circinatus manifests the type of erythema exsudativum—*i.e.*, when the dorsum of both hands and feet forms the starting point, the first and chief localization of the eruption—we shall identify it with the erythema. When, on the other hand, it appears anywhere else—for instance, on the face, the cheek, or asymmetrically on one hand only—we will be justified in suspecting that we have to deal with herpes tonsurans (that is, with an infectious disease due to a fungus), and furnish the proof by a microscopical examination.

Having thus encroached upon the field of differential *diagnosis*, I must make mention of another circumstance, namely, that there is one more chronic, very grave vesicular disease of the skin, pemphigus, the most serious form of which, pemphigus foliaceus, generally begins with the development of circinate, iris-like vesicles. In such a case, indeed, the diagnosis cannot be made at once. It will take from six to eight weeks for the character of the disease to become clear ; for herpes iris et circinatus, as a rule, remains in the eruptive stage two weeks, and in two weeks more undergoes complete involution, rarely persisting longer, while in pemphigus fresh vesicles appear even after many weeks and the process proves chronic.

This leads me to make another remark in opposition to an esteemed colleague, Duhring, who of late years has pleaded in numerous publications for the existence of a disease called "dermatitis herpetiformis." Although his view has since been shared by many, to me it is beyond question that this author is in error in his view, which would lead to serious confusion in the interpretation of the related morbid processes. For, according to him, that category includes not only the processes just discussed—herpes iris and pemphigus circinatus—but also erythema bullosum, pemphigus gestationis, and pemphigus acutus. A pyæmic, fatal disease formerly added to the group by him, one characterized not by vesicles but by pustules, and which we shall consider later under "impetigo herpetiformis," has since been excluded by Duhring himself.

The *treatment* of herpes iris et circinatus, in view of its acute and typical course, is indifferent, and only in violent and inflammatory conditions or when joint affections are superadded, as in erythema, will cold compresses, etc., be employed.

MILIARIA, SUDAMINA.

Among the acute vesicular eruptions belongs also miliaria, so-called prickly heat, which played an important part in the pathology of earlier days, even epidemics of miliaria having been repeatedly reported (Italian, migliaria).

Three varieties of miliaria are enumerated: 1, miliaria rubra; 2, miliaria alba; and 3, miliaria crystallina.

The term *miliaria rubra* is applied to an eruption occurring acutely, and generally with profuse perspiration, over the trunk and the extremities. The efflorescences consist of small, millet-seed-sized red vesicles enclosing clear fluid at their points and seated on a red base. When their covering epidermis becomes macerated, softened, and their contents are turbid, the vesicles appear opalescent and we have the second variety, *miliaria alba.*

Hebra has called attention to the fact that this form of miliaria has the importance of an exanthem due to perspiration, and hence should be termed eczema sudamen or sudamina (prickly heat; calori of the Italians). Haight has figured an instructive microscopical section of a miliaria vesicle. This shows the corneous layer alone detached in the shape of a vesicle over the opening of a sweat gland. The eruption is very frequently observed during the hot summer days, but also in winter in persons who have been in profuse perspiration. I have noticed such general eruptions sometimes even in consequence of irritating ointments. We can convince ourselves that we are dealing merely with a slight degree of eczema due to sweat, since when the macerating and irritating perspiration continues the eruption actually becomes a weeping eczema from the scratching caused by the itching. When, however, such irritations are avoided, the vesicles undergo involution with slight desquamation.

Accordingly, miliaria rubra and alba can very often be observed in connection with febrile diseases. Their development is frequently preceded by a stinging sensation as of needles in the skin. After the appearance of the eruption, however, it is the itching that troubles the patients.

But, in view of all these symptoms and the above-named anatomical finding by Haight, we have every reason to look upon the vesicles as caused, not by the irritating influence of the sweat which has exuded upon the surface of the skin, but by the accumulation of the perspiration between the epidermal layers filling the openings of the sweat glands.

Aside from this, however, there is undoubtedly an epidemic miliaria in which, according to the reports of Keesbacher (1883), Drasche and Weichselbaum (1892) of similar outbreaks in Carniola, the exanthem consists of red nodules, vesicles, and pustules on the trunk and

cervical region. In these cases the eruption proved to be a local phenomenon of an evidently infectious disease (form of malaria?), which ran its course with one or more rigors, followed by profuse perspiration, weakness, stupor, and high fever. In many cases it proved fatal, the mortality of the epidemic of 1892 being twenty-four per cent.

The post-mortem examinations have thrown no light on the disease. The bacteriological examination, according to Weichselbaum, is still *sub judice.*

Miliaria crystallina, on the other hand, unquestionably is entitled to the importance of a peculiar cutaneous exanthem, both from its clinical character and its etiological factors. The vesicles of miliaria crystallina form transparent, pale, dewdrop-like efflorescences the size of a grain of sand, which are often perceived better with the finger than with the eye ; they occur in large numbers on the trunk, especially the chest, abdomen, and the lateral thoracic region, as well as on the flexor surfaces of the extremities and the neck. They persist for several days up to one or more weeks, according to the conditions under which they appear. Their contents have a faint alkaline reaction and are never acid. During the variable stated time of their existence they do not enlarge ; only here and there some lentil- or bean-sized vesicles occur among them, but these have an exceedingly thin epidermal covering. Their contents never become turbid or purulent, but they remain the same dewdrop-like efflorescences ; in fact, this is the only exudative exanthem which continues unchanged even in the cadaver.

As a rule they disappear in the following manner: The cover of the vesicles disappears spontaneously or under the influence of the perspiration, so that there is no true desquamation. A portion of the vesicles thus undergoing resolution and leaving spots covered with skin while fresh eruptions occur, the entire process may continue for several weeks. The first eruption as well as the various relapses are usually preceded by rigors. Hebra has emphatically pointed out that we have every reason to look upon miliaria crystallina as the expression of a metastatic process caused by such conditions in the internal organs as are apt to give rise to metastasis on the common integument. Among these are endometritis, metrophlebitis, the puerperal process in general, endocarditis, typhoid fever, articular rheumatism, acute exanthemata like scarlatina (miliaria exanthematica). For during all these processes, and usually in their later course, miliaria is apt to occur, and accordingly we read of a miliaria typhosa, puerperalis or uterina, pectoralis or cardiaca.

The *diagnosis* of miliaria crystallina is not difficult, since no exanthem shares the dewdrop-like quality of the efflorescences. As the affection causes no inconvenience either by itching or more intense

alterations of the skin, it furnishes no occasion for the discussion of the prognosis or treatment ; its existence depends upon the causative febrile disease, and in a concrete case we have to consider only the prognosis of the latter, not of the miliaria.

PEMPHIGUS ACUTUS S. FEBRILIS.

To the acute vesicular eruptions must be added the so-called pemphigus acutus. By this term we understand a disease with an acute course limited to a few weeks, in which vesicles appear acutely, with or without symptoms of fever. The efflorescences are pea- to bean-sized and larger, filled with watery fluid. They occur irregularly scattered in the face, on the trunk and the extremities. After persisting for some hours or days the vesicles dry up, and when the crusts have fallen they leave red spots which later become pigmented.

The course of the disease is acute, the vesicles appearing within the first week or two in irregular outbreaks ; then the eruptions become more sparse, the old vesicles heal, and finally the entire process terminates and does not relapse. Pemphigus acutus has also been observed in smaller or larger endemics in children.

There is no doubt that for many cases Hebra's objection holds good. He maintains that some physicians have diagnosticated as pemphigus acutus such acute vesicular eruptions as occur in varioloid and varicella bullosa, in erythema bullosum, herpes iris and circinatus, even in eczema and urticaria bullosa ; or, finally, a peculiar form of impetigo of the face, impetigo contagiosa, epidemics of which were observed during a general vaccination, first in 1885 at Rugen, and subsequently elsewhere (by Protze in Elberfeld, by Géronne in Cleve, Bohemia). This disease we shall discuss under eczema. Pontoppidan has even directly misapplied the term to the disorders enumerated above. Moreover, the several acute eruptive periods of a chronic pemphigus have also been mistaken for pemphigus acutus.

In the meantime many authors, especially pædiatrists like Thomas, Moldenhauer, and others, have of late years published reports of a vesicular eruption observed in a brief period among a large number of children in one locality (occurring in isolated cases even in adults) ; the disease had a prodromal, an eruptive, and a declining stage, but terminated, without exception, favorably.

It is true there has never been a lack of opposite opinions. Some of these men diagnosticated the vesicular eruption as varicella. This would harmonize with the fact that some physicians trace these endemics to a contagium and name the affection pemphigus contagiosus infantum. Others, again, like Bohn, ascribe the epidemic to the influence of hot baths used by one and the same midwife for all children entrusted to her care. Still others, seeing that only children were affected who were under the care of one midwife, attributed

the process to infection from this person. In short, the most various and opposite views regarding the disease were expressed. I myself, however, can say nothing concerning the reported fact as to the occurrence of a vesicular eruption of an acute course chiefly in children and occasionally in adults (Köbner's case), since I have never had the opportunity of observing such an epidemic "pædophlyctis" or febris pemphigosa s. bullosa, s. ampullosa, s. epidemica contagiosa, infantum, etc.

In view of this difference of opinion, or at least uncertainty, regarding the nature and contagiousness of pemphigus acutus, some importance attaches to the finding of fungi, gonidia in large number, and scanty mycelia by my former assistant, Riehl, in a case of pemphigus acutus in a fourteen-year-old boy at my outdoor clinic. Riehl has published an accurate description with illustrations of these structures. Less weight, however, will probably be ascribed to the finding of a diplococcus by Strelitz, Faber, and others, or even to the positive inoculation by Almquist. It is not improbable that in many other similar cases such mould fungi may be the cause of the bullæ of pemphigus and thus furnish an explanation of an "epidemic" spread and "contagion."

Finally, cases of so-called *dermatitis exfoliativa neonatorum* (Ritter) and *epidermolysis bullosa hereditaria* (Köbner) may have been at times mistaken for pemphigus acutus. The latter form, described by Goldscheider, Valentin, and Köbner, it seems should be looked upon solely as a traumatic (urticarial) formation of bullæ in a skin which is congenitally subject to inflammatory irritation, and not as an idiopathic disease. Persons suffering from this affection usually are born with the peculiarity that the slightest pressure by shoes, waist, belt, seams, etc., causes the epidermis to be detached by serous suffusion, thus interfering with their movements, work, etc. In some families this peculiar vulnerability of the skin has been observed as "hereditary" in several members and successive generations, as in Lesser-Blumer's cases, and in the opinion of the former the case of "hereditary pemphigus" cited by Hebra might be classed among them.

LECTURE XX.

3. FORMS OF DERMATITIS (DERMATITIS ESSENTIALIS).

TRUE INFLAMMATIONS OF THE SKIN.

IDENTITY OF ANATOMICAL CHANGES—CLINICAL DIFFERENCES DUE TO DEGREE AND CAUSE OF THE INFLAMMATION—IDIOPATHIC AND SYMPTOMATIC —INFECTIOUS—DERMATITIS AND GANGRENE—DERMATITIS TRAUMATICA, MECHANICA, NEUROPATHICA, TOXICA, A VENENATIS, DIABETICA, ET DYNAMICA— CALORIC FORM: BURNS AND CONGELATIONS.

THE affections belonging to the group of true inflammations of the skin—dermatitides—are characterized, aside from their acute course, by the marked expression of all the manifestations of inflammation as described in general (page 144 et seq.), namely, redness, swelling, increased temperature, infiltration, and pain; and the termination of the inflammation in resolution, or suppuration, or necrobiosis *en masse* (gangrene), or the transition into chronic dermatitis. The more minute histological alterations, likewise enumerated generally above, correspond to the clinical symptoms. In this respect, therefore, the local symptoms of all the diseases belonging in this group also agree in the main. Differences appear, however, both with regard to the local and the concomitant symptoms, according to the seat, form, extent, the morphological and chemical character of the infiltration, and termination of the inflammation, and, finally, according to its special cause.

Accordingly the inflammation may under all circumstances affect either the uppermost layers of the skin only, or the cutis in the entire depth to the subcutaneous cellular tissue; it may manifest itself mainly by redness and serous transudation which terminates in resolution (dermatitis erythematosa), or by a more plastic infiltration of the parenchyma which is apt to lead to suppuration (dermatitis phlegmonosa); or it may furnish a rapidly coagulating fibrinous exudation which determines molecular decomposition (dermatitis diphtheritica); or it may be associated with the necrosis of larger masses of tissue (dermatitis gangrænosa et escharotica, gangrene); or it may be combined with the formation of bullæ by a serous effusion into the epidermal layers (dermatitis bullosa). Moreover, it

may appear circumscribed or diffuse, fixed or progressive; be afebrile or associated with fever and complicated general symptoms.

In all these various forms and degrees the cutaneous inflammation may have arisen, on the one hand, from influences which have acted on the skin directly; or, on the other hand, it may occur as a local phenomenon of some other disease. Accordingly it should be separated into (*a*) an *idiopathic* and (*b*) a *symptomatic* inflammation.

(*a*) IDIOPATHIC INFLAMMATION OF THE SKIN.

This is caused by all the noxæ enumerated above on page 92, which in slight intensity produce merely erythema, whenever their influence has been more marked or effective or the skin acted upon is more irritable. According to the general character of those factors, we enumerate the cutaneous inflammations caused by them as follows:

1. *Dermatitis traumatica s. mechanica*—the inflammation caused by a blow, push, pressure from close-fitting shoes, pressure bandages and slings, the handling of tools, rowing, scratching with the finger nails (excoriations), and traumatic influences of any kind.

This group includes also the congestive inflammations due to the restricted local circulation of the blood.

2. *Dermatitis neuropathica* — the cutaneous inflammations caused directly or indirectly by disturbances of the innervation, of a vaso-motor or trophic nature, of a peripheral or central origin.

3. *Dermatitis a venenatis, dermatitis toxica, et dermatitis a causticis*—the cutaneous inflammation caused by chemical poisons or caustic substances, such as cantharides (vesicants), mezereum, rhus toxicodendron, turpentine, caustic potash, quicklime, caustic pastes in general, and the strong mineral acids. The same group includes the inflammations due to the deposit of sugar in the skin—*dermatitis diabetica.*

4. *Dermatitis dynamica et calorica*—the inflammation caused by excessive dynamic influences and temperature, electricity, lightning stroke, great heat and cold.

The last two groups give rise to forms of inflammation with closely corresponding symptoms.

All these categories of dermatitis, enumerated according to the general and special quality of their cause, present essentially the same local symptoms as I have stated in the beginning of this chapter, and in the main the same guiding points for the prognosis and treatment. Still, some practical and noteworthy differences appear, not only regarding the general factors, such as intensity, extent, importance to the organ affected and the body as a whole, but also, if I may say so, the quality. It seems to me impracticable to pursue the

subject further in this direction, referring the reader to the general pathology of dermatitis and to special surgery. Nevertheless I shall select for discussion some forms of mechanical dermatitis, the forms of neuropathic dermatitis, as well as the cutaneous inflammations due to diabetic and caloric influences, both on account of their eminently practical importance and because their symptoms present an exhaustive scheme for the related affections in general.

As regards the symptoms of cutaneous inflammations caused by mechanical factors of any kind, the general rules of pathology apply. As a rule the inflammation begins with hyperæmia and inflammatory infiltration, and with respect to the extent, duration, and the possible terminations (resolution, suppuration, gangrene) corresponds to the intensity, duration, and special nature of the mechanical factor that caused it, as well as to the more or less favorable conditions furnished by the locality affected, the previous quality of the skin, and the general nutrition.

So, for instance, the pressure of a tight truss pad on the movable skin of the inguinal region may cause inflammation possibly associated with bullæ, while the pressure of the footwear over the firm Achilles tendon may lead to inflammation with gangrene. Pressure upon the sacral region in persons weakened by zymotic and infectious diseases rapidly produces reddening, inflammation, and gangrene—bedsores—as after small-pox, typhus, in articular and gonorrheal rheumatism, while an individual otherwise healthy bears pressure at these points for a long time without harm. With concurrent unfavorable circulatory conditions, either local or due to general causes, such as anæmia, heart disease, atheroma, etc., these injurious influences will the more readily produce inflammation and tissue necrosis. For this reason they are more apt to occur on dependent portions (in bedridden patients, on the sacrum), especially the lower extremities. Thus, on varicose legs, very slight injuries easily lead to inflammation with suppuration, ulcers, and gangrene.

Even a local circulatory disturbance alone may give rise to inflammation and gangrene, aside from gangrene following absolute arrest of circulation in embolism and ligation.

Under this head should be ranged the erysipelatous and phlegmonous inflammations occurring in varicose lower extremities and accompanying neoplastic and ulcerous processes (lupus, syphilis) of these regions.

A peculiar type is formed by the inflammation of the lower extremities appearing in consequence of syphilitic (Heubner) or idiopathic endarteritis obliterans (Winiwarter, Billroth). After months of continuous and lancinating pains, followed by slight inflammatory symptoms of the peripheral parts, the toes become gangrenous. The gangrene gradually progresses centripetally, after inflammation of

the adjoining regions of the skin associated with the same symptoms.

Here would naturally follow the "symmetrical gangrene," so-called since Raynaud's time, which springs from a local asphyxia and whose cause is a vaso-motor disturbance based on a regionary hyperæmia of relaxation and stasis. It occurs in neuropathic, hysterical, anæmic persons, like the neuroparalytic asphyxia (M. Weiss) discussed on page 98.

Essentially the same importance attaches to the inflammations of dependent portions of the body, hence mainly of the lower extremities, which likewise often terminate in gangrene—senile marasmic gangrene—and which occur in persons weakened by exhausting general diseases or rendered marasmic by great age. For this reason they should also be ascribed to mechanical disturbance of the circulation (debility of the heart and vessels).

O. Simon has given the name "multiple cachectic gangrene of the skin" to the formation of gangrenous patches, ranging in size from a pin's head to the size of a dollar or larger, which may be superficial or extend through the underlying tissues to the bone. They occur on portions of the skin previously inflamed or covered with vesicles, in cachectic children of from one to two years. After the crusts are cast off, there result sharply defined losses of substance with the appearance of having been bored out, which cicatrize by granulation. I have repeatedly seen and described such forms, resulting from eczema in children. They often lead to the erroneous diagnosis of syphilis. But I have also observed similar gangrenous destruction of portions of the skin in adults, whose skin had undergone inflammatory infiltration and whose organism had become debilitated; the patches were situated on the shoulder, the abdomen, and the thorax, in psoriasis universalis, lichen ruber universalis, and pityriasis rubra. I look upon this gangrene as the consequence of local capillary thromboses.

The *neuropathic inflammations* of the skin and forms of gangrene are manifold in kind and causation.

We must recognize as an essentially neuritic dermatitis that inflammation of the skin which, though widely known in former times, has been more clearly specified under the name of "glossy skin" since the reports of Weir Mitchell, Morehouse, and Keen upon the nerve lesions and their consequences observed during the American Civil War. Auspitz has proposed the name "liodermia essentialis" for the affection. In the peripheral distribution of an injured, irritated nerve trunk compressed by cicatrices—*e.g.*, the brachial—great pain of a continuous burning character is felt, and a moderate swelling of the hand and fingers develops with a sensation of heat and with reddening. After some months the skin becomes red,

glossy, tense, and thickened; there appear upon it at intervals bullæ (pemphigus neuriticus); groups of vesicles (herpes, zoster neuriticus?) zoster hystericus; later, excoriations, ulcers, limited necroses. There follow subsequently, on the extensor surface of the flexed finger joints, parchment-like thinning, exfoliation, and gangrene of the skin, deformity and degeneration of the nails—in short, trophic disturbances of the most variable form and intensity.

In view of the fact that all the above-named inflammations which spring from mechanical, circulatory, vaso-motor, neuro paralytic, and neuritic causes are initiated by hyperæmia, and in cases of slight intensity of the causal factor may remain in this stage, we have accordingly made mention of them in this sense above (page 92 et seq.), and in part in connection with the anæmias (page 101).

That form of gangrene which Charcot designates as *decubitus acutus* is to be traced to a central disturbance of innervation; generally it occurs in consequence of cerebral abscess and in harmony with its localization—*i.e.*, on the opposite half of the body. The rapidity of its occurrence would exclude local circulatory disturbance and atony of tissue such as obtain in bedsores after typhus, etc., and indicates a purely trophic disturbance.

Mention should also be made of the so-called *spontaneous gangrene* in hysteria, which has been repeatedly observed by neuropathologists, and instances of which have been demonstrated at our clinic by Neumann, Hans Hebra, Doutrelepont, myself, and others. The patients are, without exception, young females, with or without marked evidences of anæmia and hysteria. On a certain portion of the skin, such as the breast or arms, a sudden burning sensation is felt. The patient indicates a spot varying from a nickel to a dollar in size, on which the skin is either slightly reddened and salient or of alabaster whiteness and wheal-like. Within a few hours the integument at this point becomes discolored, blue-black or greenish-brown, sloughy, leathery, as if cauterized with sulphuric acid. The eschar is gradually cast off, and healing usually leaves a hypertrophic cicatrix. In the meantime the same process is repeated on other points, at intervals of days or weeks, with like symptoms of pain, redness, sharply limited gangrene, and cicatrization. The process lasts weeks, months, even years, and then ceases permanently. In Doutrelepont's case, after two years the form of zoster hystericus gangrænosus developed and death ensued with symptoms of tuberculosis (page 242).

Dermatitis diabetica forms a local phenomenon of the dermatoses occurring in glycosuria—*dermatoses diabeticæ*. Among the latter we include asteatosis and anidrosis, pruritus cutaneus, both general and local (vulvæ), chronic urticaria, acne cachecticorum, roseola and erythema, eczema, and dermatitis proper. At times, under the

macerating and irritating influence of the diabetic urine, perhaps also of the development of fungi favored by the latter, there occur on the skin of the prepuce, scrotum, vulva, and perineum phlegmonous inflammation and gangrene, or circumscribed abscesses and furunculosis.

In addition, we may note the occurrence of furuncle, of anthrax which often ends fatally, of diffuse dermatitis and of diabetic gangrene. These cannot be ascribed to the influence of the sugar upon the skin by way of the excretions, but to the sugar deposited in the tissues and there decomposing (acetone?). The inflammation and gangrene affect usually one or other of the toes—not always the peripheral extremity, but, unlike marasmic gangrene, often a circumscribed portion of the sole, of the ball of the toe, of the little toe, or of the dorsum of the foot The gangrene is preceded by inflammation and the formation of bullæ (Marchal de Calvi, Champouillon, Landouzy) and is generally unilateral. At times the gangrene heals.

I have described one form as *gangræna diabetica bullosa serpiginosa*, in which there occurred on the left leg—hence, likewise, unilaterally and far from the periphery—disseminated bullæ on an inflamed base, followed by eschar formation. From such centres the process advanced serpiginously in like manner, healed in the course of months, and death did not occur until the process again broke out over the ankle joint.

Finally, I have described as *dermatitis diabetica papillomatosa* an inflammation of the skin occurring in glycosuria, where in the course of years there develop on an inflamed base nodular excrescences in patches, which partly disintegrate into ulcers with undermined margins—a form on the whole closely resembling lupus.

Caloric inflammation of the skin is divided, according to the two extremes of its causation, into *burn* and *congelation*.

Dermatitis Ambustionis, Burn.

Burn (*combustio*) signifies the inflammation of the skin due to the influence of abnormally high temperature.

Its symptoms, course, and importance vary with the degree of the causative temperature, the duration of its influence, the quality of the medium, and furthermore with the extent of the burn and the individuality of the person affected.

Since the obvious local phenomena furnish a criterion for the accompanying and general symptoms, sequelæ, importance, and the mode of treatment, we divide burns, for practical reasons, into three degrees, which, however, represent merely grades of intensity, not strictly separable forms of the pathological condition.

The *first degree* (dermatitis ambustionis erythematosa) is charac-

terized by an even, diffuse redness which does not entirely disappear under the pressure of the finger, together with moderate swelling of the skin, extending as far as the increased temperature has acted.

The redness, at first vivid, then darker and shading into bluish or brownish red, is accordingly, as a rule, sharply limited. When dispelled by pressure with the finger it gives place to a yellowish tinge. It is associated with marked burning sensations and, when of considerable extent and in young or irritable persons, with moderate fever. This is the picture presented after scalding of the skin with water at a temperature of 100°–133° F., or by persons who were exposed to the hot summer's sun for several hours while swimming, rowing, marching, or whose face was slightly singed by a flame or exposed to strong radiant heat.

The anatomical effect of such moderate degrees of heat consists in an immediate hyperæmia of the smallest cutaneous vessels, followed by paresis and passive congestion. This is most clearly shown by erythema solare, the redness being exactly limited by the line where the neighboring skin was protected, as, for instance, by bathing-trunks. The swelling and yellowish tinge are the expression of a moderate exudation.

With the decrease of the swelling, heat, and pain in the affected skin and cessation of the fever, the vivid red changes in a few days into brownish red or brown, the epidermis desquamates in dirty-white branny or larger lamellæ, and within from one to three weeks the skin regains its normal appearance; occasionally it may remain somewhat darker for a longer period.

Burn of the *second degree* (dermatitis ambustionis bullosa), caused by scalding with hot or boiling water at 145°–212° F., transitory contact with flame, hot metals, great solar heat, melted sealing-wax, etc., is characterized, besides the symptoms of the first degree, by the appearance of vesicles and bullæ, the effect of a profuse serous exudation into the epidermal layers. The bullæ may develop immediately, or several hours after the action of the high temperature, upon the diffusely reddened skin. The latter may appear otherwise unchanged or else be more intensely affected. The bullæ may be single or multiple, and vary in size up to that of a hen's egg; they are tensely filled, have a yellowish translucence where the skin is thin, or where it is thick, as in the palm of the hand, form merely a firm prominence. At many points the epidermis is completely detached by the amount of the exudation and depends in shreds or appears crowded together or rolled up over larger areas.

Superficial bullæ have for their covering only the uppermost layers of corneous cells, and when pricked at once evacuate their contents completely. Other bullæ affect the entire thickness of the rete. From these the serum exudes only gradually, even after the entire

covering of the bullæ has been cut away with scissors, and the swollen rete cells are exposed in the shape of a yellowish-gray, gelatinous pulp.

In microscopic sections of the bullæ of burns the vessels appear dilated within the widened papillæ at the base and in the upper layer of the corium, the connective-tissue fibres are swollen, the meshes widened ; a moderate number of exudation cells are present in them and in the adventitious vascular space. Within the confines of the bulla the rete cells are turbid, swollen, show indications of nuclear division of an abortive or proliferating kind, and are drawn out into fibres and trabeculæ which are stretched between the cover of the bulla and the basal papillæ or the rete cells still attached to the latter. In this way they form a meshwork whose spaces contain serum, exudation cells, epithelial detritus, and fibrous coagula (Von Biesiadecki, Unna, and others)—conditions peculiar to bullæ of any kind. Parenthetically I may add that the internal processes in the formation of bullæ are always the same, whether they are due to molten sealing-wax, fire, a vesicant (Unna), or any other cause, as in zoster and pemphigus.

The course of the local process is typical. Wherever the cover of the bulla is preserved it dries up, as soon as the inflammation and further exudation have terminated, with its contents to a crust, under which the rete cells formed above the intact papillæ undergo cornification. Otherwise the cover of the bulla is detached mechanically or by the amount of the exudation, and is followed by profuse cell proliferation of the exposed rete, presenting the picture of catarrhal (epithelial) suppuration. The papillæ are laid bare in the shape of red dots, partly undermined by hæmorrhages, in a gray, suppurating network whose meshes are formed by the interpapillary projections of the Malpighian layer.

Gradually, as the inflammation abates, the detachment diminishes and the building up of the new-formed cells, which cornify in the upper layers and have meanwhile proliferated over the papillæ, preponderates. Skinning-over ensues throughout without cicatrization ; only at those points where isolated papillæ have been undermined by hæmorrhages and have undergone necrosis, white scars are left.

Even when of very limited extent, burns of this degree are associated with violent pains in the first stages. When more extensive—*e.g.*, including both hands and arms—high fever may be superadded. During the suppuration complicating inflammations may occur, such as lymphangitis and glandular swellings. The stage in which the papillæ are laid bare over large surfaces, after the pulp of the bullæ is cast off, is exceedingly painful. When the new epithelial covering forms, even before cornification, these troubles are at an end.

When still larger cutaneous surfaces are affected by burns of the

second degree—both hands and feet, forearms and legs, the face, and possibly a portion of the back, for example—or when the patient is young or a child, then we may expect the dangerous complications which more frequently accompany burns of the third degree.

This *third degree* (dermatitis ambustionis escharotica) is caused by the influence of the highest temperatures, as flame, red-hot or molten metal, exploding gas, high-tension steam, or else by boiling and caustic liquids which have acted on the skin for a longer period or possess a higher boiling point.

The characteristic point for burns of this degree is eschar formation—the immediate mortification of the skin. According to the quality of the etiological factor, the skin over the entire extent of the eschar appears as if carbonized, brownish-black, discolored or dried up, leathery, or even apparently unchanged, smooth and white like alabaster; but at all events stiff, hard, or tough to the touch, and always insensitive and lifeless. In the affected portion of the skin every vital activity has ceased—the blood and lymph current and the nutrition. According to the chemical effect, we have to deal, on the one hand, with actual carbonization (*e.g.*, by fire), in which case we may notice in the brown eschar blackish-brown dendritic marks—the carbonized contents of the superficial blood vessels. (When the skin of a cadaver—possibly of a murdered person—is burned this vascular injection is not observable, a decisive fact in forensic medicine [E. Hofmann]). On the other hand, the first effect may be mortification of the tissues by coagulation and decomposition of the protein substances or a change similar to the tanning of leather—*e.g.*, by a fall into a lime-pit—or else the skin is boiled, as in scalding by superheated steam, water, etc. Over some of the white eschars the epidermis is elevated into bullæ or thickened in lamellæ, so that we may think the part to be affected merely by a burn of the second degree. But in two or three days the true condition will be manifest, in that the portion of skin in question becomes discolored and wrinkled, and sharply defined at its margins from the surroundings.

Between the third and the fifth day a suppurating ring appears around the eschar, associated with reactive inflammation of the surrounding parts. The ring widens into a broad furrow and becomes continuous with the base, which meanwhile has likewise undergone suppuration. By this suppuration the eschar is detached in from eight to twelve days. The suppurating wound surface thus exposed is of unequal depth, irregularly pitted—a proof that the eschar formation does not extend equally deep throughout. The superficial eschars of burns protect the underlying tissues for a long time from the influence of the high temperature (Hofmann), and the incinerations of cadavers have proved in general the great difficulty

with which carbonization extends into the depth. In another place I shall discuss the more minute processes in the healing of wounds by suppuration, and shall restrict myself here to the brief enumeration of the readily perceptible *anatomical* symptoms. Profuse granulation occurs at all points, finally followed by skinning-over. The new epidermal investment proceeds mostly from the surrounding epidermis. But new islands of epidermis always appear in the midst of the granulating surface. According to the data thus far deducible from experimental and histological investigations, there are good reasons for the belief that these islands do not originate from wandering cells and connective-tissue corpuscles emigrated from the corium or the marginal epithelia, as intimated by Biesiadecki and Pagenstecher, but from preformed epithelium—namely, from remains of the rete cones which have persisted at those points where the eschar formation did not extend too deep. To this latter point I have called particular attention.

Therefore the result of the healing is new-formed connective tissue devoid of papillæ, hairs, and follicles—a cicatrix. The latter, even at the time of its formation, no longer corresponds exactly with the size and form of the original eschar of the burn, because during the granulation the underlying tissue and the surrounding parts approach each other (Billroth) ; this is still less the case later on, since the recent cicatricial tissue contracts subsequently. The cicatrix becomes more irregular, jagged, radiating, contracted, tumid, ridgy, and reticular in proportion to the greater loss of substance and the more protracted course of healing.

Carbonization and eschar formation may also here and there affect the cutis in its entire depth, with all the underlying tissue, including the bone. If this is the case to a great extent, however, we are no longer dealing with a patient—*i.e.*, a living subject—for the person surely must have died long before that time, either of suffocation or of shock.

So far I have described the anatomical symptoms, so to speak, of burns, those produced immediately by the influence of the high temperature upon the skin, and those which ensue locally according to physiological laws and represent in their entirety the processes of elimination and regeneration. These bear a constant relation to each other. To this extent the division of burns into definite degrees has a positive basis. But these local symptoms do not complete the full picture of burn ; for they are associated with very manifold grave complications affecting the body as a whole which derive their importance, not alone from the degree of the anatomical change in the skin, but predominantly from its superficial extent. For instance, they may be lacking altogether in burn of the third degree when very limited, yet may manifest themselves when the burn, in an

anatomical sense, is merely of the first and second degree, but affects a large portion of the common integument.

Let us take an average case, such as is presented when the clothing of a person has been ignited by burning alcohol, petroleum, gas, etc., the flame, as usual, immediately striking upward so as to affect largely the face and arms. Let us assume, furthermore, that the fire has been smothered in from one to three minutes by persons present. Ordinarily the following picture is presented one or two hours after the catastrophe. The hair over the face and head is singed ; burns are present over the hands and forearms, here and there about the upper arms, the face, the neck and clavicular region, the nucha, the upper dorsal region, and the lower extremities. At points where the clothes fit closely or bands constrict, the skin is least injured by a rapid flame, as for instance under the corset, around the waist, and under the garters.

The greater portion of the injury is of the first and second degree ; only limited areas about the face, the chest, generally also the back, exhibit brown carbonization, or a white eschar of the epidermis which has been detached by the exudation or mechanically by attempts to quench the fire. Hence burn of the third degree is either lacking or present to a very limited extent.

The course, then, is the following : The patient, who during and immediately after the accident was excited to the highest degree and acted almost insanely, screaming and lamenting, becomes quiet as soon as the injuries are skilfully dressed. He bears the burning sensations without complaint or at most emits faint groans or whines. Otherwise he is entirely rational and in possession of his mental powers. On being questioned he relates the details of the accident and gives the minutest particulars about everything. As a rule he has not urinated since. On introducing the catheter there is generally not a trace of urine ; occasionally a few drops are obtained which are albuminous or, more rarely, bloody. After five or six hours we notice from time to time yawning and deep sighs ; the eyelids are kept closed. On being addressed the patient looks up and still gives correct answers, but a certain apathy is unmistakably present. Then there will be often deep inspiration and eructations or hiccough. This is a bad sign. Soon there will be vomiting of remnants of food, bilious fluid, and, rarely, even blood. Hebra has stated that on opening different veins he failed to obtain a stream of blood. My own venesections produced a vigorous flow of blood. Now there rapidly follow restlessness, confusion, jactitation, clonic convulsions, opisthotonos, and absolute insensibility. Noisy delirium gives place to quiet sopor, or the latter immediately follows the former apathy. In the course of these symptoms, with rapid, shallow respiration and frequent, vanishing pulse, in the midst of a scream-

ing and noisy spell, or else in quiet sopor, death ensues in the course of eighteen to twenty-four or forty-eight hours. Sometimes this is preceded by hæmorrhages from the stomach and bladder. I have seen very few patients recover in whom ischuria was present or singultus and vomiting occurred.

Even the deep sighs and the repeated eructations are, in my opinion, ominous signs. Still, these first symptoms may be relieved. I have witnessed this once in a woman toward the end of the second day, and, as diuresis had reappeared and the vomiting had ceased, thought the patient was saved. But, after two days of well-being, at the end of the fourth day the whole series of the above-named symptoms recurred in rapid succession, and within a few hours all was over. This group of symptoms may even be observed after the lapse of one week.

At the autopsy we sometimes find ulcers in the duodenum (corrosive ulcers, according to Klebs and Hofmann); hæmorrhagic erosions on the mucous membrane of the stomach and intestines; granular degeneration of the vessel walls, muscles, and parenchymatous organs; hyperæmia of the meninges; even at a very early period nephritis (Wertheim); the blood mostly coagulated; but otherwise there are usually no alterations that could be looked upon as the actual cause of death.

The whole group of symptoms of this period which immediately follows the injury, the rapid course, and the early fatal termination make the impression of a general intoxication. It is clear that the lesion of the cutaneous organ does not cause these symptoms by way of an inflammation, for in these early stages inflammation and suppuration are so far barely perceptible. Hence attempts have been made to explain this enigmatical, immediate consequence of burns in various ways. As animals coated with varnish had been seen to succumb speedily, supposedly by suppression of the cutaneous perspiration over a large area, analogous conclusions have been erroneously drawn for man. But in burns of the second degree such an occurrence has not been demonstrated, and it remains doubtful why the intact two-thirds of the cutaneous surface and the kidneys should not be able to vicariate quickly for the suppressed portion, and why, on the contrary, the kidneys usually cease their function completely.

Following Wertheim in 1865, others (Ponfick in 1877, Von Lesser in 1880, Welti and Silbermann in 1889) have likewise called attention to the presence, in the blood of burned patients, of small corpuscles, which should be looked upon as derivatives of the red corpuscles, because many of the latter appeared shrunken at the same time; and these decomposition products, together with crystallized hæmoglobin, were found in the renal canaliculi and the capil-

laries of the arachnoid (Wertheim), in the spleen and the bone marrow (Ponfick), and in thromboses of the pulmonary capillaries. But that all this should have anything to do with the rapid death is questionable and incomprehensible. Von Lesser interprets the findings in the following manner: Many of the red corpuscles are not only destroyed, but, having been excessively heated in passing through the burnt region, they, though retaining their form, become incapable of functionating in respiration and nutrition, so that the organism becomes acutely oligocythæmic, and cooling and death must ensue. This theory fits many but not all forms of burn.

On the other hand, Hoppe-Seyler directly demonstrated (1881) that, in harmony with Tappeiner's assertion, the red corpuscles neither perish materially in large quantity, nor do they become incapable of functionating, since in experimental investigations they promptly fix and give off oxygen.

An older view, which has recently been revived, is that death after burn is due to the retention in the blood of excreta (carbonate of ammonia), or to poisonous material produced from organic substances decomposed by the heat and carried into the circulation. The anuria so frequently occurring in serious burns, as well as the negative findings in the kidneys, do not substantiate this. Even the temporary appearance of slight albuminuria or "methæmoglobinuria" (Hoppe-Seyler) affords no light; but extensive hæmatoglobinuria, such as is observed after great destruction of red corpuscles in poisoning by chlorate of potassium, pyrogallic acid, etc., has never been seen in burns. Many authors and experimenters (Falk) have demonstrated a rapid fall of the body temperature after extensive burns; while Sonnenburg maintains that the overheating of the blood is directly productive of cardiac paralysis, but that when death occurs slowly the blood pressure sinks in consequence of reflex vascular paralysis. These statements have been entirely disproved by Von Lesser and have also been otherwise interpreted. Neither am I ready to assent to Tappeiner's opinion, though based on experiments, that death from burn is due to the sudden great loss of lymph through the cutaneous bullæ; for in burn by lime and in diffuse burn of the first degree in children, or in carbonization of large areas, no loss of serum can be demonstrated, and still death ensues in the well-known sudden manner.

By purely speculative reasoning Catiano reached the conclusion that the rapid death after burns is caused by hydrocyanic acid, developed by the great heat from the formate of ammonia present upon the skin; while Lustgarten recently put forth the assumption that a poison resembling muscarin was produced under the white eschar.

I have long since pointed out that the whole group of symptoms associated with "death from burn" forces us to the assumption that

a gradual intoxication is being effected. If I do not advocate this view as quite convincing, it is not because the demonstration of the supposed toxic substance has thus far not been furnished, but in view of many important facts gathered from my uncommonly large number of observations. Foremost among them is the experience that the above-described group of symptoms ensues almost immediately after extensive burns of the third degree, death following in from four to six hours. Furthermore, in children, after scalds barely to the second degree and not extensive, hardly as large as the palm of the hand, I have observed eclampsia and death on the second day.

In view of my entire experience and the deductions from experimental pathology, I still adhere to the view that nervous shock, though not the exclusive cause, plays the greatest part. For I have witnessed an identical course even in cases where the temperature causing the burn was not high and in death from every variety of burn, scald, and caustic; and all the proposals based upon the respective theories—*i.e.*, transfusion, intravenous injection of saline solution, atropine (Lustgarten)—for the prevention of death from burns have thus far remained ineffectual.

Should the patient survive this first stage, then the cutaneous lesions occupy the foreground in the symptoms, and the further course develops in the above-described manner in accordance with the general laws governing inflammation, suppuration, detachment of eschars, granulation, and cutaneous regeneration. Still, even during the first or second week, the manifestation of intoxication may appear abnormally and death ensue by eclampsia or gradual heart failure. Aside from this, an unfavorable result can occur only by such general surgical accidents as are met in all suppurative processes—namely, erysipelas, pyæmia, exhaustion, pneumonia, and Bright's disease. In extensive burns sudden death by rapid collapse occurs rather frequently in the second and third weeks—that is, at the time when suppuration and granulation are in active progress.

The immediate *prognosis* in burns, after what has been stated, depends, in the first place, upon the intensity and the extent of the local lesion. It may be stated as generally favorable in burn of the first and second degree, but is always doubtful if the latter is very extensive or affects a delicate patient (nursling). Burn of the third degree is always of serious importance, even if of small extent, in young patients, and is almost invariably fatal when it affects one-third of the entire integument, even if mingled with lesions of the second degree. If death do not ensue as the immediate consequence of the injury, the further course and the material sequelæ (duration, disturbance of avocation by impeded function of the fingers, contrac-

tion of the eyelids, etc.) are largely influenced by the greater or lesser depth of the eschar formation, the region affected, the individuality, and, we may well add, the treatment.

The *treatment* of burns is to be first directed to the alleviation of the violent pains. In the erythematous degree we may restrict ourselves to dusting with starch, cold water applications, lead water, and, if of slight extent, painting with collodion, etc. After the involution of the inflammation no further treatment is required for the desquamation.

In burn of the second degree the first task is to relieve the sensation of tension by pricking the bullæ at their most dependent points, which will favor the escape of the serum, aided by gentle pressure with pledgets of charpie dipped in face powder. It is desirable, however, to preserve the cover of the bullæ, because it furnishes the best protection to the bared papillæ, and, moreover, because under it skinning-over is generally effected without suppuration.

In extensive burn of any degree, however, especially in the presence of bullæ and eschars, contact with the air is exceedingly painful, particularly where the epidermis is detached ; hence efforts have always been directed toward excluding the injured portions as much as possible by mild and well-adhering dressings. For burns of limited extent, olive oil, albumen, linseed oil with lime water (equal parts), ichthyol (two per cent), thiol (five to twenty per cent), resorcin, etc., on pieces of linen, gauze, or cotton, are to be recommended as dressings. These are left undisturbed during the first few days, lest by their removal the covers of the bullæ be torn off, and their drying is prevented by frequent moistening with oil carefully selected and by impermeable dressings (oil-silk). Outside of these, cold compresses may be applied in addition when the cool sensation is agreeable to the patient.

At different times other protective dressings have also been recommended, which act like the carron oil, although their authors have ascribed to them the most surprising effect, the assertion being made that they prevent suppuration and the formation of cicatrices. Thus, Nitsche recommended painting the lesions with several coats of varnish containing five per cent of salicylic acid, and then to apply the dressings. Ed. Busch advised the use of well-oiled gutta-percha paper ; Johnston, carbonate of soda dissolved in camphor water ; Herzenstein and Troizki, soda ; still others, subnitrate of bismuth with equal parts of starch as a dusting powder, burnt well-sand, also ichthyol, sozoiodol, thiol, etc., in solution of two per cent or stronger.

No matter which of these or other similar dressings is employed (iodoform dressing may be allowed to remain longer), it must be removed if, in the course of three to five days, suppuration has set in

underneath, so as to prevent decomposition of the secretion, and thereafter be frequently changed. Of course this gives the patient great pain, and, in case the lesions are numerous, causes much trouble to the attendants.

An advantage which cannot be overestimated, both regarding the points just mentioned and the entire subsequent treatment, is offered by the water bed introduced by Hebra. This consists of a roomy zinc tub placed in a bed frame. Within it an oblong iron frame, across which webbing is stretched, is kept suspended by chains coiled around two rollers, one at the head, the other at the foot of the bed. The frame has a head section and a body section. The former can be set at different heights by means of a toothed rack, and the entire frame can be wound up or down by a crank and gear wheel. Upon the stretched webbing of the frame a mattress or blanket with sheets is placed, and upon this the patient. The tub is filled in the usual manner, and then the patient with the suspended bed is let down into the water. (He is lifted from the water to attend to the calls of nature.)

At first the patient finds the water too warm, therefore the temperature should be between 88° and 91° F. This causes a chilly feeling, and the bath is quickly raised to 104° to 109° F. Then he feels quite comfortable; the pains are almost entirely relieved. But the water bed affords no protection against the first symptoms of intoxication and the acute fatal course. The patients after extensive burns die here as readily as elsewhere, but they are at least relieved of their pain.

On the other hand, the continuous bath is a real remedy and a positive benefit to the patient and the attendants during the entire period of suppuration. Think of it—while such patients in bed can never be kept sufficiently clean because the great number and extent of the suppurating wounds require too much time for dressing, and withal the lifting and turning are most painful to the patients, adhesion to the sheets and forcible detachment are almost unavoidable; here a fresh hæmorrhage, there encapsulation and decomposition of the secretion; the fever steadily kept up, danger of sepsis ever present, nervous excitement associated with every change of dressings—all the trouble and torture are done away with in the water. The patient lies and moves at will, he sleeps and eats, occupies himself, when free from fever, according to his inclination, and the wounds are continually covered, always clean, and granulate beautifully, often so luxuriantly that they must be restricted by well-known means. Accordingly Hebra's water bed is at first and subsequently the best protective against pain, and during the suppurative stage even an unequalled direct remedy: for in the water the eschars are cast off more rapidly than out of it; encapsulation

and decomposition of the pus are hardly possible ; the danger of sepsis and erysipelas is avoided ; the fever ceases at once, sleep and appetite return, and thus the system is enabled to make up the loss caused by the extensive suppuration. Lastly, in the continuous bath granulation and healing run an exceptionally favorable course, owing to the prevention of the subjective and objective troubles and dangers otherwise associated with them.

I shall not enter upon the theoretical data, resulting from the physiological experiment, regarding the condition of the body in the bath. It is of more importance here to emphasize the facts, first, that all patients affected with extensive loss of epidermis (such as burn and pemphigus foliaceus), gangrene, or suppurating wounds rapidly lose their fever, recover their appetite and sleep, and that the healing of the wounds ensues quickly ; second, that, according to the experience gained at our clinic, patients may be kept continuously in the water for more than a year (in one case as long as three hundred and eighty-five days and nights) with no other consequence than their recovery.

In private practice it is best to rig up as a water bed a long bath tub, which should be raised, and in which blankets and horsehair pillows are placed. The water should always be tempered to suit the patient, and renewed two or three times a day, according to requirements.

In treatment outside the bath, the loosened eschars should be successively removed and the suppurating wounds treated according to special surgical rules ; they are to be covered with liniment, simple ointment, cerates with or without the addition of oxide of zinc, carbonate of lead, alum, carbolized oil or paste, opiates, powdered iodoform, iodoform gauze, sozoiodol, aristol, etc , and assiduously cleansed.

The treatment of combustion wounds with iodoform, which I advocated years ago (see the second German edition of this book, page 364), has been specially recommended by some authors (Altschül, Mosetig, and others), first because of its analgesic, second its aseptic effect. I have not noticed that it alleviates the pain more effectually than the other well-known dressings.

As regards the second point, it would indeed be an immense advantage if iodoform dressings could prevent suppuration in combustion wounds, but this is an error. Whenever the anatomical lesion is such that not only the epidermis is detached but the corium forms an eschar, casting off of the necrosis and healing without suppuration and granulation cannot be prevented because they are physiological. The same remark applies to all the other drugs to which such phenomenal effects have been ascribed, as Bidder recently did to thiol. On the other hand, a serious drawback of the iodoform

treatment of combustion wounds is the gradual occurrence of iodoform intoxication, which manifests itself by restlessness, accelerated pulse, jactitation, delirium, and collapse. This is apt to occur even when the local application is very limited—*e.g.*, in burn of both hands—and the more readily when largely used over the trunk. Having observed cases in which the threatening symptoms disappeared when the iodoform was at once removed at my instigation and the patients placed in an atmosphere free from iodoform, and others which had gone too far to be saved, I must emphatically warn against the continuous iodoform treatment of combustion wounds.

Moreover, the process of granulation can be more effectually controlled, with a view to obtain smooth cicatrices, under other methods of treatment than under iodoform dressings.

If, after the eschars have been cast off, the granulations become too exuberant, they must be kept down with the caustic pencil, or a daily painting with a solution of silver nitrate (1 : 1 aq. dest.), or a dressing with charpie moistened with the same or a weaker solution, or a caustic ointment (ung. emoll. 50.0, argenti nitrat. 0.15 to 0.50). By these measures, or by energetic cauterization if necessary, repeated daily or every other day, the granulations are to be kept at their level. These procedures, formerly so painful, have now lost nearly all their terror, since the wound surfaces can be previously anæsthetized by painting with a five-per-cent cocaine solution. In this way alone can we secure smooth, soft cicatrices which subsequently contract but slightly, and thus only can we prevent contractures over joints or at the neck, blepharophimosis, and similar untoward sequelæ. But, above all, the webbing of the fingers and cutaneous folds in general can be most positively guarded against by daily cauterization until the skinning-over is completed.

Nevertheless the most appropriate treatment cannot prevent the failure of extensive combustion wounds—*e.g.*, affecting an entire extremity or the whole of the back—to heal completely even after two or three years; or the recent cicatrices from tearing again here and there, disintegrating, becoming undermined by hæmorrhages; or, finally, contractures from forming which interfere with movement.

What has been stated regarding the symptoms, importance, sequelæ, prognosis, and treatment of burns proper, applies in general also to analogous lesions, really cauterizations, due, for example, to spilled sulphuric acid, a fall into a lime-pit, etc.

In lightning stroke we find on the skin irregular red spots, or brown and discolored dendritic marks, as figured by Schefzik, which may correspond to vessels or nerves; or else there may be no trace of a lesion.

I can dispose more briefly of the disease of the skin due to abnormally low temperatures, which is known as

Congelation, Dermatitis Congelationis (Frost-bite).

Under the long-continued influence of extremely low temperature, but in predisposed persons even at a temperature of 9° to 11° above the freezing point, the portions of skin exposed to it undergo changes which, like those of burn, can likewise be divided into three degrees : dermatitis congelationis erythematosa, bullosa, et escharotica.

The *erythematous form* generally appears as the well-known frost-bites, *perniones.* They are chiefly located on the hands and feet, more rarely on the nose, cheeks, and ears. They become apparent only when the affected portions of the skin, after prolonged exposure to cold, become warm again ; hence mostly in the evening in the heat of the room, where they torment the patient for several hours by stinging pain and intense itching. They represent raised spots ranging in size from a thumb nail to a dollar, peripherally bright red, in the centre livid in color. The pain, burning, and itching regularly increase in the evening, while in the morning hours they are at most sensitive to pressure. Under the influence of the cold the capillary vessels of circumscribed regions are at first induced to contract—the place becomes anæmic, cold, and insensitive. But it seems that they are rendered paretic at the same time, for subsequently they dilate excessively, causing symptoms of passive hyperæmia, lividity, and stasis, followed by serous infiltration and indolent inflammation. The latter also leads to the exudation of bloody serum under the epidermis of the perniones, and, after the bullæ burst, to the necrotic disintegration of the upper layers of the corium, in the form of torpid, very indolent ulcers with hæmorrhagic base—pernio ulcerans. These I have sometimes observed to be the starting point of phlebitis and adenitis with violent symptoms of fever.

This form at the same time represents the *second degree* of congelation, and may occur in all persons whose skin has been long exposed to great cold.

Anæmic persons of both sexes are especially predisposed to frost-bite. Among them they occur even during the cold rainy days of autumn, or possibly in summer when the temperature sinks to from 41° to 43° F.; while well-nourished persons with good calorific powers may expose themselves even to great cold without developing frost bites. For this reason the former class of persons suffer from frost-bites regularly in every cold season for several years—provided their anæmia continues—and their hands are even during the summer of a disfiguring red color because the cutaneous vessels remain paretic.

In congelation of the *third degree* there are either large bullæ filled with bloody serum, whose base consists of tissue with hæmorrhagic suffusion, or else the skin appears merely pale, marbled with

blue spots, and cold, stiff, and insensitive withal. It will be many days or weeks before it becomes evident how far the tissue has undergone mortification. The effect of the cold, moreover, manifests itself very unequally, the mummification here and there extending to the bone and involving it in the necrosis, while at other points only the uppermost cutaneous layers are lost, and at intermediate points deeper necrosis ensues. At the border of the eschars, exfoliative inflammation and suppuration associated with fever become established. The loss of isolated phalanges or of whole limbs is the consequence of such congelations, and not rarely phlebitis, septicæmia, and death, even when the necrotic portion is amputated early.

For these reasons the *prognosis* of congelation of the third degree is very doubtful, although it may have affected merely some toes or fingers. Besides, it should be noted that it will be many days before we can ascertain the extent and depth of the injury, because the reaction occurs late and slowly and because many portions which appear lifeless may revive. As to the latter point, Billroth justly remarks that the vessels largely remain pervious and may again carry blood, and thus serve for the nutrition of the tissues wherever they have not been decomposed by the direct freezing of their watery constituents. But the patency of the vessels at the same time augments the danger of septicæmia, since the restored blood current carries along decomposed particles of tissue.

In the *treatment* of congelatio escharotica, therefore, we are at the start rather handicapped. We attempt by friction with snow to revive the frozen parts gradually and render them pervious to the circulation. The necessity for partial or total amputation of the frozen parts will become manifest in the future, in accordance with special surgical experience. In this respect different opinions have been expressed. While Billroth, in accordance with his experience, urged the earliest possible amputation, Dumreicher and Dittel advised that the limitation of the gangrene should be waited for. In congelation of the toes and fingers I myself have found the expectant plan more advantageous for the patient, since on the average more was preserved than at first seemed possible. In eschar formation extending half-way up the leg I have observed death by septicæmia after late amputation and also in the expectant period.

Where persons are found frozen perfectly stiff, attempts should first be made to restore them to consciousness in a cold room by friction and the generally well-known methods of resuscitation, after which the local congelations will come under consideration.

For erythematous frost-bites there are recommended painting with tincture of iodine, iodized glycerin, collodion, dilute nitric acid, lemon juice, glue, ointments containing basic acetate of lead (5 to 10 : 40), borax with creosote (0.5 : 20 of ointment), camphor (camphor.

rasæ 1, ceræ albæ 40, olei lini 80, bals. peruv. 1.50 parts), Peru balsam, ichthyol, thiol, beer lees, pressure bandage by means of emplastrum lithargyri adustum, friction with snow, hot hand and foot baths—medical and popular remedies in such large number as to indicate their unreliability. Ulcerated spots should be stimulated with the above-mentioned slightly caustic ointments or plasters; bullæ should be opened and their base cauterized with the nitrate of silver pencil.

Of importance is the prophylaxis, in view of which predisposed persons should wear, even when the temperature falls but moderately, warm gloves and comfortable wide shoes, because congelation occurs more readily when the part is kept anæmic by pressure. Besides, in anæmic and chlorotic patients the tendency to frost-bite should be overcome by systematic medication with ferruginous tonics and improvement of the nutrition.

LECTURE XXI.

(*b*) SYMPTOMATIC OR INFECTIOUS INFLAMMATIONS OF THE SKIN.

DIFFUSE ERYTHEMATOUS INFLAMMATION—CAUSES : TOXIC, BACTERIAL INFECTION—ERYSIPELAS : PHLEGMONOUS FORM—PSEUDO-ERYSIPELAS—CIRCUMSCRIBED FORMS : FURUNCLE, ANTHRAX (IDIOPATHIC AND SYMPTOMATIC); ENDEMIC FORMS: ALEPPO BOIL, ZOONOSES: MALIASMUS, CADAVERIC INFECTION PUSTULE, MALIGNANT PUSTULE.

THE symptomatic inflammations of the skin with respect to cause and nature form a marked contrast to the previously described idiopathic inflammations. While the latter represent a direct effect of external or, at least, only locally acting noxæ, and in every particular are proportionate to the mechanical, neurotic, chemical, and dynamic effective intensity and extensity of such noxæ, the symptomatic inflammations of the skin, once set up, assume a course and feature which do not exactly correspond with the underlying cause.

The poisons causing the symptomatic inflammations are not yet exactly known, but are merely estimated according to their general character as poisonous or irritating substances which are directly or indirectly of animal origin, whether derived from the patient's own or another human body or from an animal. It is still very largely an open question whether these substances are non-organized decomposition products of animal tissues or organized elements—micrococci, bacteria, bacteridia—or again the products of secretion or decomposition of the latter, of the nature of ptomaines. Be this as it may, it is supposed that when they are brought in contact with a raw surface of the skin or in some other way carried into the lymphatic circulation, they cause an inflammation which may spread variously as a cutaneous affection and run its course as such, or else may implicate the general organism, as, for instance, cadaveric, anthrax, snake, and glanders poisons. It may also be that these poisons reach the circulation from a diseased focus present in the body—for instance, a retrouterine or cutaneous accumulation of pus, a pustule of the skin, or some undemonstrable source—and partly inflame the skin, partly involve the body in general, as in erysipelas,

furuncle, and anthrax. In view of all this, the symptomatic inflammations of the skin may be considered both as local and as general infectious diseases. But, as has been stated, this duality cannot be established for all cases—for instance, not for every furuncle.

According to their clinical character, the symptomatic inflammations of the skin appear either in a diffuse form, like erysipelas and pseudo-erysipelas, the former with a more serous, the latter with a more plastic exudate; or else in circumscribed foci, like furuncle, anthrax, malignant pustule, glanders, and Aleppo boil. With regard to the intensity of the alteration of tissue set up, we divide the inflammations placed under this heading into the erythematous and phlegmonous.

As an example of *erythematous inflammation* there may be described

ERYSIPELAS,

St. Anthony's fire. rose—an inflammation of the skin usually beginning and associated with fever, which appears as a diffuse, painful reddening and swelling of the integument, and after an acute course usually terminates with desquamation.

The symptoms and course of the disease show an unmistakable analogy with those of the acute exanthemata. From twelve to twenty-four hours before the onset, erysipelas is usually preceded by a rigor followed by fever, gastric and general symptoms, and vomiting, like those peculiar to the so-called eruptive fever of the acute exanthemata.

The erysipelatous inflammation appears on a circumscribed portion of the skin about the size of a dollar, with sensations of tension and moderate pain or itching. It presents a red, salient spot with irregular, usually steep margins, within which the skin is smooth and glossy—erysipelas glabrum—hot, firm, and painful on pressure; when the redness yields to pressure it gives place to a yellowish tinge.

In the course of the following days the inflammation spreads rather uniformly to the neighboring skin, so that the spot in two or three days will reach the size of the palm of the hand or even twice that size. In moderate cases the process reaches its height in such an extension and within a few days, about three to five. It then remains stationary—erysipelas fixum. The fever associated with the disease, which exacerbates at night with temperatures of from 102° to 106° F., the sleeplessness, dizziness, slight delirium, dryness of the tongue. etc., disappear and the cutaneous inflammation gradually subsides. The bright red of the erysipelatous spot changes into blue, bluish red, or pale brown; the turgescence and brawny feeling of the affected skin yield slowly; the brown-colored epidermis desqua-

mates in scales or lamellæ, and the skin appears normal. Appetite and sleep return gradually during the involution of the disease. According to the extent of the process the course may occupy from one to two weeks.

From this frequently observed type there are deviations in a favorable and unfavorable sense and in different directions. Thus, the entire symptom-complex may be milder; the cutaneous affection, being very small, barely the size of a dollar, remains stationary and runs its course without fever or general symptoms. Even then the local affection may persist for many days and undergo slow involution.

Furthermore, there may be variations in the intensity and extent of the inflammation. Thus, the former may be so great that the serous infiltration within the epidermal layer leads to the formation of vesicles and bullæ rising here and there above the tensely swollen skin—erysipelas vesiculosum et bullosum. By the suppuration of the contents of the vesicles this form changes into erysipelas pustulosum, and, after drying, into erysipelas crustosum. In the corium, too, the infiltration may become very intense, and, through the mechanical pressure and compression of the vessels, lead to gangrene, as occasionally on the eyelids, the penis and scrotum, and the sacrum; or else to purulent cellular disintegration of the vessels and the formation of furuncles and abscesses.

Of more importance for the general course than these peculiarities of the local phenomena is the so-called migration of erysipelas—*erysipelas migrans*. While in the normal course the inflammation, after reaching a moderate extent, becomes fixed and soon undergoes general involution, in the migrating form it advances continuously in one or more directions, and in the opposite direction involution follows at the same rate. This progression always ensues toward the swollen, steep margins, either by their uniform advance or by jagged processes which, as Pfleger has shown, follow Langer's cleavage lines of the skin; while involution takes place from the flat, obliterated margins. Progressing in this way, erysipelas may migrate over very large regions of the skin, and even over the entire surface; or, returning to its place of origin, recommence a second cycle. Also, in the midst of the healed territory new erysipelatous centres may arise, and isolated diseased patches may be joined by streaks and lines of a delicate rosy tint, like those known as symptoms of lymphangitis, and along such streaks the patches may approach each other and subsequently coalesce diffusely. Such a course consumes from four to six weeks, during which the patients become greatly debilitated, partly by the loss of substance induced by the extensive exudation, partly by the fever which all the time keeps pace with the exacerbations of the disease, indicating them either by rise of

temperature and frequent pulse or by distinct rigors. Chronic exanthemata, like syphilis, psoriasis, and lupus, undergo involution in the course of intense erysipelas, as they do in other febrile diseases ("erysipèle salutaire").

In an equal degree as the erysipelas involves larger cutaneous surfaces and prolongs its duration, increase the opportunities for and dangers from *complications*. among which may be noted delirium, sopor, œdema of the brain, meningitis, œdema of the lungs, pneumonia, œdema glottidis, pleurisy, endocarditis, pericarditis, metastatic inflammation and suppuration of the joints, fibrous membranes, the skin and the subcutaneous cellular tissue, and pyæmic processes in general.

According to its *seat*, facial erysipelas is most frequently observed This usually starts from some part of the nose or cheek, occasionally undoubtedly from the nasal or pharyngeal mucous membrane, in which cases the process may be termed an erysipelas of the mucous membrane. The whole face may be affected successively or simultaneously. The features are then enormously swollen ; the lips are tumid and projecting; abundant saliva is discharged from the mouth ; the tongue is brownish red, dry, and fissured ; the pharyngeal and palatal mucous membrane appears as though varnished, dry and glistening ; the eyelids are œdematous, closed, and sometimes gangrenous ; the auricles are thick, prominent, the auditory canal nearly closed by the swelling ; here and there the skin is covered with bullæ and crusts. The patient becomes delirious during the high temperature (106° F.), has a full, rapid pulse, or at other times presents symptoms of depression—slow pulse, apathy, or even sopor. The cerebral symptoms are especially apt to become serious while the erysipelas affects the hairy scalp, where it is not visible until it has passed beyond to the nucha and shoulder. On the hairy scalp the disease manifests itself more by the great sensitiveness to the touch, because the hairs hide the morbid process. After the inflammation has run its course the hair is lost in large quantities ; even total baldness may ensue rapidly. This is explained by the fact that exudation takes place into the follicles (Haight), by which the root sheaths are detached from the basement membrane, and by the succeeding seborrhœa.

Facial erysipelas may end fatally during its course by the complications mentioned, œdema of the lungs or of the brain, especially in aged persons and drunkards ; but in the majority of cases it terminates in recovery.

Some persons are attacked repeatedly by facial erysipelas in the course of several years. Such patients usually develop a permanent thickening and brawniness of the skin of the cheek—elephantiasis, pachydermia.

Erysipelas may start from any other part of the body: from wounds; from inflamed and suppurating foci of all kinds; in the new-born from the inflamed umbilicus—erysipelas umbilici—often with fatal termination; from the vaccination wound—vaccine erysipelas; from the genitals of puerperæ—erysipelas puerperalis; from the extremities of persons affected with varices, excoriations, and pustules.

The last-named exciting causes of erysipelas, as well as the frequency of one or the other localizations, are connected with its special *etiology*.

With Hebra, Billroth, and most of the later pathologists, I was and still am convinced that erysipelas never arises otherwise than from the absorption of some phlogogenic and pyrogenic substances (Billroth) into the lymph vessels and juice canals of the skin. Evidence in favor of this view is furnished by the above-mentioned red streaks passing along the vessels in this disease, so that I am inclined to look upon erysipelas as a lymphangitis capillaris cutis; also by the fact that in most cases it is possible to demonstrate an inflammatory or purulent focus which is apt to produce such pyrogenic substances (organic decomposition products in general), and from which the lymphangitis and the erysipelas have started—whether it be an abscess of the skin, caries of a rib, or a collection of pus in Douglas' cul-de-sac. Another proof is furnished by the experience that as a rule the inflammation undergoes immediate involution when, by detaching the crusts from the skin or opening the abscess, egress is given to the pus, whose encapsulation and decomposition have given rise to the erysipelas in consequence of a kind of auto-infection.

As regards facial erysipelas, however, the opinion largely prevails that it might actually arise from "taking cold." I wish to lay stress upon the fact that the same remarks apply to its cause as to erysipelas of an extremity. Its source must be sought for and will be found in caries of a tooth (erysipelas odontalgicum), in eczema, lupus, scrofula, syphilis of the nasal mucous membrane, in a retropharyngeal abscess, etc. It is generally assumed that a person who has once suffered from facial erysipelas is particularly predisposed to a relapse. This is correct; yet not because the patient takes cold more easily, but because the exciting causes are such as are chronic in nature (scrofulous rhinitis, eczema and lupus nasi), and hence repeatedly furnish the material for the erysipelas. Rational therapeutics, too, keeps these conditions properly in view.

Still, this does not explain the etiology of erysipelas for all cases. At certain times, with us particularly in spring and autumn, erysipelas occurs more frequently, both in healthy persons and especially in hospital patients with wounds (traumatic erysipelas, erysipelas

nosocomiale, epidemic erysipelas), and it was assumed that this (septic) erysipelas was even directly transmissible, either by means of a volatile substance (Volkmann) or by organic germs, bacteria, micrococci (Lukomsky, Orth, Ponfick, Zuelzer, and others). Injections of erysipelatous products into animals have proved very poisonous. But this fact was far from demonstrating the poison to be organized, as it might have been of a chemical nature.

It is only since R. Koch and later Fehleisen have not only demonstrated the coccus of erysipelas in large masses in the lymphatics (never in the blood vessels and in the blood) of the erysipelatous patch, but have also made pure cultures, that we may consider it the specific erysipelas germ. This is especially the case since success has followed the efforts to produce in man, by means of the pure cultures of cocci furnished by Fehleisen, typical and even fatal erysipelas beginning with rigor and fever (Fehleisen, Janicke, and Neisser), partly experimentally, partly for quasi-curative purposes in order to effect the absorption of incurable ulcers (cancer) by an erysipelas artificially induced ("erysipèle salutaire").

However, I cannot admit that it has been proved beyond question that this streptococcus alone, and not perhaps also some other microbe or some poisonous substance which is not organized, like those mentioned above, may cause erysipelas, or, in fact, underlies erysipelas of different forms and conditions. I base this reserve on my experience in various cutaneous diseases (apart from the peculiar effect of snake poison), emphasizing especially the fact of universal application, that every purulent focus, whether small or large, whether subepidermal (pustule) or cutaneous and subcutaneous (abscess), may lead to erysipelas so long as the pus is encapsulated, and that the erysipelas undergoes immediate involution as soon as the external evacuation of the pus has been effected. I must refrain, however, from entering here more fully upon this theory.

Besides the general causes mentioned, there are, as stated, certain exciting causes and special predisposing factors. Among these must be emphasized the puerperium; puerperæ are very liable to erysipelas, since the numerous surfaces of their sexual apparatus furnish the most favorable points of ingress both for the septic substances derived from their own body and for those coming from without. With good reason, therefore, the practitioner dreads to have the puerpera attended by such midwives and nurses as have been in contact with erysipelas patients. Experience has shown that a puerperal process is very easily caused in this way. A large fraction of the "puerperal fevers" and "puerperal processes" has long been recognized as an infectious erysipelas or septic phlegmon, because there appear as symptoms, either with or without cutaneous erysipelas, erysipelatous inflammation of the vagina, uterus, and perito-

neum ("erysipelas grave internum." Virchow), with the well-known possible terminations in sloughing and frequently death.

In nurslings the erysipelas often starts from the umbilicus (erysipelas umbilici); in the vaccinated, from the point of inoculation (vaccine erysipelas).

The *anatomical* alteration of the skin in erysipelas consists essentially in an infiltration of the entire cutis—epidermis, corium, and subcutaneous cellular tissue—with an exudation which is chiefly serous. But the latter is by no means devoid of cells, although it does not contain as many exudation cells as the plastic exudate of the phlegmonous inflammation of the skin. Imbibition, opacity, division of nuclei, distortion and stretching of the rete cells into a meshwork (as in the formation of bullæ) are the effects of the exudation on the epidermis ; on the corium, absorption of the connective-tissue fibrils and dilatation of the juice spaces, while numerous exudation cells surround the dilated blood vessels. In the lymph vessels and spaces are found the dense accumulations of the above-mentioned coccus. Fehleisen describes this as follows : "The lymph vessels of the skin, as well as those of the subcutaneous fatty tissue, but especially those of the most superficial layer of the corium, are found filled with a micrococcus which forms chains. In places where the development of micrococci was particularly abundant they lie also in the lymph spaces and small juice canals of the skin. But they never penetrate into the blood vessels. In the most recently attacked portions of the skin, where no macroscopical alteration can as yet be recognized, no reaction by the tissues can be demonstrated. In the neighboring zone, however, say at the sharp border of an erysipelas marginatum, the inflammatory alteration begins. The tissue of the cutis appears swollen ; along the lymph vessels filled with micrococci there is a more or less marked infiltration with small cells. Finally, in still older portions of the affected skin can be demonstrated only an infiltration with small cells, but no cocci." Exudation also occurs into the sebaceous glands and hair follicles, which is followed by loosening of the root sheaths, subsequent falling of the hair, and long-continued cell proliferation in the form of seborrhœa. The above-described quality of the exudate and the tissues explains the clinical local symptoms as well as *restitutio ad integrum* after the absorption of the exudation.

The number of cells and the plasticity of the exudation may also rise acutely. Then there will be here and there symptoms of phlegmonous inflammation of the skin, microscopical and macroscopical abscesses, and gangrene.

In erysipelas frequently recurring at the same point, as in the face or on the legs, a portion of the serous exudation is left behind each time. The final result is a chronic œdema having the so-called

lymphatic character (Virchow), which contains a great many exudation cells. In time these develop into connective-tissue corpuscles and unite into fibrillæ (Young). In this way new connective tissue and thickening of the affected skin are produced—pachydermia. Thus, as has been stated, we may find the cheeks, ears, and lips permanently thickened after frequently repeated erysipelas, and this is true also of the leg.

Sometimes a favorable effect may be observed from erysipelas. In its course chronic inflammatory and neoplastic infiltrations, as well as benign and malignant tumors, may undergo involution and even disappear altogether, under which circumstances the erysipelas has gained the epithet of "salutary"—"erysipèle salutaire." The fact that chronic dermatoses, such as eczema, psoriasis, and even syphilides, undergo involution during erysipelas, may be due to the accompanying fever, which under all circumstances may have this effect. But I mean the local effect in so far as the erysipelatous patch and the dermatitis or neoplastic process coincide. A very remarkable case has been reported by Dr. Biedert: In a child aged nine affected with an inoperable sarcoma which filled the entire nasopharyngeal space and threatened to kill the patient by suffocation, the tumor disappeared completely in a short time during an erysipelas of accidental occurrence.

Basing on this experience, Busch, Volkmann, Fehleisen, Janicke, and Neisser—the latter two with pure erysipelas cocci, the others with erysipelatous material—have made inoculations around the malignant tumors and produced an artificial erysipelas. The effect as regards the final result was no more than encouraging; but the last-named authors, in particular, state that at the post-mortem they found histologically involution of the malignant neoplasm at the points where the erysipelas cocci had immigrated in large numbers.

The *diagnosis* of erysipelas is not difficult when the characteristics described are borne in mind. Mistakes are possible in the presence of erythema, dermatitis phlegmonosa, and with acute eczema, especially the vesicular form.

The *prognosis* in general is favorable. Most cases end in recovery. Of 241 cases of erysipelas observed in the years 1883 and 1884 at the Imperial Royal General Hospital, 32 resulted fatally—*i.e.*, 13.3 per cent. This number includes all forms—of the face, trunk, and extremities, as well as pronounced traumatic erysipelas—and all cases previously suffering from Bright's disease, tuberculosis, senile marasmus, and similar grave conditions. If the cases are properly sifted, the mortality after erysipelas is very much less. During the ten years from 1875 to 1884 there were treated at our dermatological clinic and ward 157 cases of erysipelas (79 males and 78 females), 127 of which were of the face. The mortality was 7, or 5.5

per cent. But all the fatal cases occurred in patients affected with senile marasmus or chronic Bright's disease, excepting two puerperæ. In uncomplicated facial erysipelas the mortality may be stated as from one to two per cent. Patients suffering from senile marasmus, diseases of the heart and blood vessels, drunkards, and puerperæ are chiefly in danger.

As regards erysipelas of the trunk and extremities, the prognosis depends pre-eminently upon the local causative process, and the relative mortality cannot be estimated generally. Still, caution should be exercised in the prognosis under all circumstances, as it is impossible to foretell the extent the process may assume and whether grave complications may not ensue.

I have already stated that facial and migratory erysipelas in general, and especially in drunkards and old persons, may become grave. The danger of erysipelas umbilici I have likewise spoken of. L. Fürth gives a mortality of 57—*i.e.*, 31.14 per cent—in 183 cases of vaccine erysipelas at the Vienna Foundling Asylum during the ten years from 1854 to 1864.

The *treatment* of erysipelas, in view of its general symptoms, is to be conducted in the same way as for all febrile diseases and according to the indications applying to them. Hence excessive heat, delirium, and restlessness will be moderated by cold packs, ice bags, Leiter's coil, etc.; where the exacerbation has a regular type, quinine, salicylate of sodium, and antipyrine will be administered; while nothing need be done where the fever is mild or the course afebrile. Venesection, leeches, or wet cups must be decidedly rejected.

Many have been the efforts to limit the erysipelatous inflammation and prevent its spread by local measures, but in vain. Neither the drawing of a limiting line with a pencil of silver nitrate has acted as a preventive of the spread of the erysipelas, nor can it be confined to its place by painting with collodion, varnish, tincture of iodine, carbolized turpentine, covering the surrounding parts with adhesive plaster [!], etc., or by so-called antiseptic local and internal medication.

Rational therapeutics aims first of all to discover the point of origin of the erysipelas and to render it harmless. In facial erysipelas search must be made for a gumboil, so as to open it if present, but especially the nasal cavity should be inspected, any pustules present opened, crusts and accumulations of pus softened by tampons coated with oil or ointments. I have permanently cured many a case of erysipelas which had relapsed for years, by teaching the patients to prevent the formation of crusts within the nose after the termination of the disease. In like manner we must look elsewhere —as for instance on the legs—for obvious or hidden pus cavities. for periproctitis in erysipelas of the nates, for pustules and abscesses wherever present, and open them by softening the covering crusts

or by operation. By thus removing the opportunity for the renewed absorption of phlogogenic substances, the erysipelatous process will also be most readily limited.

The local inflammation of the skin is either left untreated or covered with dry dressing cotton, ice bladders, or cataplasms (water, acetate of lead, Burow's solution, etc.), according to the disposition of the patient—that is to say, we use whatever is most agreeable or soothing. The application of linen coated with mercurial ointment is in favor for facial erysipelas, but the danger of salivation should not be lost sight of. Hueter, Neudörfer, and others claim to have arrested the spread of the local process by giving from ten to twenty daily subcutaneous injections of one to two per cent carbolic acid solution into the marginal zone of the patch. I am not able to confirm this any more than similar results claimed by others for carbolized water compresses and baths in erysipelas of the legs, or for carbolized oil, etc., in facial erysipelas

In erysipelas of the trunk and extremities simple applications of water and fat are just as effective, provided the pus cavities which are to be looked upon as the starting points of the morbid process have been freed from their crusts, laid open, and evacuated, and a favorable antiphlogistic effect is exerted upon the inflamed erysipelatous surfaces. The same is true for facial erysipelas, and the favorable result is more rarely observed only because the starting points of the inflammation are usually inaccessible (in the nose).

Indeed, basing on a large experience, I must even caution against the use of carbolic acid and iodoform in erysipelas, because the former usually gives rise to carbolic pustules and furuncles, thus furnishing new foci of inflammation and lymphangitis, and the latter directly increases the erysipelatous dermatitis.

The application of mild ointments spread upon linen (simple cerate, zinc or precipitate ointment, 1 : 40), glycerin, vaseline, Lister's paste, ichthyol, acetate of lead, etc., may moderate the tension and can be recommended as suitable, especially at the time of decrustation.

The continuous bath has in our experience proved useful in numerous cases of erysipelas of the trunk and extremities. As a rule, within from twelve to twenty-four hours the process is arrested and undergoes involution with defervescence, most probably in consequence of the favorable effect produced by the continuous bath on the foci of gangrene and pus which form the source of the erysipelas.

Finally, owing to the transmissibility of certain forms of erysipelas, discussed above in the etiology, it is well to recommend as far as possible isolation of the patients and the most conscientious disinfection of the instruments and hands brought in contact with them, in order to prevent the spread of the disease.

The PHLEGMONOUS INFLAMMATIONS of the skin are characterized by intense redness which hardly yields to pressure, heat, painfulness, considerable infiltration and swelling of the affected skin, and the usual termination in purulent disintegration or extensive necrosis (gangrene) of the tissue.

In the diffuse form the process represents the so-called PSEUDO-ERYSIPELAS (phlegmon). Generally inaugurated with rigor and fever, it appears as a firm, painful swelling and redness of the skin, spreading acutely over large surfaces—for instance, one extremity. The symptoms of inflammation may cease in a few days with remission of the fever, leaving brown pigmentation and desquamation. But more frequently there ensues rapidly, within from one to three days, a very extensive purulent breaking-down of the tissue, which manifests itself by increased fever and locally by fluctuation. After opening and evacuation of the ichorous masses of pus, which usually contains an admixture of larger shreds of tissue, there is often observed an enormous destruction of the subcutaneous cellular tissue and the underlying structures, fascia, muscles, denudation or necrosis of the bones, and opening of the joints. In this respect the worst form is phlegmon of the hand, which early jeopardizes the phalangeal bones and joints.

Besides the local consequences there is danger of adenitis by extension, also of pyæmia, icterus, metastases, long-standing cachexia, or rapid death.

The *cause* is probably always an infection which is in general septic, whether from without by an intoxication following inoculation of a wound (excoriation) with cadaveric poison, the matter of variola, puerperal secretion, ichor, and decaying animal substances of any kind ; or from within by an analogous poisoning, by way of the lymph channels, from a purulent focus. Whether and to what extent we have to deal here with specific microbes (streptococcus pyogenes, etc.), and such only, or perhaps with chemical decomposition products of the microbes (ptomaines) or of the albuminous substances of the tissue elements, is still undecided in the present state of our knowledge. In the latter case the effect really represents metastatic phlegmons such as those described after variola vera or to be observed in puerperæ.

To this same class belong the phlegmons caused by snake poison, the sting of the scorpion, etc., which spread from the wound and pass as a local affection or end fatally by general blood poisoning.

As regards the *treatment*, which is essentially surgical, I emphasize only the necessity of making early and deep incisions, even before an accumulation of pus can be demonstrated.

In a circumscribed form, phlegmonous inflammation of the skin

appears in the diseases known as furuncle or anthrax and in the zoönoses—glanders, the pustule of cadaveric infection, and malignant pustule.

FURUNCLE represents a circumscribed, firm, inflammatory nodule of the cutis, in the centre of which necrosis of the tissue usually takes place, the necrotic mass being eliminated in the shape of a plug or core. Its development is marked by circumscribed pain and hardness of the skin. Redness and increased local temperature do not occur until the following day. The swelling, hardness, and redness extend so that the nodule, which is but moderately salient, may reach the size of a hazelnut or walnut. In the case of follicular furuncle which is clearly formed around a hair follicle there is early perceptible a yellow spot pierced by the hair, or, above it, a vesicle filled with blood or serum ; but in the so-called furuncle of the cellular tissue a yellow point is seen only after several days, or else there is merely the prominent bluish-red and thinned skin. Meanwhile purulent disintegration has occurred in the centre, associated with continued pain and a sense of throbbing. After opening, the pains moderate, but do not disappear until, in the course of from eight to ten days. the necrotic mass has been detached by suppuration. This is expelled or removed in the shape of a yellowish-green, tough plug soaked in pus. The cup-like, gaping cavity left behind closes gradually by granulation.

Sometimes the furunculous nodule opens at several points and numerous plugs are detached. The skin then appears honeycombed with holes—furunculus Vespajo. This is really a compound furuncle. In irritable patients fever may accompany the inflammatory and suppurating period.

ANTHRAX (carbuncle), according to its cause, clinical course, and importance, must be divided into two varieties :

1. *Common anthrax* or furuncular anthrax, furunculus vulgaris, also known as anthrax or carbuncle simply. This begins as an infiltration of the skin and subcutaneous cellular tissue similar to a furuncle, but the size of a dollar, the palm of the hand, or larger ; it usually springs from an agglomerated furunculus Vespajo, is very hard, generally very painful, and scarcely movable. It is most frequently situated at the nucha, but occurs also in the face, on the cheek, lip, or on the back in the sacral region. The associated pain and fever are often very intense, and, if located at the nucha and in the face, delirium and sopor may be present. The covering skin becomes necrotic to a variable extent and form, changing to a bluish-black pulp or a leathery dry eschar. When this is cast off there follows further exfoliation of the deeper connective tissue, which is irregularly interspersed with purulent foci, disintegrated shreds of tissue, and thrombosed blood vessels. It is only after the infarcted masses

have all been liquefied and expelled by a limiting suppuration that a red, granulating, often very deep wound is exposed which heals regularly.

Even with this favorable course carbuncle is a serious disease. But it becomes dangerous when the infiltration and gangrene progress and limitation does not ensue, since death may occur from pyæmia or with symptoms of cerebral œdema. Persons of advanced age are always in great jeopardy and the prognosis in this disease must be guarded.

Regarding the *anatomical* conditions of furuncle and anthrax little is definitely known, since the diseases have probably never come under examination before the onset of tissue necrosis. As far as can be learned from the clinical picture and the form and constituents of the expelled plug, the inflammation and mortification usually start from a hair follicle or a sebaceous gland (Billroth) and the adjoining tissue, as Rindfleisch maintains, possibly from the connective-tissue trabecula which passes from the bottom of the hair follicle into the subcutaneous cellular layer (Wertheim). Some believe that a thrombosis of the vessels supplying the base of the follicle precedes the inflammation and tissue necrosis, but this has never been proved. In carbuncle the anatomical relations are still more complicated.

Furuncle and anthrax, however, must be considered in connection pathologically, since both usually occur under similar conditions, and a series of furunculous attacks very often terminates with a carbuncle.

Furuncles occur either sporadically or in large number successively—that is to say, furuncles appear during several months, and even years, with brief interruptions or continuously, on different parts of the body or mainly on certain regions. This is termed furunculosis, a chronic affection which may debilitate the patient by the frequent pains, fever, and suppuration, or endanger life by the intercurrence of an anthrax.

According to their *cause*, we may distinguish idiopathic and symptomatic furuncle and carbuncle. The former arise spontaneously in healthy persons, when they are usually solitary ; or else singly and in successive numbers in consequence of irritation of the skin by repeated douches. cold-water cures, submersion in the water bed, and scratching—as in itching skin diseases. eczema, prurigo, scabies, pediculosis vestimentorum. They may also occur metastatically. furuncles of infection, derived from pustules and purulent foci peculiar to these dermatoses.

Furuncle and anthrax may be called symptomatic when they occur. as experience shows they do, with or after general nutritive disturbances, chronic indigestion, senile marasmus, and diabetes.

The *treatment*, in a direct sense, is almost powerless against the

affections here discussed. The course of a furuncle cannot be altered or cut short either by the application of ice or moist heat or by early incision. Hence it is to be recommended to apply the one or the other, whichever moderates the pains most, according to the sensation of the patient. In the early period cold is usually most grateful; during the suppurative disintegration warm fomentations by cataplasms or indifferent ointments and plasters are preferable. The removal of the necrotic plug before it is completely loosened is absolutely useless.

In anthrax, on the other hand, the indication is to apply ice most energetically and to make early deep incisions—during which the firm tissue creaks—when, according to circumstances, necrotic portions may be cut or scraped out, or various incisions may be made into the infarcted tissue from the periphery and base in order to open as many purulent foci as possible. When disintegration sets in warm fomentations or disinfectant dressings (carbolic paste, trimethylamin) are indicated.

In furunculosis the several nodules are treated symptomatically, like the sporadic furuncles. Besides, efforts should be made to ascertain the underlying general cause and to remove it by appropriate measures—cures at spas, improved nutrition, bitters, soda, iron, thermal waters, etc. As a rule baths do not prove favorable; but often alum and soda baths (about two pounds to the bath) and sublimate baths (℥ iiss. to the bath) have been recommended and found effective. I myself have frequently resorted to similar measures, because furunculosis is an exceedingly troublesome disease which calls for renewed efforts for its cure.

It is most unscientific to treat the furuncles with carbolic acid on principle, which is largely done, because that drug itself gives rise to ever-new outbreaks of furuncle.

2. *True anthrax* (carbunculus verus, charbon, *malignant pustule*) begins with itching and burning as a red, salient spot like an insect-bite. Very soon the epidermis above it is raised to a vesicle by hæmorrhagic serum. The vesicle may dry in the centre, enlarge peripherally, or coalesce with neighboring vesicles. In the meantime an infiltration of the skin has formed at the base; its size ranges from a dollar to the palm of the hand; it projects in the shape of a flattened sphere, is very firm and slightly painful. Soon a hæmorrhagic bulla rises upon it or a portion of the skin changes to a dry, discolored eschar. The latter sinks somewhat below the level, so that the surrounding swelling overtops it like a wall and sometimes overhangs it. Often hæmorrhagic vesicles occur subsequently over this portion. The seat is most commonly the dorsum of the hand, more rarely the face, lip, ala nasi, cheek, or the eyelid, which then swells enormously and is densely infiltrated.

Should the process not terminate fatally in a short time, the greater portion of the infiltrated tissue becomes necrotic, and, when this has been cast off, granulation and cicatrization ensue. Lymphangitis and suppurating axillary adenitis or sloughing of the pectoral muscles are of frequent occurrence. The *prognosis* is favorable only when the affection is locally limited and no grave symptoms present; otherwise it is very doubtful, for death follows but too frequently in a short time under pyæmic general symptoms.

Anthrax also appears at times in the form of the so-called external œdema of anthrax (malignant œdema), in which gelatinous infiltration predominates and which is misunderstood. Extensive dark-gray to grayish-black eschars often arise suddenly on the œdematous nodule (Bollinger).

As a third form W. Koch enumerates embolic cutaneous anthrax, which has been described by Weigert-Waldeyer, and which, when observed in a man dead from typical anthrax of the lung and intestine, appeared in the form of punctiform, lentil-sized and larger hæmorrhagic spots and nodules, and of hæmorrhagic umbilicated vesicles seated on the level or on nodules These lesions appeared chiefly on the face and trunk.

The best *treatment* is purely symptomatic. Cold or warm fomentations, according to the degree of the accompanying inflammatory symptoms, especially of the surrounding parts, and according to the lymphangitis present in greater or less intensity. Cauterization with fuming nitric acid of the malignant pustule or other infective nodules and pustules, recommended by some, is occasionally effective if thorough, but if only partial it is rather injurious; wounds are made which are favorable to the further spread of the bacilli. Of late excision or curetting of the pustule has been advised by many as useful; one practitioner favors treatment by inunction.

The cause of anthrax and the carriers of the infectious material are the bacteria, anthrax bacilli, which have been constantly demonstrated, since the time of Pollender and Davaine, in the blood of affected animals and in the tissue of the pustule. According to the reported examinations by Wagner, Rob. Koch, and Turner, we find in the cutis and as far as the fatty layer streaks and patches of inflammatory cell infiltration, hæmorrhages, necrotic shreds of tissue of discolored aspect which are either firm or undergoing liquefaction, and only in the early stage more hyperæmia and serous infiltration; the epidermis is here and there adherent, later detached, especially at the centre. The anthrax bacilli fill the blood and lymph vessels and (in Wagner's cases) the enlarged papillæ so densely that they hide all other tissues. They are fewer in the neighborhood, but are invariably present under the epidermis wherever it still adheres firmly to the underlying tissue (R. Koch); they are absent

wherever the epidermis is loose or detached, because at such points the organisms of decomposition which have taken root from without overwhelm and destroy them. It is also known that in the cadavers of animals dead of anthrax the bacilli disappear rapidly.

Although for the present of no importance in human pathology, we must mention here the very promising results obtained in cattle by Pasteur's methodical protective inoculations with his attenuated cultures. In this respect, and regarding the manner and mode of infection, together with the symptoms of general and intestinal disease in anthrax (mycosis intestinalis), I refer to books on special surgery and pathology.

In view of its source, anthrax occurs usually in knackers, cattlemen, and persons coming in contact with the bodies and offal of animals affected with anthrax; now and then it is due to the bites of flies that have fed on the bodies.

GLANDERS in man, maliasmus (malleus humidus, equinia, farcy), which arises by the transmission of the same disease from the horse (animals) to man, manifests itself either as a local affection in the same way as all phlegmons produced by inoculated organic poisons, by inflammation, suppuration, gangrene, lymphangitis, adenitis, metastatic suppuration, and may prove fatal by pyæmia or terminate in recovery after the slough is cast off.

Otherwise general infection ensues, with or without preceding local affection. This manifests itself as a general blood-poisoning, by rigors, fever, pain, swelling, œdema, exudation, hæmorrhage, suppuration around and within the joints, and local inflammatory foci on the skin on which are developed numerous pustules of various size, furuncles, hæmorrhagic nodules, and abscesses of very variable extent. The nasal mucous membrane also is often swollen, inflamed, and furnishes abundant purulent secretion. The disease either runs its course with violent fever, head symptoms, complication with pneumonia, and affection of the spleen and intestine, ending fatally within a few days or weeks—acute glanders; or else it becomes chronic, the violent general symptoms abating while the abscesses continually recur, and death ensues after years of marasmus; or finally, in rare cases, the series of local inflammations ceases and the patients recover.

At the autopsy of patients dead from glanders we find, besides the nodules and purulent foci in the skin, similar ones on the mucous membrane of the nose, pharynx, larynx, and bronchi, patches of pneumonia, and manifold alterations of the parenchymatous organs, muscles, and the vascular system.

The diagnosis of glanders requires some care, since it may be readily mistaken for variola, and particularly for pustular and gummatous syphilis.

To insure infection direct contact with a diseased animal is not necessary. Mere sleeping and prolonged stay in a stable that formerly harbored glanderous horses, or even handling the cadaver of one, is sufficient; for the glanders poison has been proved to be volatile. The transmission of the disease from man to man has also been observed.

The glanders bacillus is to be looked upon as the cause of the disease (Löffler, Schütz, Weichselbaum). This consists of microbes the size of a tubercle bacillus, but broader, 2 to 5 μ long, 0.5 to 1.4 μ broad, which forms spores (Weichselbaum). Pure cultures of glanders bacilli injected into horses, rabbits, guinea-pigs, and field mice produce typical glanders. Numerous bacilli are found in the resulting nodules and abscesses, also in the blood and urine. White mice are immune.

POST-MORTEM PUSTULE occurs in persons who handle human or animal cadavers and their products (hides of animals), such as physicians, attendants in dissecting rooms, knackers, and hide dressers. The affection occupies a portion of the dorsum of the hand, in the form of a hæmorrhagic bulla or a very painful follicular furuncle. The course is quite analogous to that described for other forms of infection: either acute local inflammation with lymphangitis, gangrene, necrosis of the soft parts and bones, and recovery; or acute pyæmia and death; or chronic marasmus, with or without recovery.

Another form of inflammation caused by cavaderic infection, which leads to proliferation of the papillary body and peculiar indurations and warts, we shall discuss hereafter.

Reports are also found in the literature of endemic furuncles, such as anthrax hungaricus, esthonicus, bothnicus, Aleppo boil, etc.

ALEPPO BOIL or evil (bouton d'Alepp) alone among all these seems to possess the importance of a special disease.

The first reports of the affection date from the middle of the last century, and agree in the main with those made to the present time. According to them it is an endemic disease, occurring especially in the regions about the Orontes, Euphrates, and Tigris (Pococke, Willemin, Rigler, J. E. Polak, and others), and in particular cities, Diarbekir, Aleppo, Ofra, and others, but no longer met with autochthonously at a few hours' distance from them. All the natives are affected with it, usually between the second and the seventh year, never before the second But every immigrant likewise contracts the disease after a brief sojourn in those localities, certainly after a longer residence.

Similar reports are made, however, from other tropical and subtropical regions, from Delhi in the East Indies (Delhi boil), from

many regions in Africa, from Algiers, where the French troops suffer largely, from Biscra (Biscra button), from Polynesia, etc.

The disease is described in the following manner :

Without any known cause there appears on some part of the skin, usually the face or the extensor surface of the hands and feet, frequently in the neighborhood of the joints, but, it is said, never on the palm of the hand or the sole of the foot, a red, urticaria-like spot the size of a lentil or larger, slightly salient and somewhat itchy. Very slowly, in the course of several weeks or two to three months, the spot develops into a firm nodule the size of a pea, bean, walnut, or larger, which is reddish brown, of an oblong rounded shape, resembling a furuncle, and prominent. Its median surface is covered with thin, dry scales or yellowish-brown crusts, or it appears superficially excoriated. About the fifth or sixth month the central portion usually undergoes rapid disintegration from the surface, and a loss of substance gradually results, which appears as a crateriform, torpid ulcer with viscid serous secretion. After a longer or shorter time the base and margin become more succulent and bleed more readily on touch than they formerly did. At the same time granulations start from the bottom, and the loss of substance closes rapidly, while the infiltration of the circumference disappears. A cicatrix remains, whose extent and form correspond usually with those of the boil.

As a rule the patient has but a single nodule (male button) ; but cases occur in which there are several, from eight to twelve and more, present at the same time (female button).

The disease occurs but once in a lifetime.

But few European physicians know this disease from personal observation, and I myself had not seen it until 1884. Hence a certain distrust has always prevailed regarding the reports and descriptions, and was justified on principle, since a number of diseases which had long been stated to be peculiar and endemic affections proved, on more careful study, to be well-known morbid processes ; this was the case with the siwwens in Scotland, the radesyge in Norway, the skerljevo in Fiume, etc. Geber, who had the opportunity of seeing the disease in its habitat, moreover has shown that all sorts of well-known ubiquitous chronic processes, such as syphilis, lupus, etc., were indiscriminately reported as Aleppo evil. Still, this does not prevent us from recognizing the Aleppo evil as a peculiar disease, which is distinguished by special characteristics from similar affections.

In November, 1884, the disease came under my observation in the person of a physician, Dr. F., who had lived in Ofra, and in three members of his family. The physician had immediately below the left internal malleolus a bean-sized, oval, brownish-red nodule of a soft-elastic consistence, whose surface was smooth except at the

slightly depressed central portion, where it was covered with a thin crust. The nodule was seated in the superficial cutis, with which alone it could be displaced ; on pressure it was slightly painful. At that time the boil was three months old and had begun one month before the patient had left Ofra.

His six-year-old boy exhibited a deep cicatricial loss of substance at the margin of the left auricle in consequence of a boil which had begun in September, 1883, and by June, 1884, had ulcerated and healed. In the doctor's wife, who had left Mesopotamia with the family two and a half months before, a nodule had formed four weeks previously while she was back in Europe ; it was the size of a small pea and was located on the wrist in front of the condyle of the right ulna. A child of six months had a nodule of nearly the same size in the middle of the forehead, which had formed two weeks before in Vienna.

Despite the similarity of the boil with a nodule of lupus, syphilis, sarcoma, leprosy, keloid, and furuncle, it cannot be mistaken for any one of them on careful examination.

The histological examination of the nodule excised from Dr. F. showed a dense cellular infiltration which affected mainly and uniformly the papillary layer, but extended also through the corium into the coil glands and fatty layer along the vessels which it surrounded, together with the vessels of the sebaceous glands and hair follicles. In the central portion of the nodule the infiltration had advanced into the rete, obliterating the outline of the papillæ, and showed many cells indicative of involution, with irregular swellings or granular opacity. At the marginal portion of the nodule the infiltrated papillæ were widened, the cones of the rete were broad and had proliferated deep into the corium.

Nothing positive is known regarding the *etiology* of this peculiar affection. The theories formerly advanced with reference to climatic and telluric influences, mode of life, racial and social conditions, are untenable. An infectious cause seems very probable ; but thus far no one has succeeded in demonstrating the supposed animal or vegetable parasites or in transmitting the disease experimentally (Flemming, Schlimmer, Carter). In the nodule examined by us no microbes of any kind could be found.

LECTURE XXII.

B. CHRONIC INFLAMMATORY DERMATOSES.

CHRONIC DERMATOSES PRESENTING THE CHARACTER OF INFLAMMATION (EXUDATIVE PROCESSES, HEBRA).

ANATOMICAL IMPORTANCE AND CLINICAL SUBDIVISION OF THE CHRONIC DERMATOSES CHARACTERIZED BY INFLAMMATORY PROCESSES—SQUAMOUS DERMATOSES—PSORIASIS.

With the study of the chronic dermatoses characterized essentially by the signs of inflammation, we enter the very heart of the field peculiar to dermatology. Many of the affections discussed above, especially the acute inflammatory, are, in view of a portion of their symptoms, at all times duly recognized by medical and surgical pathology and thus brought to the knowledge of the student It is otherwise with the diseases to be discussed in this group, which largely represent independent affections of the skin.

As indicated by their common characters as chronic inflammatory dermatoses, all of them are based on a nutritive process which develops chronically, and can be termed inflammatory because we find in them generally some symptoms of inflammation, but not the entire symptom-complex peculiar to it. Of the inflammatory symptoms sometimes vascular injection (redness) predominates, sometimes exudation, or again proliferation of the tissue elements. At the same time these processes affect either chiefly the papillary layer, or the deeper layers of the corium, or the glands only with their immediate surroundings, or mainly the epidermis. Since, occasionally, there may occur an aggravation of the inflammatory process to an acute typical inflammation, and many of these forms, like eczema, usually result from acute beginnings, it is hardly feasible to separate the large number of the diseases of this group according to these more minute anatomical differences.

Hence it is preferable to follow Hebra in utilizing, besides the prominent anatomical alterations, also the obvious clinical characteristics in their division, and accordingly to recognize five groups of the chronic inflammatory dermatoses :

1. Squamous dermatoses, scaly eruptions—psoriasis, pityriasis

rubra, lichen scrofulosorum, lichen ruber, pityriasis rubra pilaris, psorospermosis cutanea.

2. Pruriginous dermatoses—itching eruptions, eczema (scabies), prurigo.

3. Folliculitides, acneiform eruptions—acne, sycosis, acne rosacea.

4. Pustular eruptions—impetigo (ecthyma).

5. Bullous eruptions—pemphigus.

Let us consider first the diseases belonging in the first group :

I. SQUAMOUS DERMATOSES.

PSORIASIS.

The term psoriasis, since the time of Willan, is applied to the skin disease which is characterized by the presence of dry, white, glistening scales which lie in punctiform masses or larger flat plates on a sharply demarcated, red, easily bleeding base.

The exceedingly variable forms of the disease all result from the same kind of primary efflorescences. These appear as pinhead-sized, bright-red to brownish-red nodules, which under the pressure of the finger become paler and invisible ; within a few days they become covered with a small white epidermal scale. When this is detached with the finger nail, which is easily done, many minute bleeding points appear on the red base. These correspond to the same number of papillary vessels, which, being swelled by hyperæmia, project and are wounded by the scratching nail.

When many such primary efflorescences are present simultaneously on the skin they present the picture of psoriasis punctata. By peripheral extension of the redness, swelling, and subsequent scale formation, this variety changes into psoriasis guttata and nummularis, accumulations of scales in the form of drops and coins resting on a red, slightly swollen base of the same size ; these spread until, in the course of from one to three weeks, the patches reach the size of a dollar and larger, which always exhibit the same characteristics—lightly adherent scales and an easily injured red base. When the patches are of larger size the superficial scaly plate is surrounded by a red border. This enables us to recognize, as the process advances, redness and swelling of the skin precede the formation of scales. By the direct extension of the several patches and the coalescence of neighboring ones, larger, irregular, uniformly red and scaly, sharply limited psoriatic patches with red borders result—psoriasis figurata, geographica, and finally psoriasis diffusa and universalis.

Generally, however, the several patches undergo involution after they have reached the size of a dollar or the palm of the hand, and have remained stationary for a time (recognizable by the absence of

the advancing red border). While the redness and swelling diminish, the production and accumulation of epidermis lessen, the scale becomes thinner, looser, and after the redness has entirely vanished the last remnant of the scale is cast off. The affected spot is covered with smooth epidermis and is normal in color or brown-pigmented; the latter where the hyperæmia has been of long standing or, as on the lower extremities, where the return flow of the blood is more impeded.

As a rule this recovery ensues simultaneously over the entire extent of the patch; but in some and occasionally in all patches the process differs, in that first the older central portions grow pale and heal, while the peripheral redness and scaling continue. Thus arise red, desquamating circles which may reach a considerable extent—psoriasis annularis (lepra Willani)—and, by the coalescence of similar circles, serpiginous lines—psoriasis gyrata.

The manifold forms under which psoriasis may appear, therefore, represent only different developmental and involutional stages of the same process, which are continually changing. Hence, as Hebra has shown, annular psoriasis in particular should not be regarded as something different and described under a separate name (lepra Willani).

The development and involution of the several psoriatic patches sometimes proceeds very rapidly, within a few weeks, sometimes very slowly. In the former case the masses of scales are looser, glistening white, easily detached so that they are shed in large quantities in bed or when stroked with the palm of the hand. Their production and desquamation follow each other quite rapidly. But over patches which long remain stationary the epidermal scales accumulate in the form of thick, firmly adherent, hard, and usually dirty-white to brown masses.

With regard to localization, arrangement, and extent psoriasis presents great variability. Cases may be seen where only one or a few patches exist, others with numerous disseminated patches or disease of the whole integument. The arrangement is usually irregular. On the trunk the discrete patches may lie in parallel rows following the course of the ribs (cleavage lines of the skin). The extensor surfaces of the extremities, especially the knee and elbow joints, next the hairy scalp and the sacral region, form the most frequent seat of psoriasis, and hence are almost regularly beset with stationary, old spots covered with thick, dirty scales. On the hairy scalp the scales accumulate in the form of thick, irregular masses resembling dry mortar, in which the hairs are felted and which adhere very firmly.

But any other part of the skin of the face, trunk, and the extremities may become affected with psoriasis; some patients are attacked

regularly on the flexor surfaces of the body ; the palm and sole alone are almost uniformly exempt, even when the disease is otherwise universal, and suffer only very exceptionally, contrary to the frequency with which they are attacked by syphilis—a form of disease which is often misunderstood owing to the common designation of psoriasis palmaris et plantaris (*i.e.*, syphilitica).

Form, localization, and extent, the general picture of psoriasis, are also exceedingly variable in every single patient, according to the variety and the stage of the disease.

The course of psoriasis is extremely chronic, but not uniformly so, being composed of irregularly alternating periods in which the process advances and retrogresses.

Rarely the disease occurs, in a patient previously healthy, without prodromata, with an acute general eruption or with a few disseminated spots which enlarge tardily and multiply by sparsely appearing new ones. But, as a rule, a patient will carry for years old, dry, hard patches, over the knee and elbow joints and on the hairy scalp, rarely at other points, which change almost imperceptibly. Then, without demonstrable cause, new patches appear elsewhere, sometimes sporadically, at other times in large numbers. These patches extend constantly, and are increased by additional efflorescences until within a few weeks they occupy a large portion of the cutaneous surface. After some time the patches undergo involution, the eruption of fresh nodules ceases, and the psoriasis disappears, excepting a few remnants which are mainly restricted to the above-named points of predilection. Then follows a period of several weeks or months of relative health, and then another period of exacerbation. This may continue for many years or a lifetime, during which the several stages of decline and increase of the disease are very variable, both as regards duration and the intensity of the exacerbation or remissions, and are obviously uninfluenced by season, external conditions, etc. During one of the exacerbations the psoriasis may become general, the skin from head to foot being uniformly red, covered with desquamating scales, hot, dry, here and there glistening, satiny, tender, and tense. The skin of the face appears shrunken, the lower eyelid ectropic, the patient sits with joints flexed because every attempt to stretch them is followed by fissures of the epidermis and bloody rhagades. The hair of the scalp falls easily, and even transient or permanent baldness may result. This condition is associated with continual chilliness, violent itching, also fever, gastric symptoms, hiccough, loss of sleep, appetite, and flesh, and other dangerous accidents. But this state, too, may be followed by involution to a moderate degree after many months, and even within a short time by proper treatment. Some patients repeatedly experience such exacerbations of their disease. Exceptionally a

person may be affected for a lifetime with limited psoriasis (of the extensor surface of the joints, genital region, etc.), without the involvement of other parts.

In the same way as the hairs manifest their implication by rapid falling, so the nails are diseased during long-continued psoriasis, becoming dry, opaque, brittle, and caseous.

FIG. 25.—PRIMARY NODULE OF PSORIASIS, EXCISED INTRA VITAM. VERTICAL SECTION (LOW POWER).

c b, part of the nodule within which the papillæ (to the right of *c*) are considerably lengthened and widened; at *b* the rete is thickened by swelling of the cells without material proliferation; around *d*—*i.e.*, corresponding to the base of the nodule—there is considerable cellular infiltration about the blood vessels of the papillæ and of the superficial portion of the corium, even around the vessels passing along the efferent duct of a sweat gland.

Like Hebra, I have never seen disease analogous to psoriasis on the mucous membrane of the buccal cavity, though I have observed in some psoriatic patients gray patches, which, however, were due to syphilis or corresponded to leucoplakia buccalis non-syphilitica (Schwimmer).

Of the subjective symptoms I mention, besides those named above

(itching, insomnia, loss of appetite), rheumatic pains in the joints which usually accompany the acute outbreaks, thirst, and dryness of the mouth.

Anatomical examination shows that psoriasis is based locally on an inflammatory alteration of the skin which affects mainly the papillary layer. In microscopic sections of recently attacked portions of the skin excised *intra vitam* (Fig. 25) we find the mucous layer enormously developed, within the papillæ the vessels and meshes dilated, and numerous cells deposited, especially around the former, at whose walls the network also appears denser.

These histological findings (which are analogous to the most recent reported by Kromayer) in a nodule excised immediately after its appearance, upon which no scales were as yet visible (as at *a*, Fig. 25), taken in connection with the clinical symptoms, prove that those authors are in error who, like Robinson and Jamieson, look upon the proliferation of the rete as the primary and essential feature in psoriasis, and who, in view of this erroneous conception, included it among the "epidermidoses," as Auspitz did.

Every one who has observed an acute general eruption of psoriasis knows that it consists of red, smooth points which pale under pressure. These cannot be distinguished from the eruption of variola, herpes tonsurans, or eczema papulosum, and become characteristic only after three or four days by the appearance upon them of white heaps of epidermis.

In the primary efflorescences, as well as on the advancing border of the old patches, we can clearly recognize that the inflammatory vascular alteration and cellular infiltration are the primary features in psoriasis, and that the accumulation of scales is secondary, since every progressing patch shows a red, hyperæmic border. Whenever the latter is absent or disappears, as during internal medication, we may positively assume that the local process is decreasing. The redness always diminishes first, and the scales fall subsequently when normal epidermis has formed beneath them on the no longer inflamed papillary layer.

The accumulation of scales, being a product of proliferation of the rete, is caused by the abundant afflux of plasma from the hyperæmic vessels—*i.e.*, by the inflammatory condition; owing to the rapid reproduction the epidermis cells reach the surface in the condition of the cells of the granular layer—in other words, before they have found time to undergo the normal process of cornification.

The skin corresponding to the old patches of psoriasis shows a thickened corium infiltrated with cells as far as the subcutaneous cellular layer, dilated vessels, slight serous imbibition, here and there pigmented cells. Over very old patches, especially of the legs and the sacral region, I have often noticed a connective-tissue outgrowth

of the papillæ in the form of hard warts. While the alterations corresponding to the recent psoriatic patches may disappear even spontaneously without leaving a trace, the warty outgrowths can be removed only by operation (by cauterization or scraping).

As to *diagnosis*, psoriasis appearing in isolated patches offers the least difficulty. We need but bear in mind the above-described symptoms, especially the thick accumulation of white scales which are easily detached with the finger nail, the bleeding points thus laid bare, the red base, and the sharp limitation of the circumscribed patches. The differentiation, however, may be difficult in general and diffuse psoriasis, which greatly resembles eczema squamosum, pityriasis rubra, and lichen ruber; in psoriasis limited to the hairy scalp, which may be mistaken for eczema capitis, favus, herpes tonsurans, seborrhœa, and lupus erythematosus; in isolated psoriatic circles, which must be distinguished from annular syphilis, herpes tonsurans, lupus serpiginosus, and eczema marginatum; and, finally, in psoriasis nodules appearing acutely over the whole surface, which in the first few days may look like papular syphilis, herpes tonsurans maculosus, and even the efflorescences of variola. In making the differentiation we must bear in mind, besides the peculiarities of psoriasis, the characteristics of the various processes discussed in the respective chapters.

In addition it must not be forgotten that psoriasis may be combined with other cutaneous diseases, especially often with eczema, which may develop directly on the psoriatic patch. In the case of variolous disease the small-pox pustules are always numerous and intense on the psoriatic patches which are particularly hyperæmic.

The *prognosis*, as regards the local alteration caused by psoriasis, is favorable in so far as the skin may return to its normal condition everywhere; at most, on the lower extremities and the seat of old stationary patches a dark pigmentation may remain. The prognosis is favorable also with reference to the general health, which often is entirely undisturbed or affected only during acute outbreaks, especially in general psoriasis, but even then merely temporarily. Permanent disturbance of important functions in consequence of psoriasis is very rarely observed.

The prospect is less favorable, however, as regards the course of the entire disease and its curability. We can never tell whether or how often and to what degree exacerbations will occur, and the term curability applies only to an existing psoriasis. But the occurrence of relapses can neither be prevented nor limited. On the contrary, every psoriatic patient must be prepared for temporary aggravations of his disease. In this sense the affection is absolutely incurable.

Another aggravating fact is that the tendency to be affected with psoriasis may be transmitted from the parents to the children.

Many attempts have been made to ascertain the *cause* of this troublesome, disfiguring, and intractable disease, in the hope of mastering it. Unfortunately we are unable to name any cause, at least none that is dyscrasic. Psoriatic patients are all healthy, robust persons in good condition and appearance ; feebleness among them is a positive exception. Hebra's objective criticism has long disposed of the favorite general etiological factors formerly advanced, such as the herpetic and psoriatic dyscrasia, suppressed menses, etc.

Psoriasis cannot be produced by external irritants. Only in persons already suffering from psoriasis, or disposed thereto, numerous efflorescences appear, as a rule, at points artificially irritated—*e.g.*, pin scratches or eczematous spots—at a time when the disease exacerbates. Psoriasis is absolutely non-contagious and hence can never be directly transferred.

E. Lang and W. Wolff have found in the basal layers of the psoriatic scales fungi which, however, have never yet been brought into etiological connection with psoriasis, particularly as these findings have not been confirmed.

Heredity is the only unquestionable etiological factor, for we rarely meet a psoriatic patient whose parents or other ancestors have not suffered from the disease. But, as only a few children or members of a family are affected, we are not dealing with a true heredity of the affection, but with the tendency, the quality of the skin.

Psoriasis occurs most frequently about the time of puberty and of vigorous middle age, but often enough in childhood. I have seen an eight-months-old child of a psoriatic father affected with numerous efflorescences. The disease frequently continues active to an advanced age.

The *treatment* of psoriasis, in the present state of our knowledge, can do no more than remove the existing morbid alteration of the skin and limit fresh eruptions. An actual suppression of the latter, or even a permanent cure, is beyond our power. To bring about at least the first-named result is a great gain to the patient and presupposes extraordinary skill in the use of the drugs and procedures at our command. These are internal and external.

Numerous remedies have been recommended by the internal use of which it was hoped to cure psoriasis—*e.g.*, mineral acids, mineral waters, diaphoretics, cod-liver oil, antimony, manganese, plumbago, baryta, mercury, iron, anthracokali, blood-purifying drinks, sarsaparilla, spoiled maize flour, lemon juice, etc., or special diet such as a purely vegetable or purely animal diet ; but from none of these can we expect the least effect upon psoriasis, according to our experience at this institution. The only useful drugs are arsenic and tar and its derivatives (carbolic acid).

Arsenic is employed in skin diseases in the form of Fowler's solu-

tion (liquor potassii arsenitis), Pearson's solution (liquor sodii arseniatis), Donovan's solution (liquor arseni et hydrargyri iodidi), and the Asiatic pills (arsenic with pepper or arsenic with opium). We have found arsenic most reliable in the first and last mentioned forms.

Fowler's solution is given in doses of six drops three times a day in 20.0 of distilled water or camomile tea. If no gastric symptoms arise the daily quantity of the solution is increased by one drop every third or fourth day. If begun with twelve drops the increase is made at longer intervals. In this way we may go as far as thirty drops a day, but stop at the dose where an involution of the process becomes perceptible. Yet even when recovery is almost complete the drug is not suddenly discontinued, but decreased gradually to twelve or six drops. In this manner Fowler's solution can be continued for many months without danger. With a like object Lipp uses arsenious acid (white arsenic) by subcutaneous injection in doses of from 0.003 to 0.03. We inject 0.2 of Fowler's solution or 0.02 of the arseniate of sodium daily or every other day in carrying out a methodical cure of psoriasis.

The Asiatic pills are prescribed after the following formula: acidi arseniosi 0.75, pulv. piper. nigri 6.0, pulv. acaciæ 1.5, pulv. althææ rad. 2.0, aquæ font. q. s. ut fiant pil. no. 100. Consp. pulv. pip. nigr. Sig.: Three pills daily.

We begin with a dose of three pills daily, which are to be taken immediately before dinner, increase every fourth or fifth day by one pill, and may in this way reach from eight to ten pills a day. A quantity of five or more is divided into two doses, three pills at noon and two at night, or four and three respectively, etc. Here, too, we stop at the dose where an effect is noticeable. When symptoms of gastritis occur, nausea, colic, diarrhœa, the dose is again diminished. Colic is prevented by the addition of opium (0.15 to 0.75 of arsenious acid and 100 pills).

Under the subcutaneous injection of Fowler's solution and of arseniate of sodium we may occasionally observe an improvement in the psoriasis after a week, but on the average it will not be noticeable until the fourth to the sixth week. The improvement manifests itself, as in the case of the Asiatic pills, not in the shedding of the scales, but first in the lessening of the hyperæmia which forms the basis of the scales. After that the scales fall in a few days all together, so that it appears as if recovery had occurred suddenly.

The total number of Asiatic pills which should or may be given cannot be stated in advance. That they can be administered for many months until the quantity reaches from three to four thousand (*i.e.*, 20–30 grm. of arsenic), we have convinced ourselves in the treatment of lichen ruber. In psoriasis this is not advisable. When

no improvement has occurred after reaching from four to six hundred pills, not much can be expected for the case and another mode of treatment should be begun. Experience has shown not only that some psoriatic patients do not improve under arsenic, but that in the same person the effect is favorable at first, while in a second or third year the drug proves inert. After the use of arsenic, dark pigmentation is apt to remain on the affected points for a considerable time.

As regards the internal use of tar, its effect upon chronic cutaneous diseases has long been known by the laity and the medical profession. Tar water and other preparations, however, owing to their disgusting taste, are borne by very few patients. Even the tinctures and pastils made by French and domestic manufacturers, which on the whole do not taste badly, are refused by most persons.

In carbolic acid we possess an excellent tar preparation which, given in the form of pills, is well borne and acts in an analogous fashion to arsenic. We order: acidi carbolici 10.0, extr. et pulv. glycyrrhizæ q. s ut ft. pil. no. 100, of which from five to ten pills are given daily. The drug can also be given for weeks in larger doses, but I think this is unnecessary. Excepting some slight irritation of the kidneys, I have never noticed the least ill effect from it

Some authors have recommended the balsamics, especially copaiba balsam, as well as tincture of cantharides, phosphorized oil, tinctura maidis (Lombroso), and many others, for internal use. I have had no experience with any of them nor have I read much in their favor.

A noteworthy method of treatment of psoriasis by internal medication is that recommended by the Norwegian physician Greve (1881), which has been carried out with good results by Haslund since 1882, and consists in the administration of large and increasing doses of iodide of potassium. He begins with 3–4 grm. of iodide of potassium per day in aqueous solution, raises the quantity every three or four days by from 1–2 grm. so that the patients reach from 30–40 grm. a day within a few weeks. Some patients in the course of six weeks to two months have consumed as much as from 1,500–2,000 grm. of the iodide of potassium without losing weight to any material extent; a few even gained in weight. I have consistently carried out the method in many cases, and cannot deny its efficacy.

The local or external treatment of psoriasis is connected with much trouble, but has the advantage that it is uniformly effective, provided the measures and methods are skilfully selected and used.

One part of the treatment is to be directed toward the removal both of the scales present at the time upon the epidermis and of those newly forming from day to day, in order that the second part of the treatment, the direct application of the remedies to the affected skin, may be possible.

The removal of the accumulated scales is effected by softening them, rendering them friable, and detaching them by mechanical or chemical means. To this end we make use of fats, of water, of maceration by the perspiration, and of caustics.

The fats to be used are olive oil, lard, cod-liver oil, or glycerin, vaseline, etc. But they must be applied in such large quantities, and so persistently rubbed in and divided, that the maceration and detachment of the epidermis exceed its regeneration. Where the disease is locally limited—for instance, at the elbow and knee—simple ointment or cerate may be spread on linen, applied, and fastened with flannel. The most intense macerating effect is produced by cod-liver oil, which is also well borne by most persons. It becomes objectionable only through its odor, the damage it does to the bedding, and, finally, through the production on some skins of a very troublesome papular eczema. In the latter case the inunction is interrupted, the skin is dusted with starch, and the oily linen is removed until the eczema has disappeared and another procedure can be substituted. In a few cases of general psoriasis I have observed under cod-liver oil a detachment of the epidermis over large surfaces and denudation of the corium, with consequent violent pain and fever, even typhoid symptoms as after burns. For the treatment of this condition the patients were placed in the continuous water bed.

Water, for macerating the epidermis, may be employed in the form of Priessnitz's packs for single limbs, parts, or the whole body, according to the extent of the psoriasis. Water is used most judiciously in the form of baths, which, as a rule, should be prolonged, according to Hebra's method, for from three to six hours daily and longer. It is used thus partly as a macerating agent, partly as a medium for the methodical application of the mechanico-chemical treatment by means of soaps and friction by which the epidermis on the psoriatic patches is energetically detached, and, finally, as a vehicle for the employment of special remedies—for instance, tar (tar baths) or Vlemingkx's solution. Indifferent and sulphurous thermal baths and hydropathic cures, provided the skin is daily exposed to their influence for a sufficient length of time, are useful to this extent in psoriasis; the last-named act, moreover, through the cold in moderating inflammation.

Rubber coverings, caps for the head, jackets, drawers and shoes for the trunk and extremities. gloves for the hands, produce a very intense and rapid maceration of the psoriasis scales; when worn for a long time they even render the psoriatic patches paler. But these, too, occasionally cause an artificial eczema or considerable swelling of the skin.

Soaps—best, green soap, and, for the face and head, spiritus saponat. kalinus or naphthol soap—serve partly for macerating the

epidermis, partly for the removal of the macerated scales and fatty substances applied to the skin ; partly, also, they are direct remedies.

A rapid exfoliation of the epidermis is effected by a course of inunctions with soft soap. This is somewhat diluted with water, rubbed into the skin with the palm of the hand, and allowed to remain. The procedure is repeated twice daily for seven days. The epidermis becomes brown, wrinkled, and mortified, and is cast off in the succeeding three or four days in large shreds, after which a bath is taken (Pfeuffer's method).

Thick, hard masses of scales are removed in from twelve to thirty-six hours by the application of strips of flannel coated with soft soap and tied down, or by coll. emplastrum ; this may leave the skin raw. The method is at times suitable for psoriasis of the knees and elbows.

Stronger caustics—such as concentrated potash solution (1 : 2), acetic acid, citric acid, hydrochloric acid, etc.—will find application only now and then when all other methods of maceration have failed to remove the epidermis.

Finally, use may be made of the purely mechanical procedure, by means of the curette, friction with sand and pumice stone, at a few points in order to remove very hard epidermic masses.

The true remedies for psoriasis are those which are capable of reducing the hyperæmic swelling and inflammation of the skin that set up the formation of scales. First among these stands tar, aside from those named above which, like the cold wet packs, soaps, etc., act partly in this direction.

Altogether, tar is the best agent for dispelling chronic or subacute hyperæmia of the papillary layer, and hence acts satisfactorily in psoriasis. Here we gain the strange experience that in psoriasis tar can be applied to the raw bleeding surfaces of the skin without increasing the inflammation, while in eczema it intensely irritates the skin deprived of epidermis.

Of the varieties of tar we employ those mentioned above : oleum fagi, beech tar ; oleum rusci, birch tar ; more rarely oleum cadini, from Juniperus oxycedrus and tinctura rusci (olei rusci 50.0, ætheris, spir. vin. rectif., āā 75.0, olei lavandulæ 2.0). Less serviceable is a derivative of tar, resineon, which likewise represents a fixed oil.

The tar is usually applied in the following manner : The psoriatic patches having been cleared of their epidermis by soap in the bath, a thin layer of tar is rubbed in energetically once or twice a day, or at night only, by means of a bristle brush, after which the patient dresses in flannel clothes. This procedure is repeated every day.

A more energetic effect is produced by the tar bath. The patient in the bath is first thoroughly rubbed with soap ; immediately after-

ward all affected spots are painted with tar and the patient is returned to the water, where he remains for from four to six hours. At the end of the time he is washed and dried, and then treated with some other drug.

As possible injurious effects of the tar should be noted : First, a local inflammation of the skin where two adjoining surfaces come in contact and warm each other—for instance. scrotum and penis. This is prevented by the insertion of pledgets of lint or cotton coated with dusting powder. Second, symptoms of acute absorption of tar—tar intoxication. It happens at times that, after the first extensive application of tar, so much of the latter is absorbed into the blood as to cause a group of symptoms of poisoning. The patients suffer from fever, nausea, eructations, coated tongue. vomiting of tarry black masses, diarrhœal stools of similar liquids, ischuria, strangury, and the emission of tarry black urine. After from twenty-four to forty-eight hours profuse perspiration sets in, the symptoms abate, diuresis becomes freer, the urine is olive-green and later clear, and the attack is over. After that the patients usually bear the drug well. It is advisable, however, in order to guard against such complication, to paint only limited surfaces during the first days and to watch the urine. As soon as the latter becomes olive-green the tar is stopped. After a while the system gradually becomes accustomed to the treatment. In young persons and children this caution is all the more necessary.

The third noxious effect of the application of tar is the occurrence of numerous acne nodules, especially on the extensor surface of the lower extremities and hairy parts. The nodules are painful, hard, and have a black point or a hair in the centre ; on their appearance the use of tar must be interrupted.

Sulphur is used in the form of natural or artificial sulphur baths. For the latter we make use of Vlemingkx's solution, a sulphuretted lime, which, prepared after Schneider's modification, is official in Austria. The solution is used like the tar ; the patient, after being rubbed with soap in the bath, is painted with it and left in the water for several hours ; or else it is applied after the bath and left on the skin the whole day. In the latter case the skin becomes very dry and the solution causes burning ; hence it is best used in alternation with other drugs. On parts with delicate skin the solution may even exert a caustic effect and lead to the formation of an eschar ; therefore it should never be applied to the face.

An excellent effect is produced by Hebra's modification of Wilkinson's ointment, which unites the action of sulphur, tar, soap, and fat (sulph. citrini, olei fagi, āā 50.0 ; sapon. viridis, adipis, āā 100.0 ; pulv. cret alb., 10.0). The ointment is rubbed in twice a day for six days, no bath being taken during that time. Only after the

epidermis has been cast off, on the tenth to the twelfth day, should the bath be permitted.

White precipitate ointment, of a strength of 2.0–5.0 to 40.0 of emollient ointment, thinly applied with a brush to raw psoriatic patches, is suitable for psoriasis of the face and hairy scalp and some patches on the body, as it is colorless and odorless. If used extensively it is apt to cause salivation. Rochard's ointment, a mercurial preparation, acts still more energetically. Its formula is: iodi puri 0.50, hydrarg. chlor mitis 1.50; leni igni fusis, adde ung. rosat. 70 0. This often causes an annoying eczema

Besides those named, use can also be made of ointments containing acidulated nitrate of mercury, protiodide and biniodide of mercury, subnitrate of bismuth, oxide of zinc, salicylic acid, carbolic acid (1 : 40 to 5 : 40); but on the whole all these have but little effect.

All drugs heretofore known, however, are surpassed in their action upon psoriasis by chrysarobin, which was first introduced into practice by Balmanno Squire in 1878. It is obtained by extraction with hot benzol, in varying percentage, ranging as high as eighty to eighty-five, from goa powder, a dirty grayish-green powder consisting largely of the woody and medullary fibres of a leguminous tree indigenous to Brazil, in which country and in the East Indies (Goa) it had been effectively used for some time for various skin diseases, especially ringworm. A patient having called attention to the curative effect of goa powder upon psoriasis, Balmanno Squire first used the latter, and subsequently the golden-yellow powder obtained from it by extraction. This product was first supposed to be chrysophanic acid (Attfield), but, in accordance with Liebermann's investigations, it is now known as chrysarobin.

Chrysarobin is a yellow substance composed of delicate, needle-shaped crystals; it belongs to the phenol group; is almost insoluble in water, but dissolves easily in hot alcohol, benzol, glacial acetic acid, and hot fats and vaseline. It is best used in the form of an ointment: chrysarobini 10, vaselini or unguent. emoll. 40, and a weaker one of half the strength. The bulk of the scales having been detached by a bath and soap, the chrysarobin ointment is thinly applied by means of a bristle brush; this is done once or at most twice a day for several days. During this time bathing and washing are abstained from. Some of the spots appear remarkably white and free from scales after from four to eight applications, others after from twelve to sixteen or twenty, while the adjoining skin is discolored bluish red or violet brown.

Aside from the strikingly rapid effect on the several psoriatic patches, the drug has the advantages of being odorless, of causing no pain even on raw bleeding surfaces, of leaving the skin supple, and of doing away with the troublesome and expensive bathing.

Its drawbacks are : staining of the linen (violet-brown), the nails, the hairs, and the healthy skin, on account of which it can hardly be used about the face ; moreover, its inflammatory action, manifested by diffuse reddening or painful swelling or the formation of acne and furuncles—forms of dermatitis which often spread over the entire body, are associated with fever and intense disturbance of the general health, and require from two to three weeks to subside. Nephritic symptoms come but rarely under observation.

In order to restrict the application of the drug to the psoriatic patches and guard the skin against staining and irritation, Pick wisely recommended it to be used suspended in gelatin (gelatin 50, water 100, chrysarobin 10 parts). This must be liquefied on a water bath before application. The same end is served by suspension in alcohol, collodium, traumaticin (Auspitz), linimentum exsiccans (Pick), epidermin (S. Kohn), which are most to be recommended ; while mixtures with glue and varnish (Unna) have an irritating effect. The brittleness of the pellicles formed by the last two methods can be modified by the admixture of glycerin (Unna) or a subsequent painting with glycerin (Pick, Auspitz).

Anthrarobin, which is allied to chrysarobin and was first recommended by Behrend, while not nearly so effective as the latter, likewise stains the skin and the linen, but is better borne by the skin and may be used to advantage in fifteen to twenty per cent strength.

Pyrogallic acid, a body chemically related to chrysarobin (bioxyphenol), has been tried and found useful for psoriasis by A. Jarisch at our clinic. The ointment (pyrogallic acid 1 part, vaseline or emollient ointment 10 parts) is, like chrysarobin ointment, odorless and not painful ; and while it does not act so promptly as chrysarobin, it has an excellent effect. It never gives rise to intense inflammation unless it is spread on linen and applied. As a disagreeable incidental effect of pyrogallic acid, a sensation of dryness and itching occasionally occurs, when its use must be suspended and the itching spots painted with plain fat or with tinctura rusci ; or else follicular nodules and pustules form, which must be softened by means of mild ointments or plasters. Somewhat more alarming is the occurrence of strangury and the excretion of olive-green or tarry black urine, accompanied by moderate febrile movement and nausea in some patients whose whole body has been repeatedly covered with pyrogallic ointment. The symptoms are the consequence of the extensive absorption of the pyrogallic acid and its excretion by the kidneys. The condition soon passes away. Aside from these accidents no harm need be feared from the application of the pyrogallic ointment, and hence its use is to be warmly recommended in practice. The ointment is likewise rubbed in with a bristle brush once or twice daily for as long a period as may be necessary. Now and

then a bath may be taken. Both the psoriatic and the healthy skin are stained brown for a considerable time by this ointment. The use of pyrogallic acid in gelatin or traumaticin, with alcohol, collodium, or glue (Unna), for psoriasis is less suitable.

In the hope of finding in one of the many substances obtained in the various modes of distillation and chemical separation of tar its active properties without its disagreeable incidental qualities, and after excluding those bodies hitherto shown to be unsuitable for our purposes—like resineon, the benzols, and carbolic acid among the phenols—I have, on the advice of Prof. E. Ludwig, introduced naphthol into our therapeutics, which I have found effective and indicated in all cases where tar had been employed, and so also in psoriasis.

There are two isomeric bodies bearing the name of naphthol, chemically distinguished as alpha naphthol and beta-naphthol. The one used by me in the treatment of various cutaneous diseases is beta-naphthol, in practice briefly designated as naphthol.

Naphthol—*i.e.*, beta-naphthol—having the chemical formula $C_{10}H_8O$, is manufactured on a large scale and comes on the market in large pieces. These are dark violet-brown, have a crystalline structure, and are easily crushed to a coarse powder of a reddish-brown color. Since the drug has come into great demand it has been furnished as a white crystalline powder which is obtained by recrystallization from the above-described form.

Naphthol has a faint odor, recalling that of carbolic acid, but more sweetish ; its taste is sharp like red pepper. It is soluble in an equal weight of alcohol, but only sparely in water, more readily in equal parts of water and alcohol, and quite rapidly in oils and solid fats. Hence it may be employed in dilute alcoholic solution or in the form of oils and ointments. In addition I have advised a naphthol soap and a naphthol-sulphur soap, which are suitable for certain forms of disease. Naphthol solutions and ointments, at first colorless, become reddish in contact with the air ; the linen impregnated with it is also sometimes stained, but is easily cleansed by washing with soap.

Naphthol, especially when applied to the skin in the form of an ointment, is rapidly absorbed in large amounts and excreted by the kidneys, according to J. Mauthner as a sulphate of naphthol. The latter renders the urine turbid, but it is cleared by the addition of alcohol. In man, however, we have never observed albuminuria, or hæmoglobinuria from the decomposition of the red corpuscles, which latter Neisser has found in rabbits and dogs poisoned with naphthol. In a word, we have noticed no ill effects, although we have used naphthol ointment in from five to fifteen per cent strength on several hundred patients, applied to the whole body several times daily for a period of weeks.

Still, treatment by means of naphthol ointment should be carried

out with care, as in the case of tar, chrysarobin, and pyrogallic acid ; the drug, especially in young patients and those with tender skin, should not at first be applied in too concentrated a form and over large surfaces, and the urine must be watched.

Even greater care must be exercised in the treatment with an alcoholic solution of naphthol (0.5 to 5 per cent) ; and it is not advisable to leave it to the patient, as might be done with salicylic, carbolic, and boric acid and similar solutions, without the regular supervision of the physician. Even a 0.5 per cent solution, painted on three or four times, produces a dry disintegration of the epidermis —an effect corresponding to that of tar, which is sufficient in some cases. A stronger solution causes an irritation of the skin resembling a toxic erythema, urticaria, and even slight dermatitis.

In psoriasis, naphthol ointment (ung. emoll. 100 parts, naphthol 15 parts) does not act so promptly as chrysarobin and pyrogallic acid, but it often acts quite rapidly, especially upon old psoriatic patches at the elbow and the knee, and is to be particularly recommended for psoriasis of the face, head, and hands, as it does not discolor the skin and hair. It is brushed on twice a day for six days ; on the seventh it is washed away with soap, and so on. The application and washing may also be used on alternate days. Now and then the ointment causes a sensation of burning of the skin which lasts for some time ; this may be moderated by dusting with starch. Inflammation of the skin, furuncle, or eczema never results.

Hydracetin (P. Guttmann) and hydroxylamin, 0.2–0.5 in alcohol (Fabry), are not to be relied upon in psoriasis ; they produce such pronounced local and general toxic effects that their employment is to be condemned.

Aristol has been very warmly recommended by Eichhoff (five to ten per cent mixtures). Its effect in psoriasis is very variable ; but, as it is otherwise innocuous, it may be used at times.

Gallacetophenon (ten to twenty per cent), which has been tested by Spiegler at my clinic, seems to be useful.

In estimating the curative effect of a drug it must not be forgotten, as I have pointed out before, that at the time of the periodic spontaneous involution of psoriasis all remedies act more promptly, since the hyperæmia on which the process is based is decreasing and no fresh nodules are formed ; and that in the stage of exacerbation, the advancing hyperæmia of the several patches, and the continual formation of new nodules, every mode of treatment must be less effective.

From this large array of drugs and methods the physician must choose the most appropriate, taking into consideration at all times the form and intensity of the disease and the individual and external conditions of the patient.

The duration of the treatment, *cæteris paribus*, varies greatly in different persons and in the same patient at different periods—sometimes it is very brief, at other times almost interminable. Recovery may be expected most speedily when the psoriasis is in its declining stage, but nearly all remedies fail when the patients come under treatment during the stage of exacerbation. I have already stated that permanent recovery from psoriasis is unattainable by any method of treatment.

LECTURE XXIII.

PITYRIASIS RUBRA—LICHEN SCROFULOSORUM—LICHEN RUBER.

PITYRIASIS RUBRA (HEBRA).

MANY authors and practitioners apply the term pityriasis rubra, which has been current since the time of Batemann, to all cases in which large surfaces or the whole of the skin appears chronically red and scaly; they do so for the reason that the etymological significance of the term exactly corresponds with the appearance of the disease (*πίτυρον*, bran, and *ruber*). However, such a condition of the skin may be due to several quite different processes—eczema, psoriasis, lichen ruber, pemphigus foliaceus, etc.—and in all such cases it signifies nothing but a certain stage of any of the processes named. On this account we do not employ the term pityriasis rubra for such forms of disease, but the name of the affection to which the erythema and desquamation are due—that is, eczema, or psoriasis, or lichen ruber, etc.

By pityriasis rubra we mean a peculiar disease, first described by Hebra, which is of very rare occurrence and is characterized, besides its chronic course, by the fact that it presents no other form of eruption (neither nodules, nor vesicles, nor pustules), but that from the start and during its entire course there is nothing but reddening and desquamation of the skin.

The opportunity rarely offers to see the disease in its first stages; twice only has it come under my eye at this period. In these cases the disease began on the flexor surfaces of the joints. The skin at the bend of the thigh, the axilla, and the popliteal space appeared bright red over rather circumscribed areas, with slightly raised temperature, and covered with small, thin, moderately desquamating scales; no infiltration, no weeping, no efflorescences were present.

The majority of cases came under observation in more advanced stages, when the disease had spread over the greater part of the body or involved the whole integument uniformly.

At all points the skin ranges from bright to bluish red and is livid, especially on the lower extremities, the epidermis becoming detached in thin, small scales or in larger thin lamellæ; but nowhere is there any true accumulation of scales, or complete exfoliation of the

epidermis, or weeping. The skin of the face and the head exhibits the same quality, while the palms of the hands and the soles of the feet are very pale or injected and covered with a shining, thicker accumulation of epidermis. The temperature of the skin is raised. The redness pales under pressure, leaving a yellowish tinge. Subjectively the patients experience a moderate itching and continual chilliness.

The disease occurs without demonstrable cause and without any known prodromata, on several or many points simultaneously, especially the flexor surfaces of the joints. Within a few months, or from one to two years, it spreads over the whole body, retaining its original character.

The course extends over many years and never exhibits a change, in the sense of an involution ; there is only an increase in the trophic disturbance of the skin, due to the chronic hyperæmia.

While the skin showing the above-named quality retains its flexibility and elasticity from one to three years, so that the patients may follow their vocation with no greater inconvenience than excessive itching, disturbed sleep, occasional indigestion, and continual chilly sensations, in the further course there will be scattered thickenings of the common integument, partly by œdematous swelling, partly by a greater accumulation of scales. In this stage the appearance most closely resembles that of chronic general psoriasis or a general eczema.

In the meantime the color changes from a bright-red to a more livid hue, and the skin evidently begins to shrink, becoming, as it were, too narrow for the body. Owing to the tension of the skin the mouth can be only partially opened ; the lower lids are ectropic, the fingers semi-flexed ; over the extensor surfaces of the knees and elbows the skin is smooth, glossy, and thinned ; over the legs it is tightly drawn. with a satiny lustre, and can hardly be pinched into a fold ; the skin of the soles is in a similar condition, the epidermis being excessively thinned, so that walking is interfered with through the sensitiveness of the plantar surface ; the hairs on the head and body become thin and fall ; the nails of the fingers and toes are thin, glassy, brittle, or thickened and cheesy. During this time the general nutrition has likewise suffered greatly, the subcutaneous cellular tissue has nearly disappeared, and a general marasmus has developed.

At the points where the skin is tense, especially on the legs and joints, the epidermis cracks extensively or is detached for some distance. thus giving rise to scattered shallow, sometimes very large raw surfaces, at times even to ulcers resembling bed-sores.

In one case I have observed, three times in the course of two years, spontaneous gangrene of the skin over the right shoulder, the thigh, and the anterior surface of the abdomen. This began with

the size of a dime, and, progressing at first uniformly, later only at a portion of the periphery, led to losses of substance the size of the palm of the hand and lasted several months before it healed.

These patients, and all other cases which have become known to Hebra and myself, after a few or many years of the disease died of marasmus, with or without complicating pneumonia, diarrhœa, and tuberculosis.

In view of this experience, the *prognosis* of the disease must be said to be unfavorable, although I believe I have cured one case that I have lost sight of, and although one of my colleagues who is familiar with the subject informed me verbally that he himself had once suffered from pityriasis rubra and had recovered.

Nothing is known about the *etiology* of the disease. Hebra observed about fifteen cases, but I have met with only six. All the patients were males, one of them in the twenties, the rest between forty and fifty years, and the disease had begun in those years, excepting one which dated from childhood.

The minute *anatomical* alterations in pityriasis rubra have been studied by Hans Hebra in the two cases on which a post-mortem examination was made. In one the disease was of a more recent character, and the skin under the microscope exhibited a moderate inflammatory infiltration. Similar results were obtained in later examinations by Elsenberg (1876), Petrini (1889), and Jadassohn (1892). In the other very advanced case extreme atrophy of the skin was found—namely, disappearance of the rete and the papillæ, sclerotic change of the connective tissue and predominance of the elastic fibres, abundant pigment deposit in the corium, and obliteration of the sweat and sebaceous glands and hair follicles. In both cases there was in addition tuberculosis of the lungs, the intestines, and, in the more advanced case, also a tubercular nodule in the cerebellum.

In connection with the last finding Fleischmann has called attention to the fact that he had observed a disease corresponding to pityriasis rubra in children, in whom solitary tubercles in the brain were found at the post-mortem In one autopsy I found atheroma of the cutaneous arteries (so did Petrini). The findings mentioned do not offer a sufficient basis for the etiology of the process.

The *diagnosis* of this cutaneous affection is not easy. Its positive characteristics are, as above described, only sparse; and hence we must establish also the negative fact of the absence of the symptoms peculiar to psoriasis, lichen ruber, squamous eczema, pemphigus foliaceus, and many chronic erythemata. In every case, therefore, these diseases must be considered in the differential diagnosis and be excluded.

As regards the *treatment* of this disease we must consider the

symptomatic indications. In many instances we have observed that tar and fats merely increase the local process. In one case which was more than two years under my treatment, I employed methodically by the mouth arsenic, carbolic acid, and Zittmann's decoction, without any result; locally, according to the change in the cutaneous symptoms, continuous baths, tar baths, modified Wilkinson's ointment, strapping with diachylon ointment, rubber clothing, inunction with cod-liver oil, simple fats, etc., with the only result of temporarily mitigating some of the symptoms.

In but one recent case, that of a young man, has recovery been obtained from the internal use of carbolic acid, after all local measures had made the cutaneous affection worse.

LICHEN.

The word lichen is still largely used in different meanings, most frequently, it is true, after Willan, to designate small nodular efflorescences in general, without reference to their nosological importance. If to-day such expressions are used as lichen syphiliticus, lichen lividus, lichen urticatus, lichen agrius, etc., they are meant to designate in the first place merely the physical form represented by small nodules, and not the idea of a disease. Hebra was the first to connect this idea with lichen.

According to Hebra the term lichen as applicable to a morbid process should be restricted to a disease in which nodules form and persist in a typical manner, and which in their entire chronic course undergo no change into efflorescences of a higher grade—*i.e.*, vesicles or pustules—but suffer involution as such.

We know only two diseases presenting this fixed character, which were first established as pathological entities by Hebra, namely: (1) lichen scrofulosorum, (2) lichen ruber.

LICHEN SCROFULOSORUM.

This dermatosis is characterized, aside from its chronic course, by very low, slightly resistant, pale red to brownish or livid red nodules, arranged in groups and patches varying from a dime to a dollar in size, here and there in curves and circles. The nodules are millet-seed- to pinhead-sized, and are crowned with a small scale or more rarely with a very minute vesicle containing pus; after a prolonged existence they undergo involution. They cause but little itching, persist for many years almost unchanged, then retrogress completely with slight desquamation of the epidermis and fading color, without leaving a trace of their presence.

The regular and predominating seat of the exanthem is the trunk, back, and abdomen. At first there are only isolated patches of these nodules; later the adjoining groups may approach each other and

thus represent an apparently diffuse disease, in the confines of which the skin is dirty brownish-red and covered with thin, easily detached scales. Still, its composition of different groups can be recognized, as well as the nodules constituting the latter.

Besides the groups mentioned and the confluent patches, there are also some disseminated nodules and others arranged in circular lines; in addition to spots ranging in size from a cent to a dollar, which are characterized by the fact that the openings of the sebaceous glands and hair follicles are slightly prominent and more distinctly marked (lichen pilaris, cutis anserina)—the beginning formation of nodules.

Finally, though rarely, through the involution of the central and the formation of marginal nodules, there occur large circles containing several rows of nodules, and, by the confluence of these, serpiginous forms of lichen scrofulosorum.

The development is exceedingly slow and imperceptible, and the course very indolent. When the eruptions have become more numerous, after months, analogous nodules and groups appear also on the flexor side of the upper and lower extremities; those on the legs acquire a livid halo (lichen lividus); other efflorescences form on the face.

As a concomitant symptom we find in intense cases eczematous disease of the scrotum and the pubic region, with the secretion of a most offensive sero-fatty fluid which dries into crusts having a rancid odor; also pustules and crusts (eczema impetiginosum) at the mons veneris, due to inflammation of the several hair follicles; finally, nodules and pustules, resulting from hæmorrhage and exudation into the hair follicles of the lower extremities, which are surrounded by a hæmorrhagic areola (acne cachecticorum).

Almost without exception (in about ninety per cent) patients affected with lichen scrofulosorum have an indolent swelling of the submaxillary, cervical, and axillary glands which may reach the size of a nut, or even of the fist, and sometimes suppurate; at times also periostitis, caries, and necrosis, with or without scrofulous ulcers of the skin, which latter is generally cachectic, giving a peculiar dry, fatty sensation to the finger.

The process is to be found without exception in young persons who are markedly scrofulous, for which reason the term lichen scrofulosorum appears to be well chosen. This would at the same time give the supposed cause of the affection. We have only rarely observed the disease in scrofulous persons in the twenties, but never in older or otherwise healthy-looking individuals; the majority were before or at puberty.

I have shown by microscopical examination that the local process of lichen scrofulosorum consists in a cell infiltration and exudation in and around the hair follicles and their sebaceous glands (folliculitis),

as well as into the papillæ immediately adjoining the opening of the follicles (Fig. 26). Every nodule, therefore, corresponds to a follicular opening and its neighborhood. The papillary swelling and infiltration represent the nodule, and the accumulation of hyperplastic epidermis or of exudate in the follicular opening represents the central scale or pustule.

The latest investigations by Jacobi and Sack have practically shown the same result. Still, these authors, like Michelsohn, Hallopeau, Darier, and others, in view of the presence of giant cells, and, according to Jacobi, of some bacilli, were inclined to look upon this

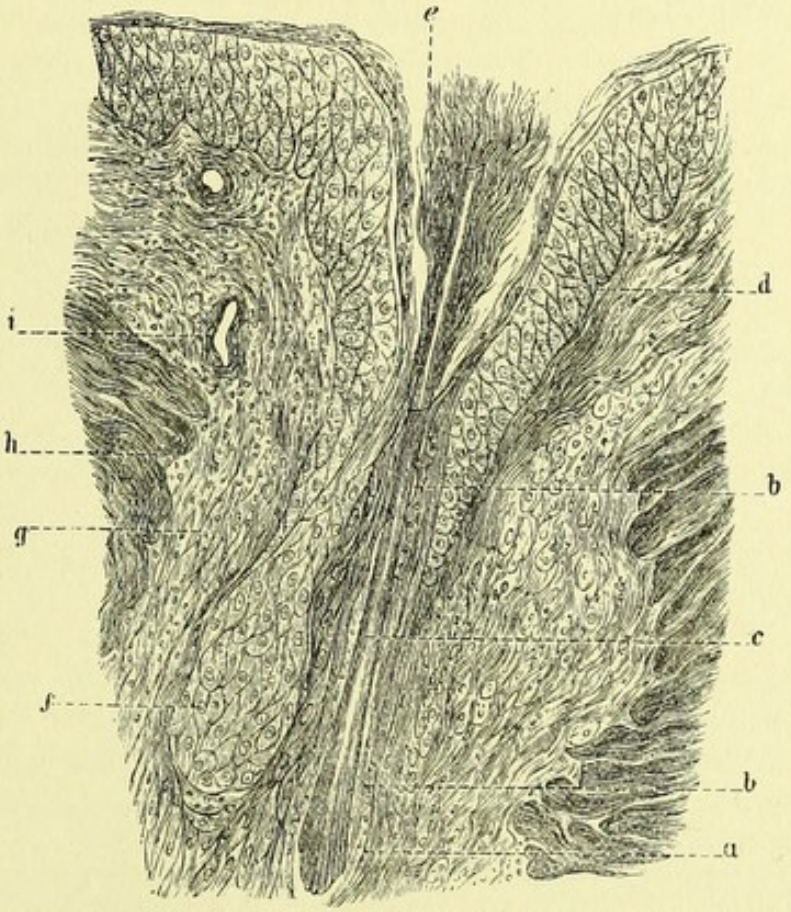

FIG. 26.—SECTION OF A NODULE OF LICHEN SCROFULOSORUM. (HIGH POWER.)

a, hair follicle ; *b b*, root sheath of the hair shaft (infiltrated with cells) ; *c*, hair ; *d*, rete Malpighii, its cells displaced longitudinally, exudation cells between them ; *e*, accumulated epidermis in the opening of the follicle ; *f*, sebaceous gland ; *g*, inflammatory (cellular) infiltration into the perifollicular connective tissue continued into the papillæ ; *h*, adjoining normal connective tissue of the corium ; *i*, blood vessel.

cutaneous affection as analogous to the miliary tubercle, and hence as a variety of cutaneous tuberculosis—a view which is by no means supported by the clinical course of the disease. But it is correct, as shown above, that the persons affected almost without exception present evidences of so-called scrofula.

The process in the main may be called benign because it may

completely disappear and only a few follicles perish by suppuration and cicatrization (Fig. 27). The spontaneous course may last several years.

The *diagnosis* of the disease as thus characterized is not difficult if we bear in mind the uniformity of the nodules, their occurrence in groups, their main seat on the trunk, their softness and slight prominence, and their complication with the above-described glandular swellings and the signs of scrofula.

It may be confounded with : 1. Papular eczema, which occasionally occurs in young children in the form of flat, scaly nodules that, corresponding as they do with the hair follicles, may, like the latter,

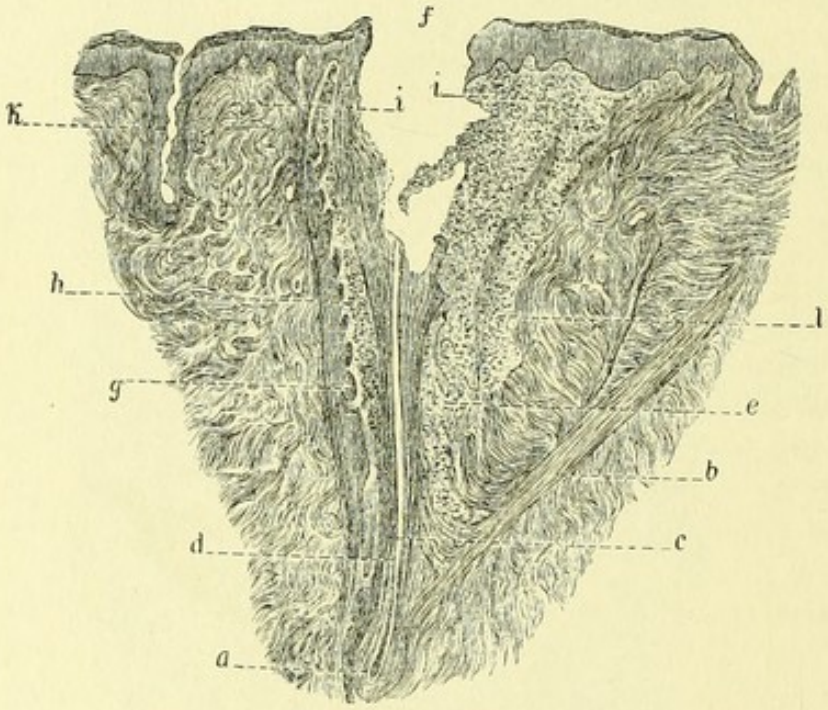

FIG. 27.—SECTION OF AN EFFLORESCENCE OF LICHEN SCROFULOSORUM.

a, hair follicle ; *b*, arrector pili muscle ; *c*, root sheath of the hair shaft, *d* ; *l e*, cellular proliferation around the hair follicle ; *f*, opening of the follicle ; *g*, root sheath of the hair detached from the follicular wall by exudation cells, *h* ; *i*, papillæ of the cutis infiltrated with cells ; *k*, opening of a sweat gland.

be arranged in circular lines and groups (lichen eczematodes, figuratus of authors) ; still the localization is not so typical, and in the acute development the process may increase to an eczema vesiculosum.

2. The small papular syphilide, so-called lichen syphiliticus. Here the nodules, as a rule, are not in groups, but mainly arranged in circular lines, very hard and shining, projecting above the level of the skin, chiefly located on the flexor surfaces of the joints, and there is rarely an absence of one or another larger lentil-sized efflorescence among the smaller nodules. Since these never occur in

lichen scrofulosorum, the differentiation will be made by the demonstration of a single larger, harder papule.

The variety of lichen to be next discussed, lichen ruber, has such marked characteristics that it cannot be readily mistaken for lichen scrofulosorum.

Recovery from lichen scrofulosorum is certain to ensue if the patient's nutrition is improved. Under such circumstances the general quality of the skin approaches the normal and the nodules undergo involution. Great aid is given by the internal use of cod-liver oil, with or without iodine—*e.g.*, iodi puri 0.15, olei morrhuæ 150.0—one tablespoonful morning and evening.

Improvement is more rapid when fat is applied to the skin by inunction with cod-liver oil two or three times a day. In from six weeks to three months the most intense form of lichen scrofulosorum may thus be made to disappear completely, while at the same time the glandular swelling and the symptoms of acne cachecticorum, eczema scroti, etc., are recovered from.

LICHEN RUBER.

This remarkable, both mysterious and dangerous cutaneous disease was first recognized by Hebra as an affection *sui generis* and called by him lichen ruber. What this investigator taught at the time with reference to the symptomatology and termination of the disease has been supplemented by later observers, on the strength of which we and most of our specialists are forced to distinguish two forms of lichen ruber: lichen ruber acuminatus and lichen ruber planus.

Lichen ruber acuminatus is the form originally described by Hebra. It is characterized by disseminated, red, conical, very firm nodules, ranging in size from a millet seed to a pinhead, crowned with a thick accumulation of epidermis. When the nodules are closely crowded together they have a rough feel, like the outer surface of a grater, and finally coalesce into diffuse, red, scaly patches.

The process begins with a rather acute eruption of the above-described nodules, which are either scattered over the whole body or confined to some spots (flexor surfaces of the joints, trunk). At first the nodules are irregularly disseminated, but soon arrange themselves in straight or curved lines, or else crowd irregularly together, numerous fresh nodules arising between those of the first crop.

In this way, within three or four months, trunk, face, and extremities are covered with more and more closely packed nodules, while the healthy islands of skin become fewer and smaller. Then here and there, by the complete coalescence of the closely packed nodules, diffuse morbid patches appear within which the skin is uniformly thickened, red, scaly, fissured, crossed by deep furrows, and

dry, similar to an old squamous eczema. Only at the margin of such diffuse patches we find several rows of the above-described conical primary efflorescences of lichen ruber, crowned with scaly tips.

With such a spread of the process, which preserves its character unchanged, from many points simultaneously, in the course of one or more years the disease may become general and uniform—lichen ruber universalis. In such a case the skin from head to toe appears reddened, thickened, furrowed by the deepened normal lines of the skin, covered with numerous thin scales; the skin of the face is dry, fissured, and scaly, the lower eyelids ectropic, the upper lids pendulous, the scalp scaly, the hair thin and falling (effluvium capillorum). Later the hair of the beard and body is likewise lost. Motion at the joints is restricted and painful, owing to the thickening of the skin, which is marked with rhagades; the palms and soles usually contain a thick ridge of epidermis which keeps the fingers strongly flexed; the nails of the fingers and toes are thickened, brittle, and opaque. The patient feels very ill, is often troubled with intense itching and sleeplessness; there is impaired nutrition and continual chilliness. Such an intense degree of lichen ruber may persist for several years without undergoing spontaneous involution, at least in our experience. Under the influence of so serious an affection the body gradually emaciates and the patient dies either of inanition or an intercurrent disease.

Lichen ruber planus (lichen planus, Wilson). Here the eruption consists altogether of flat nodules which are not scaly and from the start tend to form groups and patches; the nodules have a peculiar waxy lustre and cupped appearance. They range in size from a millet seed to a pinhead, and even much smaller, barely the size of a pin point. The larger, fully developed ones are brownish or pale red, sometimes quite pale, with a hair-like red border at the base, with a waxy lustre, roundish or polygonal, and very firm. Many even of the smallest nodules show in the centre a minute depression, as if made by a needle puncture, which may appear as a shallow umbilication or a fine point. The nodules thus described are at first irregularly disseminated and appear chiefly on the flexor side of the elbow or wrist joint, in the popliteal space, on the glans penis, the scrotum; often, too, first on the palms and soles or the dorsum of the hand, or anywhere on the trunk, the extremities, the fingers, the vermilion border of the lips, the eyelids, and the cheek.

Very early the nodules form streaks or, following the arrangement of the follicles, circles on the trunk; more frequently, and subsequently at most points, especially the legs, they are crowded together like a mosaic. As the older central efflorescences sink down and become dark brown, while at the periphery a fresh circle forms

of flat, umbilicated nodules with a waxy lustre, patches result which range in size from a lentil to a penny or even a dollar, and present a peculiar appearance, like a dark gem in a setting of pearls. The larger, older patches are distinctly sunken in the centre (atrophic), of a livid to sepia-brown color. Finally, larger surfaces may be uniformly covered with the eruption, the skin then appearing diffusely brownish red, thickened, and granular, looking and feeling like shagreen leather. Upon such places alone distinct scales may form, but not on the several nodules and patches. They never change into vesicles or pustules.

On the mucous membrane of the cheek, the tongue, the hard palate, and the border of the lips lichen ruber planus occurs in the form of hard, punctiform or fissured, silver-gray patches ; in one case I have even, like Touton, observed it there primarily, before the implication of the common integument.

The course and duration of the disease are extremely chronic. Many nodules disappear after some weeks, leaving depressions which are at first dark brown, later glistening white and atrophic like cicatrices ; but otherwise the exanthem persists and increases by the continual formation of new lesions.

Contrary to lichen ruber acuminatus, which rapidly increases until it becomes general, lichen ruber planus remains in some cases restricted to definite regions for one or more years. Whether it may persist longer in this way and eventually undergo spontaneous involution I do not know, since all cases coming under observation were treated at once. It is certain, however, that most cases in time become general.

Although these two forms differ from each other in type by their appearance and course, they represent essentially but one process, as I have convinced myself as early as 1876 by clinical observation and anatomical examination. The two forms are often found combined ; for instance, lichen planus being present on the penis, the hands, palms and dorsum, and soles, while lichen acuminatus predominates on the trunk ; moreover, the nodules of lichen ruber acuminatus in undergoing involution (under medical treatment) assume the exact appearance of the efflorescences of lichen ruber planus. Basing on this view, I then proposed to call the form first mentioned, which was described by Hebra, lichen ruber acuminatus ; and the second, which Wilson (1869) originally termed lichen planus, lichen ruber planus.

As regards the influence of the disease on the general health, it seems to be throughout less than that of lichen acuminatus. In but one case of lichen planus have I observed rapidly progressing emaciation, sleeplessness, dimmed sight, and headache, which conditions yielded only to treatment. As a concomitant symptom of lichen

ruber planus itching should be mentioned, which is at times moderate, but sometimes so intense as to disturb sleep for a long time. The itching ceases only with the general involution of the exanthem under treatment.

The *prognosis* in lichen ruber is unfavorable, in so far as the disease, if left to itself, is not recovered from, but becomes general and finally leads to fatal marasmus. This is true of lichen acuminatus in general and also of lichen planus universalis. The first fourteen

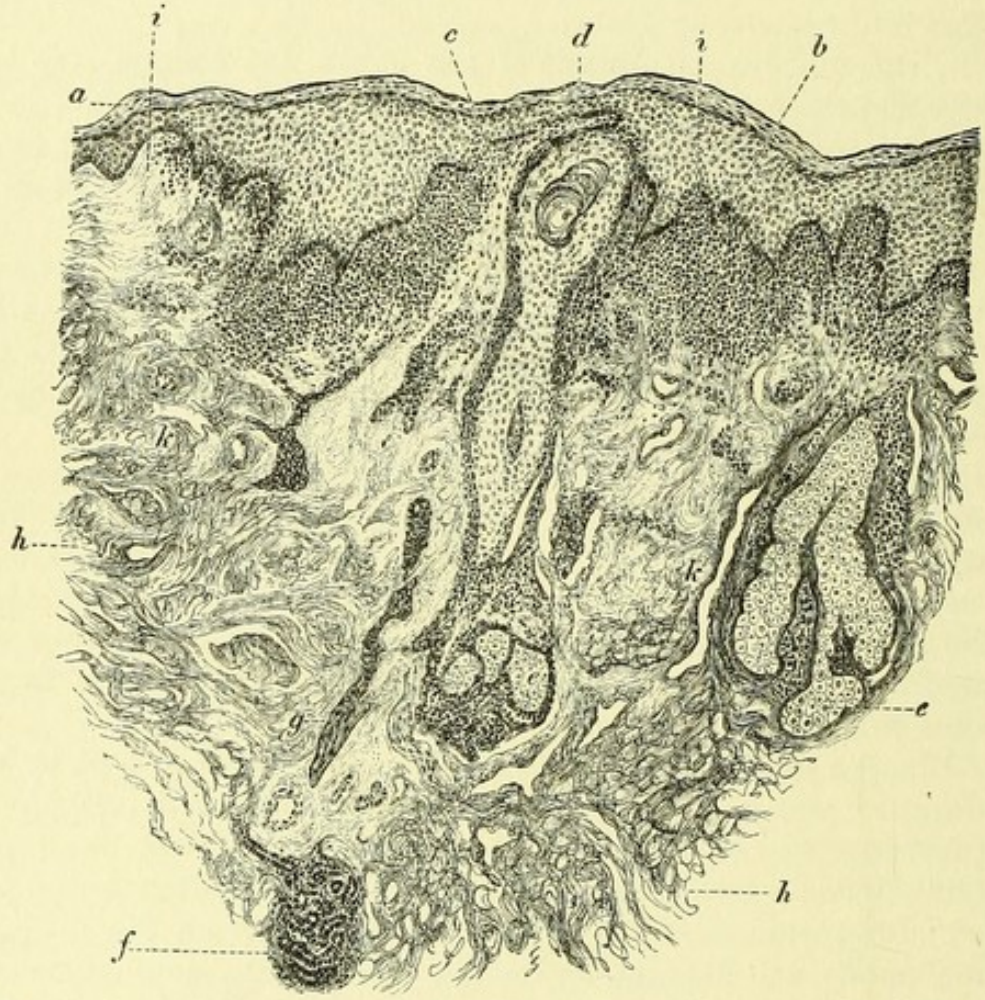

FIG. 28.—LICHEN RUBER PLANUS. PRIMARY NODULE, VERTICAL SECTION. (LOW POWER)

a, epidermis; *i i*, limits of the nodule within which the rete, *i*, is widened. Cellular infiltration of the papillæ and the upper corium at *b*, around the vessels along the hair follicle, *c*, the sebaceous glands, *e e*, and the efferent duct of the sweat gland, *g*; *f*, normal coil gland; *h h*, normal connective tissue of the corium with blood vessels, *k k*, free from cellular infiltration.

cases observed by Hebra succumbed to the disease. But since, thanks to Hebra, we now possess an effective mode of treatment, we may give a favorable prognosis in lichen ruber, as we are able to cure the patient with certainty. It is true, however, that both I and others have observed one or more relapses after months and even after five years.

As to the *cause* of lichen ruber we are still completely in the

dark. We cannot inculpate any constitutional factor, since all cases thus far observed occurred in persons otherwise in good health. The disease, moreover, is neither infectious nor hereditary. But, as in the case of psoriasis, when the disease is present a cutaneous irritation, a pin or other scratch on the skin, may be followed by a more rapid development of nodules about and along it. This explains the occasional occurrence of the nodules of lichen on the extremities in parallel lines corresponding to the scratch marks, and hence I cannot agree to conclude from their arrangement parallel with the course of the cutaneous nerves that the disease has a nervous cause (Köbner), nor to distinguish a separate type as lichen ruber neuroticus (Unna) in cases of more marked itching and general irritability.

Lassar found in lichen ruber minute bacilli in the lymph spaces of the cutis, but the discovery thus far lacks confirmation.

Of our lichen ruber patients two-thirds were men and one-third women. The majority of the cases occurred in persons between the tenth and the fortieth year. I have observed the disease once in a child of eight months, twice in children between three and four years, and twice at the age of seventy.

Lichen ruber planus is rather frequent, since I see from twenty-five to thirty cases every year, some among them being mixed forms. Lichen ruber acuminatus, however, is so rare that many experienced assistants and other physicians have never seen a case. This fact probably explains some of the misunderstandings regarding the disease that have arisen in recent years. Once I have observed lichen ruber associated in the same patient with psoriasis and once with an annular papular syphilide. In a few cases I saw pea-sized and larger vesicles occur together with the newly developing lichen nodules; others (Unna, Hans Hebra, Hallopeau, Lavergne, Róna) observed them upon nodules undergoing involution, so that for some weeks the appearance might resemble pemphigus.

Last year, in one patient at our clinic, there occurred, simultaneously with the first acute eruption of lichen ruber planus, numerous vesicles scattered over the whole body. They were associated with fever and continued to develop for three or four weeks. The sudden cessation of the vesicular eruption showed that the latter was not an accidental (pemphigus) complication, but was to be interpreted as a peculiar mode of onset of the lichen process.

The meaning I intend to convey is that some of the alterations on which the process is based may at times come into the foreground in so prominent a manner as to change the clinical picture in a way that would readily confuse the observer, by the occurrence of an atypical form.

Thus the hyperæmia may cause intense and punctiform or diffuse redness, and even infiltration (dermatitis—lichen ruber scarlatini-

formis, Hallopeau), on which nodules appear only subsequently; or else the hyperæmia leads at the same time to a diffuse serous infiltration of the epidermis, which is detached in large, thick pieces; or it causes an acute, extensive effusion of serum, and hence the formation of isolated or numerous vesicles (lichen ruber pemphigoides), like the above-described exceptional clinical picture; or else there may be punctiform and linear hæmorrhages.

We shall also meet with excesses of the several anatomical changes in other directions, and become acquainted with their altering influence upon the typical clinical picture. These can be understood only when the anatomical changes are known.

No positive data which would explain the fatal marasmus have been discovered at the post-mortem examination of patients dead of lichen ruber.

Now, as regards the *anatomical* alterations in the skin, it is obvious that we shall find some diversity of opinion. The investigations of Hebra, Neumann, Biesiadecki, myself, Obtulowic were directed to the only form known at the time, subsequently distinguished by me as lichen ruber acuminatus. The later observers named above, and their successors to the present day, studied only lichen ruber planus, for the majority of them have never seen a case of lichen ruber acuminatus. All agree in this, that in lichen ruber acuminatus the hair follicles and their immediate surroundings furnish the chief seat of the disease, which manifests itself in vascular dilatation and perivascular and focal cellular infiltration of the perifollicular layers of the cutis. This was found associated with a hyperplasia of the cells of the outer root sheath in the lower portion of the hair shaft, conical processes of hyperplastic cells projecting from the root sheath, the hair follicles becoming consecutively distended; there was also cellular infiltration of the papillæ in the neighborhood of the follicle, and proliferation of the superincumbent rete. There is nothing characteristic of lichen ruber in this finding, for the conical processes of the root sheath especially have been observed also in other chronic inflammatory processes of the skin, such as prurigo (Derby), chronic dermatitis, and chronic eczema. Similarly affected, but not so typically corresponding to the follicles and sebaceous glands, is the superior layer of the cutis and the papillary layer in lichen ruber planus (Fig. 28). At the umbilication of the several nodules in lichen ruber planus, the papillary layer is atrophied to an extent involving several papillæ, and Biesiadecki has called attention to the fact that this point does not correspond to the opening of the hair follicle, but to the attachment of the respective arrector pili muscles, which he supposed to be in a state of permanent tetanus. It is certain that, early in the development of lichen ruber acuminatus, at times the skin of the whole body shows a condition

like that in lichen pilaris—*i.e.*, a prominence of the hair follicles through contraction of the arrectores pilorum. The papillæ surrounding the atrophic and later cicatricial-looking centre of the several nodules show dilated meshes and vessels, together with cellular infiltration; they regain their normal condition.

These anatomical findings do not give sufficient explanation of the nature of the process. I think, however, that we are by no means justified in considering the two forms as separate processes by giving a different interpretation to the histological alterations; some looking upon the formation of scales (hyperkeratosis) in lichen ruber acuminatus as the primary feature, and the hyperæmia and cellular infiltration as secondary (Besnier, Robinson, H. Hebra, and others). This has given rise to unspeakable confusion, since some authors accept as lichen ruber only lichen ruber acuminatus, others only lichen planus (Wilson).

I have always in both forms of lichen ruber, as in psoriasis (page 304), considered the accumulation of scales, the "hyperkeratosis" or "parakeratosis," not as the primary feature, but as the physiological sequel of the chronic inflammatory and infiltration process, and find that of late more and more investigators accept this interpretation (Kromayer, Max Joseph, and others). Assuredly we have to deal here with a serious and specific trophic disturbance, a peculiar variety of degenerative metamorphosis of the inflammatory product, the emigrated and proliferated cells of the cutis and epidermis, which manifests itself in the marked local tissue alteration (atrophy) and the subsequent general marasmus. Possibly Biesiadecki's statement regarding the colloid degeneration of the walls of the papillary vessels has an important bearing on this point.

As stated above, one or the other tissue alteration may at times come so prominently into the foreground as to affect a corresponding clinical change, thus transforming the typical picture of lichen ruber in a striking and confusing manner, which can be understood only if interpreted in this way. So are explained the above-mentioned cases of diffuse dermatitis of a desquamative and pemphigoid form by the acuteness and intensity of the hyperæmia on which the process is based. The following is the history of a case of a unique form of lichen ruber planus, which I have termed lichen ruber moniliformis:

A man, aged forty-five, presented a dense eruption covering the neck, the acromial and axillary regions, the bends of the elbows and popliteal spaces, the abdomen and the gluteal region. The efflorescences projected in the shape of threads, ridges, and sausage-like swellings. They were red in color, with a yellowish tinge on their crests, smooth and very firm here and there, but usually notched at regular intervals so as to resemble a coral necklace. The eruption, while similar to xanthoma and keloid, looked like hypertrophic

cicatrices from burns. The general direction of the efflorescences was parallel to the longitudinal axis of the body, but they were joined together everywhere, especially in the bends of the joints as well as on the neck, by oblique and transverse ridges, here and there forming a very close network. The immediate neighborhood of these networks of spherical swellings, and the fields of skin between the meshes, were covered with dark-red or brownish characteristic nodules of lichen ruber planus, which were partly grouped in dense masses, partly arranged in streaks or disseminated. The whole region of the nates represented a diffuse, sepia-brown spot, the neighboring cutis, toward the sacrum and the posterior surface of the thighs, containing numerous nodules of lichen, between which only punctiform pigment spots were present. The abdomen, while similar, showed numerous wheals resembling keloid, but largely bearing dendritic markings. The raphe of the scrotum was occupied by a single thick wheal, its right half by smaller ridges.

On closer inspection it was found that in this case the small lichen nodules had developed to a larger size up to that of a pea, which were faceted and firm, and that such a hyperplastic enlargement of these nodules when arranged in rows had resulted in the notched, long wheals. The histological examination, too, showed that the anatomical constitution of the large nodules and streaks was like that of the small lichen nodules. Analogous forms were subsequently observed by Róna and Dubreuilh (lichen ruber en bandelettes anastomosées, réticulaire).

This occurrence proves at the same time that in lichen, as in psoriasis, the cell infiltration, the inflammatory process in the corium and the papillary layer, represents the essential factor of the process, and that it is quite incorrect to include it among the epidermidoses as Auspitz did. Normally the infiltration of the nodules rapidly undergoes a fatty, perhaps colloid or waxy metamorphosis and disappears, leaving the affected spots atrophied. The yellowish, lardaceous coloration of the larger, older, and some of the smaller wheals, which produces a deceptive likeness to xanthoma, is probably the expression of the fatty or waxy metamorphosis. In this exceptional case, however, the infiltration, usually confined to minute patches, developed to pea-sized nodules.

I have also observed an abnormal termination of lichen ruber in the opposite direction. In a woman aged forty-five I saw in the cervico-clavicular region, the back, and the right thigh extensive, flat, and slightly depressed reticulated cicatrices with a whitish lustre and a reddish-brown border. They reminded me of the cicatrices of lupus erythematosus. After several months' observation it happened that fresh lichen ruber nodules appeared on the adjoining egions and other parts of the body, and it became clear that I had

to deal with lichen ruber, and that in this case, instead of small patches of cutaneous atrophy and sepia-brown coloration which usually result, the lichen ruber had terminated in extensive, punctate, and reticulated glistening white cicatrices.

Finally, through excessive hyperkeratosis—*i.e.*, proliferation and accumulation of the epidermis of the follicles and sebaceous glands—or possibly through the combination of intense cell infiltration of the papillary layer with hyperplasia of the epidermis, appearances may result which closely resemble ichthyosis and would justify the term "keratosis follicularis" (Morrow, White). On the palm and sole the patches somewhat resemble tylosis, flat or verrucous and corn-like, while the nodules may be recognized at their margins; on the forehead, cheek, chin, and other points rich in follicles, such as the eyebrows, scalp, and pubes, as disseminated or crowded, warty nodules with firm or crumbling cone of fatty scales which evidently belongs to a dilated follicle; the base being usually brawny, brownish-red to sepia-brown in color.

The *diagnosis* of lichen ruber, owing to its pronounced clinical character, is certain to be made; still, as the disease is rare, it is rather difficult to the inexperienced. This is true especially of lichen ruber acuminatus.

In the stage of the formation of disseminated nodules it may easily be confounded with psoriasis punctata or papular eczema and with a number of morbid processes which are partly very rare and little known, partly open to discussion in general and in special cases. These are certain forms of toxic eczema with the formation of follicular nodules and of extensive papular and cup-shaped scaly crusts, also acute forms of keratosis which are exceedingly rare, and finally the diseases to be discussed hereafter—namely, "pityriasis rubra pilaris" of Besnier (Devergie) and "psorospermosis cutanea" of Darier and others. Still more difficult is the differentiation of lichen ruber acuminatus universalis from general psoriasis, pityriasis rubra, squamous eczema, and other processes which present the characteristics of general redness, infiltration, rugosity, and desquamation of the skin, with degeneration of the nails and hairs and ectropium.

Here, as in lichen ruber planus, the student or practitioner is advised to bear in mind the type and not the previously described excessive and atypical forms—*i.e.*, the very characteristic miliary and larger, umbilicated, firm nodules with waxy lustre and the patches surrounded by them.

The diagnosis is most difficult when the lichen ruber begins on the palm and sole, because the violent itching suggests eczema; the brawniness, local ichthyosis, tylosis, and psoriasis vulgaris and syphilitica; and because, owing to the thickness and tension of the epidermis, the nodules here do not project, are not markedly umbili-

cated, and, in general, little characteristic. Not easily recognizable, as well, is the form beginning on the buccal mucous membrane, cheek, tongue, and lips, if no efflorescences have as yet appeared on the common integument. The isolated, dry, firm, sepia-brown, slightly desquamating, and very itchy patches on the legs, which often persist for years in spite of every treatment, are also difficult to distinguish from psoriasis and eczema. In general, however, the diagnosis of typical forms of lichen ruber planus is rarely in doubt.

In lichen ruber universalis the diagnosis from general psoriasis is most difficult. As a rule, however, in lichen ruber the formation of scales is relatively slight and the skin is considerably thickened, while in psoriasis we find numerous and easily detached scales, which latter at other points form thick accumulations. Moreover, psoriasis, even at its highest degree of development, undergoes temporary involution, so that some healthy islands of skin may be visible. Finally, in psoriasis, even if general, the palm and sole are usually not affected, or at least will be rarely so intensely diseased as in lichen ruber.

Chronic general eczema is more easily excluded, because the characteristic features of this affection, such as weeping, etc., are found at many points.

Pityriasis rubra universalis will be more easily differentiated by the absence of infiltration of the skin, which, on the contrary, appears thinned, even atrophic, and produces only very thin lamellæ and branny scales.

Lichen ruber planus, with its disseminated or regularly arranged umbilicated nodular eruptions and patches depressed in the centre, is most frequently mistaken for a papular syphilide, all the more because the glans penis, as a rule, also shows some efflorescences. I must refer to the above-described characteristic features of these polygonal nodules and patches, their waxy lustre, the slight umbilication of each efflorescence, and the dry quality of the latter, even when located on the genitals. Still, the correct diagnosis of such a morbid picture requires great care. The diagnosis becomes particularly difficult when the lichen ruber begins on the palm, because then the nodules do not project markedly and are but little characteristic. A confusion with syphilis of the palm can be avoided by the granular appearance of the wheals and the violent itching, but the difficulty is greater with eczema and psoriasis.

The *treatment* of lichen ruber is clearly defined. While in the first fourteen cases observed by Hebra all the various internal and local remedies proved ineffectual and failed to avert the fatal issue, every case that has since occurred has recovered under the consistent use of arsenic as devised by Hebra. Now we can without hesitation

promise complete recovery to every lichen ruber patient, excepting only the highest degree of marasmus after the disease has become general.

Young children I prefer to treat with Fowler's solution, two drops per day and increased very gradually. Adults are treated with Asiatic pills, or hypodermic injections of Fowler's solution or arseniate of soda, as described under the treatment of psoriasis.

Before the lapse of six to eight weeks—that is, until the patient has reached from two hundred to five hundred pills—no improvement is to be observed as a rule ; a number of fresh eruptions continue to occur, and but few of the older efflorescences undergo involution. After from five hundred to six hundred pills the involution becomes more marked and the formation of nodules more sparse. But the latter occur even in the last stages and after the old eruptions have completely disappeared. For this reason we continue the arsenic in moderate doses, about six pills per day. for three or four months after the disease seems to have entirely disappeared.

We begin with three pills per day, adding one every fourth or fifth day until eight to ten per day are reached ; this quantity is maintained until the involution of the process is nearly complete, then it is reduced to six and is kept up for three or four months, counting from the apparent recovery.

In moderate cases of lichen ruber a total number of eight hundred to fifteen hundred pills suffices, but we have gone as high as three thousand, and I know one patient whose general eruption did not fully disappear until, in an uninterrupted course of treatment lasting two years, he had taken about forty-five hundred Asiatic pills.

The report of such instances can hardly be called superfluous, because young practitioners are either frightened by such large amounts or would be frightened by others unless reassured by the experience of older physicians.

How thoroughly habituated the system may become to a methodically increased consumption of arsenic I have had occasion to observe at the meeting of naturalists in Graz (1875). Two arsenic eaters were presented (by Dr. Knapp), each of whom took a piece weighing four and eight grains respectively, and stated that he repeated this every two weeks. By following the above-described method—*i.e.*, the gradual increase and decrease, and remaining at the dose where the effect becomes visible—the patient can suffer no injury.

Subcutaneous injections of Fowler's solution usually act rapidly. Fifteen to twenty injections of a full dose of Fowler's solution (4.0 in distilled water 20.0), extended over twenty to thirty days, sometimes suffice to dispel the eruption ; or else arseniate of soda 0.1 ad aq. dest. 10.0, increased from one-fourth to one syringeful. But after

the treatment by injections I have more often observed an œdema of the eyelids, sensations of heat and burning in the skin, and intense lentiginous and chloasma-like pigment spots of the skin, as well as rapid relapses, than by ingestion.

Heroic doses cause a speedy disappearance of lichen ruber. One of my patients by mistake took within four weeks six hundred Asiatic pills—*i.e.*, 4.5 grammes of white arsenic—without experiencing any ill effect except a peculiar uneasiness in the abdomen. The lichen ruber, which had existed for eight months, had entirely disappeared in about two weeks. Nevertheless we must avoid giving such toxic doses. For some patients it will be best to give minimal doses, say of the Asiatic pills at most five per day, to be reduced to two again gradually.

As regards the often very troublesome itching and the sleeplessness, they are best treated by local measures, such as painting with carbolic or salicylic acid (1 : 40 of alcohol, with 1 gm. of glycerin), dusting with starch, steam and douche baths, inunction with indifferent fats or ointments containing carbolic acid, salicylic acid, or zinc. But, as a rule, the itching is not alleviated until the process tends largely toward involution.

Painting with tar has proved but little effective against the itching or the process as such, and the same remark is true of sulphur, soda, alum, and sublimate baths, etc.

American authors recommend for lichen ruber, especially lichen ruber planus, acetate of potash in doses of 5.0 ad aq. dest. 150.0 per day, as especially effective, the eruption being said to disappear completely within from three to six weeks. I have been unable to confirm this favorable action.

Unna has cured a whole series of lichen ruber cases within three weeks, without any internal medication, simply by the application of an ointment containing : hydrargyri chloridi corr. 1 0, acidi carbolici 20.0, unguenti simplicis 500.0. The affected skin is rubbed with this twice a day and covered with wool, or the patient is placed in bed. Bockhart and Touton confirm the statement.

I have followed this method exactly in cases of lichen ruber acuminatus and lichen ruber planus, both general and regional, in hospital patients who were treated in bed and in private practice, but unfortunately I have never noticed the least effect from this ointment.

But from a ten-per-cent pyrogallic acid ointment, chrysarobin, or emplastrum hydrargyri we may at times observe rapid desquamation and flattening of the lichen ruber planus patches. Much more frequently, however, the thick patches occurring so commonly on the legs resist for months and years all forms of local and constitutional treatment when the general eruption has long disappeared.

In several cases I have witnessed relapses after a few months up to one or more years. One case had as many as four relapses.

PITYRIASIS RUBRA PILARIS, Besnier (*Pityriasis rubra*, Devergie).

Under this name Besnier, in a very thorough paper (1889), described a form of disease as a morbid process *sui generis* which was first reported by Rayer as a peculiar form of "psoriasis universalis," and later by Devergie, by Richaud (1877), and Brocq (1884) as pityriasis rubra. According to the conception of the latter authors, as well as of Besnier, the disease is represented by a series of cases noted and published, partly formerly, partly since, by various authors, such as Hutchinson, Hillier, Tilbury Fox, C. Boeck, and others, but especially by French authors from the material derived from the Hôpital St. Louis. Moreover, this morbid process is presented in a very instructive form by a considerable number of plaster casts, from Baretta's skilful hand, in the museum of the Hôpital St. Louis.

At the International Dermatological Congress in the year 1889, contrary to the opinion of most of the members who spoke on the subject, I decidedly took the stand that neither from the description of Besnier and the other authors nor from the examination of Baretta's casts could I distinguish this disease termed "pityriasis rubra pilaris" from that which I have called lichen ruber acuminatus—*i.e.*, from the form originally described by Hebra as lichen ruber; in other words, that I must hold pityriasis rubra pilaris of Besnier as identical with lichen ruber acuminatus.

All that I have since read, heard, and gathered in discussions on the subject has not tended to change this opinion; and it is all the more difficult for me to understand the violent attacks directed against the position by many dermatologists, because Besnier himself, in the paper mentioned above, particularly acknowledges that his pityriasis rubra pilaris is identical with the forms described by Robinson and Taylor as lichen ruber, but which these authors had cited as lichen ruber acuminatus; and, furthermore, because Brocq likewise, in one part of his paper on dermatitis exfoliativa, asks himself: Might not these two affections be identical?

In fact, the cutaneous symptoms described as peculiar to pityriasis rubra pilaris agree so completely with those given under lichen ruber acuminatus that I could record the former only by repeating the latter.

On the other hand, according to the description of Besnier and the authors agreeing with him, the course of pityriasis rubra pilaris differs materially from that of lichen ruber acuminatus in so far as the former appears to be much more benign. For, while the eruption in pityriasis rubra pilaris also often extends over many years, is likely to relapse when apparently cured, and the patients are much troubled

with itching and become debilitated, many having recovered only under a methodical arsenical treatment, yet not a single case has ended fatally, and some patients recovered temporarily or permanently in a relatively short time, either spontaneously or under a mild external treatment and without the use of arsenic.

In this respect, according to my experience in recent years, it may eventually be found that lichen ruber acuminatus indeed at times runs a more rapid and benign course than that observed formerly by Hebra and ourselves. This would completely harmonize the opinions now at marked variance with each other.

It has been asserted that there is an essential difference between the two processes, because their histological bases are thoroughly unlike, since lichen ruber acuminatus springs from an inflammatory basis, while pityriasis rubra pilaris is said to begin as a hyperkeratosis in the epidermis. But in my opinion these conditions do not vary because of different processes, but in accordance with the concrete case and the several regions of the skin—that is, they are identical for both processes. Just as in the case of "pityriasis rubra pilaris," so excellently described by C. Boeck, and in other similar cases, the most intense inflammatory symptoms of the skin may precede the nodular eruption or may be wanting, while in other cases or in different parts of the body the epidermal cones, the hyperkeratosis, may develop without obvious or histologically marked inflammatory processes (Neisser) of the papillary layer, so may the like be observed in lichen ruber and will be found described by us; and exactly as lichen ruber acuminatus may occasionally or regionally heal without leaving a trace on the skin, so may pityriasis rubra pilaris disappear without residual depressions (Boeck, Neisser).

While on this question of the identity of lichen ruber acuminatus with pityriasis rubra pilaris many may still be in doubt or decidedly opposed—and these I hope will be convinced by further observations—stress must be laid on the fact that there is absolutely no clinical relationship between pityriasis rubra of Hebra (page 318) and pityriasis rubra pilaris.

A position which is still unsettled in the system is occupied by the morbid processes described in recent years as *keratosis follicularis* and *psorospermosis follicularis*. Their clinical picture likewise recalls that of lichen ruber acuminatus, but we shall treat of them in another place.

LECTURE XXIV.

2. PRURIGINOUS DERMATOSES—ITCHING ERUPTIONS.

ECZEMA.

DEFINITION—POLYMORPHISM AND VARIABILITY OF THE SYMPTOMS—TYPICAL COURSE OF ACUTE ECZEMA—CHRONIC ECZEMA—ANATOMICAL LESIONS—SPECIAL FORMS OF LOCALIZATION—IMPETIGO FACIEI—ECZEMA MARGINATUM—DIAGNOSIS.

ECZEMA (ἐκ-ζέω, to boil up, to effervesce), or salt rheum, is a disease of the skin which is very often acute, but usually chronic. It occurs in the shape of irregularly scattered or closely aggregated papules, vesicles, and pustules, or as diffuse redness and swelling of the skin. In the latter event the surface is scaly, covered with papules, vesicles, or pustules, and weeping or covered with yellow, gummy crusts.

In addition to this polymorphism of eczema, the symptoms are extremely variable. Hence many physicians and writers are not convinced of the relationship of all the forms mentioned, but regard many of them as special diseases.

We shall arrive at a comprehensive and harmonious conception of eczema if we consider all the factors—the appearance, course, causation, and history of the entire process. It then becomes evident that all the morbid forms mentioned very often exist upon the skin at the same time; that, during the course of the disease, the various lesions of the integument are in a state of constant change from one variety to another; and that we are always able to produce artificially in all parts of the skin and in every individual all the different forms, together with their polymorphism and their transitions.

We will first consider the processes which are exhibited by the skin after artificial external irritation by heat, sulphur ointment, tincture of arnica, turpentine, etc.

The kind, intensity, and duration of the morbific action and the individual irritability of the skin determine whether one or the other lesion of eczema develops—viz., papules or vesicles, diffuse redness with scaling or weeping. The irritability of the skin and the single

occurrence or repetition ot the irritation determine whether the eczema will be acute or chronic.

If the irritative action is mild, irregularly scattered, pale or deep red, firm, itching papules as large as a pin's head soon make their appearance (eczema papulosum). Their number continues to increase during the first few hours or days Then the papules grow smaller and disappear. If the irritation is more severe the papules are converted into clear vesicles by an increase of their serous contents (eczema vesiculosum). These may also disappear in a few days by the evaporation and absorption of their contents. If the irritation is still more prolonged or intense, the skin becomes diffusely reddened, swollen, hot, painful, and œdematous (eczema erythematosum). This condition may also undergo resolution in a few hours or days, leaving behind moderate exfoliation and dark pigmentation. Finally, in the highest grades of irritation the diffusely reddened and swollen skin is covered by closely aggregated vesicles (eczema vesiculosum), a majority of which soon burst or are scratched off and exude their fluid contents in clear drops. This constitutes weeping eczema (eczema madidans).

If the tops of the vesicles are removed mechanically by rubbing or are washed off, the skin is exposed as a dark-red surface, covered merely by the rete, and sprinkled with small depressions which correspond to the base of the destroyed vesicles ("état ponctueux" of Devergie). The fluid of this eczema rubrum now exudes more freely. It is light yellow in color, sticky, of a neutral reaction, and deposits flocculent albumin on boiling or the addition of nitric acid. The fluid is blood serum and is not a pathologically constituted secretion. It dries into yellow, gummy crusts, and, like semen, stiffens the underclothing which it impregnates.

With the stage of vesiculation the eczema reaches its anatomical height; with the weeping it reaches its clinical acme. It remains in this condition for a few hours or, if the irritant is renewed, for several days, and then resolves. At first the eczema fluid dries into yellow crusts, which become yellowish-brown from admixture with blood (eczema crustosum); beneath these secretions the fluid becomes greenish and purulent (eczema impetiginosum). Here and there the flabby crusts burst, the purulent fluid escapes, and the weeping red surface of the papillæ becomes visible. In the meantime the inflammation and swelling diminish, the more scanty secretion is no longer able to raise the crusts, which grow dry, hard, and firmly adherent. Under their protection a firmly adherent epidermal covering forms, and finally the crusts are detached. The diseased skin is then exposed, little swollen but still hyperæmic and desquamating (eczema squamosum). Finally the last remains of congestion and desquamation are lost. The skin is normal in color,

except that there may remain a somewhat darker pigmentation for a time. It is followed by complete *restitutio ad integrum.*

A moderate eczema of this kind, which extends, we will say, over one forearm, runs its course in two to four weeks. The symptoms described constitute, as a whole, an acute eczema.

From this description various important data may be gathered :

1. That the disease begins with punctate or diffuse redness and swelling of the skin (eczema erythematosum) or itching papules (eczema papulosum), but that it may not develop beyond these lower grades ;

2. That the stage of vesiculation (eczema vesiculosum) and weeping (eczema rubrum, madidans) is the acme of the process ;

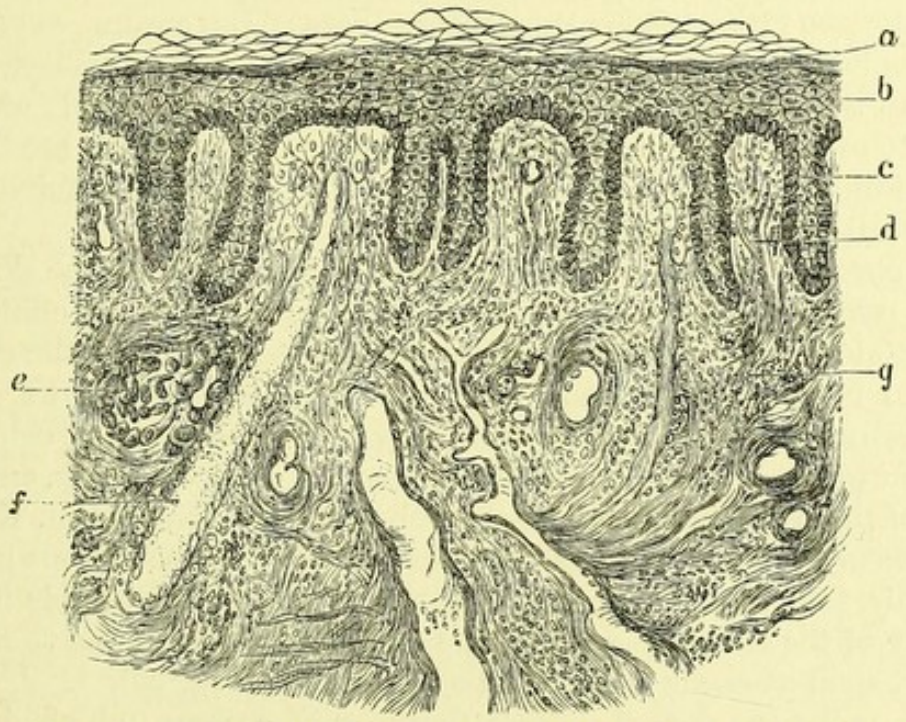

FIG. 29.—CHRONIC ECZEMA. VERTICAL SECTION THROUGH SKIN OF UPPER ARM. (LOW POWER.)

a, epidermis ; *b*, rete with thin, corneous covering, the basal cells deeply pigmented, *c*; *d*, papillæ, widened and increased in size, in them, as in corium, *g*, cell infiltration and dilated vessels ; *f*, hair follicle, and *e*, coil gland, in process of destruction.

3. That the formation of crusts (eczema impetiginosum and crustosum) and the stage of the red, scaly surface (eczema squamosum) are forms of resolution ;

4. That acute eczema runs a cyclical course.

The changes described constitute the essential symptoms of eczema and are found in the most varied combinations as regards localization, course, complications, causation, etc.

In chronic eczema these symptoms do not run a cyclical course in one outbreak, but persist for a long time or recur repeatedly. Exacerbations and remissions may occur upon certain parts of the

skin, or for years the eczema may appear, now here, now there (eczema recidivum). These forms are the most polymorphous and variable, because all possible degrees of development and resolution may be present at the same time; here papules or vesicles, there red, scaly patches or weeping patches, again pustules, rhagades, and pigmented spots.

Anatomically eczema in all forms and stages constitutes an inflammation with predominance of serous exudation (G. Simon, Hebra, Wedl, Kaposi, Neumann, Biesiadecki). I need not enter into the histology of the papules and vesicles, because the changes in the epidermis, papillæ, and corium are the same as in erythema papulatum and herpes (*vide* page 215, Fig. 21). The more intense the local inflammatory phenomena (eczema rubrum, madidans) the more the exudation affects the deeper layers of the corium, extending into the layer of adipose cells. The connective-tissue corpuscles proliferate, the meshes of the tissue become more distended, and the exudate cells are increased. Within the rete all changes are found, from simple swelling and separation of the cells into a network to proliferation and purulent destruction.

In chronic eczema which occupies the same part of the skin for years, permanent changes develop in the tissues and are characterized, clinically, by darker pigmentation and thickening of the epidermis and corium, and by more pronounced cutaneous furrows. Histologically these changes are shown by a dense deposit of cells and pigment in the corium, especially around the dilated vessels, enlargement of the papillæ, dilatation of the lymphatics (Neumann, Klebs), sclerosis of the connective tissue, obliteration of the sebaceous glands and hair follicles (Wedl), degeneration of the sweat glands (Gay), atrophy of the fat cells—in fine, the changes of degenerative hypertrophy, such as occur in elephantiasis arabum (Fig. 29).

We will now describe those features of eczema which depend upon the accompanying circumstances, such as the causation, localization, extent, but particularly the acute or chronic course.

ACUTE ECZEMA.

This affection appears upon one or more parts of the body and runs the course already described. It often extends by continuity from the original site; in the centre it usually reaches the intensity of an eczema vesiculosum, rubrum, or madidans, while at the periphery there are only a few papules, vesicles, or red patches, separated by healthy patches of integument. New outbreaks of the disease may occur in remote parts of the body.

With the occurrence of an acute eczema the integument is morbidly affected in such a way that it is attacked by eczema after slight irritation—the friction of the underclothing, scratching, the heat of

the bed, or even reflex vascular changes. This is particularly true of the face (ears, eyelids), which is at once attacked by eczema in a reflex manner when an acute outbreak of the disease has occurred upon a remote part of the body—for example, the scrotum.

The outbreak of acute eczema is usually preceded by chilly sensations, even a pronounced chill and fever; these symptoms usually accompany the process to its acme, accompanied by insomnia, restlessness, and gastric symptoms. They only subside when the exacerbations cease. During the period of resolution sleep is still disturbed by itching.

Acute eczema occurs very often as eczema papulosum, due to the heat of the sun or to sweating; in infants it is often a general eruption, corresponding mainly to the follicles, and hence appears figured (eczema lichenoides, figuratum s. folliculare). It often accompanies other itching diseases of the skin (prurigo, scabies). Eczema erythematosum is found mainly on opposing macerating folds of skin (eczema intertrigo). The most frequent form of acute eczema is moist.

Various peculiarities depend upon the special localization. The most frequent sites are the joint flexures, the cutaneous surfaces of the genitalia which are exposed to the action of sweat, and of the pendulous female breast, and particularly the face, with the ears and scalp.

Acute eczema of the face and scalp, usually preceded by a chill, begins with a feeling of burning in the eyes (conjunctivitis), with redness and swelling of the face; the eyelids are œdematous and can be opened very slightly or not at all; the ears are thickened and stand out from the skull; the lips are swollen, and the tyro is apt to make a diagnosis of erysipelas. On careful inspection it is found that the redness and swelling are by no means so pronounced as in erysipelas and the fever is not so severe. Stupor or symptoms of cerebral compression are never present.

In oblique illumination or on palpation it is found that the skin is covered with little papules which gleam like water (developing vesicles). Within twelve to twenty-four hours these have developed distinctly and burst. The characteristic weeping and crusting then begin. A large amount of fluid exudes, particularly from the ears. The integument of the external auditory canal is swollen to impermeability, so that difficulty in hearing or deafness may be present. Swelling, weeping, and formation of crusts develop very gradually on the scalp and cause agglutination of the hairs.

An eczema of this character lasts three to six weeks, according to the severity and extent of the disease. Even after it has completely run its course a condition of eczema squamosum often remains on the scalp, likewise dryness, thickening, and fissuring of the epi-

dermis in the furrow behind the conchæ. Exacerbations often start from the latter locality. Facial eczema, in general, relapses very often from the most varied causes.

In acute eczema of the hands and feet the vesicles are usually very tense and provided with thick walls. The feeling of tension and numbness of the fingers and the pain are pronounced. The contents of the vesicles often become purulent (eczema pustulosum), followed by considerable œdema, exposure of the corium with consequent sensitiveness, granulations at their edge, and shedding of a few of the nails. Some individuals are especially predisposed to such eczema, and are repeatedly attacked under the influence of habitual hyperidrosis and local asphyxia. After repeated relapses the acute outbreaks run into a chronic form and the disease spreads serpiginously beyond the margin of the member. In children an eczema with large vesicles occurs upon the fingers.

Acute eczema of the penis and scrotum is accompanied by very considerable œdematous swelling and pronounced weeping.

Upon the integument of the joint flexures, genital folds, pendulous breast, and all cutaneous folds which are macerated by mutual contact, acute eczema often occurs as diffuse redness (erythema intertrigo), from which weeping surfaces (eczema intertrigo) are produced by detachment of the epidermis.

This eczema possesses great importance in infants, in whom it develops at the bottom of cutaneous folds, in the neck, or on the inner surface of the thighs. It is often overlooked by the nurse, who is afraid to separate the folds because this causes pain. The dermatitis is not infrequently intensified and results very acutely in punched-out, syphilis-like ulcers, gangrene, phlegmons, croupous and diphtheritic inflammation. In favorable cases these conditions recover with loss of substance and cicatrization. I have also seen a fatal termination at the end of a few days, after eclampsia and collapse.

General acute eczema is a veritable torment both to the patient and the physician. The disease does not extend uniformly from crown to foot, but is composed of numbers of patches of every possible degree and variety, papules, weeping and crusted surfaces, etc., whose peripheries are more or less in contact. As a rule the febrile symptoms are tolerably severe, and every new eruption is followed by an exacerbation. For this reason the patient is usually confined to bed, apart from the fact that walking about while dressed, even if subjectively possible, acts injuriously on account of the rubbing of the clothes and the adhesion of the underclothing. When the process extends over the entire body, two to three months, often a longer period, elapse before complete resolution. The nutrition of the patient is notably impaired by the fever, anorexia, insomnia, and the actual loss of blood plasma.

Lymphangitis and furuncles may develop during the course of the disease. The patient is not entirely restored even after the lapse of several months. Rhagades are left over here and there, in the furrows of the ears, in the flexures of the joints, and may form the starting point of fresh eruptions. The furuncular inflammations recur in certain cases for many months, even one to two years, and finally the skin becomes so sensitive to all external influences which may produce eczema (heat of the sun, sweat, water, etc.) that fresh attacks are very frequent.

CHRONIC ECZEMA.

This disease occurs either as a chronic relapsing eczema, in which the symptoms of acute eczema are constantly present on different parts of the skin, or as true chronic eczema of the same locality. This develops either as the residuum of an acute eczema which has not entirely run its course, or it develops from slight beginnings by persistence and repetition of the attacks.

Chronic eczema exhibits essentially the same phenomena as the acute form, and the changes in the skin are due to the repetition of the inflammatory processes. They consist of thickening of the epidermis and corium, darker pigmentation, degenerative changes, and follicular atrophy.

Chronic eczema may become acute at any moment, and then appears in a weeping or crusted condition. Usually, however, it occurs as an eczema squamosum. The attendant itching is usually very severe and induces violent scratching. This acts as a cutaneous irritant and may excite a fresh eczema.

The localization of chronic eczema exhibits certain peculiarities. It is found most frequently upon the flexures of the joints, the groove behind the ear (usually symmetrically), the scalp, face, male genitals, and the anus.

Chronic eczema of the scalp is very frequent, usually associated with eczema of the face, and occurs as eczema impetiginosum or squamosum. The scalp is covered with crusts or exfoliating scales of epidermis ; after these are scraped off the skin appears red and is moist in places. The scaliness and redness extend to the forehead and back of the neck. Loosening of the hairs and alopecia are constant effects of a long-standing eczema of the scalp. Its occasional exacerbation into the weeping stage causes agglutination and matting of the hair (plica) in women, or, in rare cases, the development of numerous follicular pustules (sycosis capillitii) upon the intensely inflamed scalp. It often lasts many years, is rarer in men than in women and children. In the latter it is often due to head lice. In such cases we usually find islets of eczema upon the vertex and occiput, containing large, dry crusts which cover foul-smelling secretion.

After their detachment the skin is in part red, moist, smooth, and in part covered with red, moist, papillary excrescences, two to four millimetres high, which bleed readily (achor, mucor granulatus, tinea granulata). Of course the evidences of the head lice and their "nits" are also present. Such eczemata are constantly associated with considerable swelling of the cervical glands, and this may lead to an erroneous diagnosis of scrofula.

Chronic eczema of the face is either confined entirely to certain regions or is more marked in such places. The conchæ of the ears usually appear thickened and rigid, the thickened epidermis on the ridges is fissured or covered with crusts, the external auditory canal is partly closed by scales of epidermis. Acute outbreaks occur very often upon the ears. The crusted and scaly eczema of infants (crusta lactea, milk crusts, porrigo larvalis, lactumen) occupies mainly the cheeks, forehead, and ears. Painful furuncles in the auditory canal, and rhagades in the naso-labial folds and at the angle of the mouth, are not uncommon.

A very alarming complication of eczema larvale infantum, which has come under my observation in several cases, is an acute outbreak of numerous vesicles, partly scattered, partly arranged in groups. The vesicles are as large as a lentil, filled with clear serum, and the majority are umbilicated. They look like varicella vesicles, but undoubtedly do not belong to this class. The integument which has been attacked in this manner now appears still more swollen, even tense. The little patients have high fever (40° C. or more) and are very restless. The vesicles develop very acutely (sometimes over night), in large numbers, and often continue to appear, in successive crops, for three or four days or even a week. Those which appeared first undergo desiccation, rupture, and expose the corium, or they become encrusted and fall off. The largest number of these varicella-like vesicles are found upon the already eczematous skin, but smaller groups appear upon the previously intact skin of the neighborhood, upon the forehead, ears, neck, and even the shoulders and arms.

The outcome of this peculiar affection always is, in the majority of instances, favorable ; the exposed surfaces are covered with new integument in two to three weeks. In many places pigment patches are left over, or even flat cicatrices. The previously existing eczema changes its character only in so far as it has been influenced by local treatment. In a child æt. six months, under my observation, death occurred after eclamptic seizures on the sixth day of the disease, when the eruption was everywhere recovering and complete defervescence had ensued.

I am in doubt with regard to the proper term to apply to this varicella-like eruption, which has never been seen by experienced pædiatrists, while ten cases have come under my observation. *Eczema*

herpetiforme seems to be a suitable appellation. I am also ignorant of its causation. I suspect, however, that we have to deal with local contagion by a fungus, which finds a suitable soil in the epidermis that has been loosened by the eczema and by its vegetation produces the peculiar eruption. The cases were all so rapid in their course that no opportunity for microscopical examination has hitherto been obtained. The threatening fever is not due to blood infection, but to the local dermatitis.

Chronic eczema of the nasal mucous membrane is very frequent in young people, combined with scrofulous affections of the eyes; the former is due to irritation of the nasal mucous membrane by the tears. The nares are closed by the eczema crusts, the children breathe with the mouth open, the pharynx is covered with nasal mucus and inflamed. Snout-like thickening of the lips develops as the result of attendant lymphangitis.

In adults chronic eczema of the nasal mucous membrane, often due to chronic coryza, is annoying on account of the crusts and fissures, and often gives rise to sycosis or furuncles and to relapsing erysipelas of the face.

Eczema of the lips occurs in connection with eczema of other parts of the face, especially of the nose. A peculiar form is that which occurs in women of middle age, more rarely in men, and affects mainly the border of the lips. This is fissured, thickened, and covered with hæmorrhagic crusts. This eczema itches intensely, causes frequent acute exacerbations, and is extremely obstinate.

Chronic eczema of the scalp and eyebrows results not infrequently in sycosis (eczema sycosiforme). The margins of the eyelids are often affected, and this is followed by blepharadenitis, unless the latter has given rise to the eczema. At the corners of the eyes it occurs in the form of rhagades. As the result of protracted eczema the lids become thickened and hang lower, so that the palpebral fissure is narrowed.

Nothing need be said concerning chronic eczemata of the trunk, with the exception of eczema of the nipple and mamma. It is extremely rare in men (usually unilateral), but is frequent in women (puerperal women and nurses, with or without scabies). The nipple may swell to the size of the finger and its surface may be red or moist, or it may be covered with thick crusts in which painful and bleeding fissures often develop. At first sight it may resemble carcinoma. It is not infrequently complicated by mastitis.

Eczema umbilici usually affects the retracted umbilicus of fat people, and is due to the accumulation and decomposition of the cutaneous secretions. It is relieved with difficulty.

Eczema of the male and female genitalia is an extremely annoying and frequent disease. In men it affects chiefly the scrotum,

either in places where the skin is permanently in contact with the thigh, or, after lasting for years, it spreads over the entire scrotum, to the penis, perineum, often to the circumference of the anus and the integument extending to the sacrum. A scrotum which has been the site of eczema for ten to fifteen years appears thickened with enormous folds and furrows, scratched here and there, scaly, and slightly covered with crusts. The itching is extremely violent, and as a rule occurs several times a day in paroxysms.

In eczema of the anus the fissures often extend far into the rectum. Defecation is difficult on account of pain. Constipation and diarrhœa alternate. In the course of years the rectal mucous membrane becomes enormously swollen, fissured, and warty, simulating cancer. The annoyance is increased by mucous secretion and occasional hæmorrhages.

Upon the female genitalia chronic eczema attacks chiefly the labia majora, more rarely the labia minora and introitus vaginæ. The integument is thickened and excoriated. Leucorrhœa is usually present, and indeed often gives rise to the eczema.

In the upper and lower limbs the joint flexures are frequent sites of chronic eczema. As a rule, it is symmetrical. It is chiefly annoying from its interference with walking, due to the pain occasioned by forced extension and the intense itching. It occurs isolated or accompanies eczema of other parts or other itching diseases, such as scabies, ichthyosis, and prurigo.

Upon the hands and fingers eczema appears in various forms, but most commonly as the result of the frequent action of irritating substances, such as soda and water in washerwomen (eczema lotricum), servants, waiters; of powdered substances, in spice peddlers or bakers; of mineral acids, turpentine, corrosive sublimate, etc., in typesetters, mirror makers, hat makers, etc. The intensity, extent, and form of the eczema will vary according as these substances act upon one portion of the hand or another. These "trade eczemata" occur usually in more or less sharply defined patches of thickened, red integument, covered with callous epidermis, pustules, and crusts.

Under such circumstances the finger nails are diseased; they become dry, brittle, fissured, and crumbling. Even though the hand is not the site of eczema, the changes just described may develop in the nails, by reflex means, whenever there is persistent eczema of any other part of the body—for example, the scrotum.

An interesting form of eczema of the palm of the hand, which occurs particularly in women, is not due to irritation connected with the occupation. It is manifested by the formation of a dirty, yellowish-brown, dry, callous but smooth thickening of the epidermis of the palm and the flexor surfaces of the fingers (eczema tyloticum). The condition is only recognized as eczema by the occasional itching

and the appearance of miliary vesicles during the scratching or after the use of potash soaps. Bullous and pustular eczema also occurs in a chronic state—*i.e.*, with constant exacerbations—in the cyanotic hands of chlorotic persons.

Great practical importance attaches to chronic eczema of the legs. This has been regarded as a sort of necessary derivation for remote pathological changes—for example, menstrual anomalies, hæmorrhoids, hepatic and cardiac affections. The serous exudation, the salt rheum, has been regarded as curative and perhaps even as vicarious for other secretions. Hence a cure of this condition was regarded as dangerous.

Unbiassed observation shows that eczema of the legs exhibits essentially the same symptoms as eczema of other localities. It merely differs from other forms in regard to the local tissue changes which are due to the etiological factors—viz., varicose veins, ulcers and cicatrices, pachydermia glabra.

The highest grade of the disease is chronic general eczema, in which, from vertex to toe, the skin is red and thickened, here scaly and fissured, there weeping or covered with crusts, and a kaleidoscopic picture is presented composed of all the different localized forms. The hairs of the head are falling out, the nails degenerated, the eyelids ectropic ; the patients scratch themselves constantly and suffer intolerably. Even such conditions are curable if the cause can be removed.

I must also refer to two special forms of the disease. *Impetigo faciei contagiosa* (Tilbury Fox) or *parasitaria* (myself) is characterized by an acute eruption of superficial vesicles, from the size of a pin's head to that of a lentil, on the face, scalp, and neck. They appear every few days to a week, at first disseminated, later aggregated, and dry very rapidly into gummy crusts, beneath which the epidermis forms. Some increase to the size of a dollar in the shape of concentric rings of vesicles, like herpes tonsurans or pemphigus serpiginosus. There is great swelling of the submaxillary glands. Numerous cases have been reported since Fox's description, even in adults, but chiefly in children of the same family, school, or hospital ward ; hence the suspicion that the disease is contagious. This was confirmed by the fact that I found a fungus among the epidermis cells of the walls of the vesicles. Geber, Lang, and Weyl believe, however, that the disease is a form of herpes tonsurans vesiculosus. I grant the correctness of such an opinion in the cases observed by these writers. When it develops in a typical form this impetigo is a peculiar disease and probably contagious in character, although our finding of a fungus has not been confirmed. The cocci (staphylococci), which have often been found, and with which inoculation experiments have been made, have occasionally produced vesicles or pus-

tules, but not the typical picture of impetigo contagiosa (Pogge, Lustgarten, etc.). In my first publication I called attention to the fact that the disease may occasionally be mistaken for varicella or pemphigus, because the vesicles are occasionally as large as a pea or bean and full, while usually they are very flat and rapidly dry into crusts. In recent years it has been observed in large epidemics after vaccination, or in the adults who have come in contact with endemics of so-called *pemphigus acutus contagiosus neonatorum* of children's hospitals. Hence it has been supposed that the two processes are identical and that impetigo contagiosa is a pemphigus contagiosus adultorum (Pontoppidan). I agree with Faber that the etiology of the disease is as yet unexplained, but it appears more probable that the disease is due to a micro-organism which enters the epidermis from without than that it is an infectious disease. This impetigo contagiosa is to be distinguished from impetigo faciei, which is usually associated with the presence of head lice and nits. This would explain its cumulative occurrence and the frequent relapses to which Unna called attention. Impetigo faciei is observed mainly in children, but not infrequently in adults who come in contact with the former. The process runs its course spontaneously in two to six weeks, and more rapidly if treated with zinc salve and washings of green soap.

Eczema marginatum (Hebra) is also a peculiar form of eczema. It is characterized by circles and segments of circles, from the size of a kreutzer to that of the palm of the hand, or even larger. The periphery is composed of red papules, vesicles, and crusts enclosing a darkly pigmented, scratched area. Peripheral spread takes place from a few papular centres. The usual sites are the scrotum, thighs, and the folds of the pendulous breast, but they may also be scattered over the body. From the genital folds the eczema circles spread far over the thighs, nates, and sacral region. Maceration by sweat (intertrigo), cold-water cures, and wet belly-bands are exciting causes of the affection. The presence of fungi in the epidermic layers has been proven by Köbner, Pick, and myself, but it is doubtful if the condition is identical with herpes tonsurans. It is distinguished from the latter by its obstinacy (fifteen to twenty years or more), intense itching, slight contagiousness, and the great tendency to local relapses. Perhaps the form described by Hans Hebra, in which discrete, dry, frequently relapsing eczema patches appear upon the joint flexures, and in which fungus elements were found, also belongs to this category.

The *diagnosis* is evident, as a general thing, from the symptoms already described. It must not be forgotten that the unity of the process is best seen by a comparison of all the diseased parts of the

skin, and that it is an inflammatory process, so that, unlike neoplastic infiltrations (lupus, syphilis), the redness always disappears under the pressure of the finger, and that all the other signs of inflammation may be observed.

From the course of the disease, for example, we may distinguish eczema papulosum figuratum s. folliculare of the trunk from lichen scrofulosorum and ruber, because in the latter the papules are stationary, while in the former they change rapidly, soon grow pale, or become vesicular. It is also distinguished anatomically from the small papular syphilide, which does not grow pale under the pressure of the finger, because it consists of a dense infiltration.

Eczema vesiculosum is not apt to be mistaken for herpes. In the latter the vesicles are arranged in groups, in the former they are closely aggregated, without any regular order. Greater care is necessary in order to distinguish bullous eczema, especially of the hands and feet, from pemphigus.

In eczema acutum crustosum et impetiginosum, it is merely necessary to remove the crusts and see the red, moist surface of eczema rubrum madidans in order to distinguish it from other processes which form crusts.

The differences between circumscribed scaly eczema and psoriasis or pityriasis rubra have already been explained. The differentiation is more difficult in chronic general eczema. It becomes easier when weeping surfaces are found. In eczema discoides and firmly infiltrated eczema of circumscribed parts of the skin, especially of the palm and dorsum of the hand, rubbing with a concentrated solution of potash will enable us to distinguish them from syphilitic patches or psoriasis; in eczema weeping points and vesicles will at once appear.

From diffuse palmar and plantar psoriasis (syphilitic) chronic eczema of these parts is distinguished by its irregular scaliness; at the edges it fades out imperceptibly in places, or, if sharply defined, is surrounded by normal integument. Eczema of these parts may also be mistaken for ichthyosis and lichen ruber. In general it is often possible to differentiate them from other affections only by observing the course of the disease or the action of remedies.

Eczema squamosum capitis must be distinguished from psoriasis, seborrhœa, lupus erythematosus, favus, herpes tonsurans.

Finally, it must not be forgotten that in many cases the diagnosis of eczema is not enough if the disease is only a complication or sequel of some other skin disease (scabies, prurigo) or a reflex effect of neurotic or general nutritive conditions (anæmia, scrofula). Hence it is always desirable to determine the etiology of each case.

LECTURE XXV.

ECZEMA (*continued*).

CAUSES—PROGNOSIS—TREATMENT.

FROM the standpoint of *etiology* eczemata are (1) idiopathic and (2) symptomatic.

Idiopathic eczemata are those which are produced by external injuries that irritate the skin. Hence they may be called artificial eczemata. They play a great part in practice and are often due to the physician himself. They owe their development to the same series of chemical, dynamical, or mechanical irritants which sometimes produce mere erythema, but, when acting more severely or when the skin is more irritable, give rise to eczema Among these agents may be mentioned : croton oil, tartar emetic in watery solution or in ointment, cantharides, mezereum, mustard, horseradish, potash lye, corrosive sublimate solutions, sulphur, soap, water in the shape of baths and compresses, and an entire series of local applications in dermato-therapeutics. It seems to be unknown to many physicians that a single application of a mustard poultice may give rise to general acute eczema which may last several months or even years. Unguentum hydargyri often produces a papulo-pustular eczema (eczema mercuriale) in hairy parts, or even eczema vesiculosum, madidans. The most violent effect is produced by tincture of arnica, which is extremely inefficient as a therapeutic agent, but in a certain degree of concentration will almost always produce intense eczema with confluent vesicles from the size of a pea to that of a bean.

To this category belong the artificial eczemata due to the constant contact of workmen with mineral acids, the juices of plants, turpentine ; with water, lye, and soap (waiters, washerwomen); with powdered substances (spice peddlers, millers, bakers); the eczemata following cold-water cures, etc.

Among the thermic influences we may mention eczema solare, usually papular, and eczema caloricum (from the heat of fires), which often exhibits large vesicles ; cold, dry air of winter produces eczema squamosum.

Sweat produces papular and erythematous eczemata (eczema sudamen, intertrigo); the form which develops beneath rubber clothing

belongs to this category. Mechanical influences, pressure, and friction rarely give rise primarily to eczema, but are very often effective when the integument has already been eczematous from one of the previously mentioned causes. In such cases the pressure of the hat band, garter, glove, collar, etc., may suffice to produce a fresh eruption of eczema.

In this way scratching itself produces eczema, inasmuch as the irritation of the follicles and papillæ may give rise to hyperæmia in the shape of lines and bands, and thus to disseminated or aggregated exudative forms of eczema Hence every eczema may be the cause of fresh eczema, on account of the associated scratching; for the same reason it occurs in all itching diseases, such as scabies, prurigo, urticaria, ichthyosis, pemphigus pruriginosus, and pruritus.

A cutaneous factor in the production of eczema is the existence of varicose veins in the lower limbs. They induce itching; as a result of the scratching a few papules and excoriations are produced; in the course of months and years this leads to occasional hæmorrhages, the formation of crusts, and thus to an intensification of the eczema.

Symptomatic eczemata are those which are the result or reflex of a morbid condition of the general organism or of some organ (excluding the skin). Thus, chronic and frequently relapsing eczema is found in individuals suffering from dyspepsia, diabetes, albuminuria, and is especially frequent in women suffering from dysmenorrhœa and uterine affections or who are chlorotic or anæmic. The eczema increases and diminishes with the relapse or improvement of the primary disease.

Eczema may also develop in a purely neuropathic manner; for example, in some women, during every pregnancy or after the cessation of lactation.

In children eczema of the face is very frequent after chronic affections of the eye and ear, while in adults it is more frequently due to other causes—for example, varicose veins. But the main part in the etiology of the disease is played, not by age, but by the individual irritability of the skin. The two sexes probably furnish an equal contingent of cases, but among hospital patients males number two-thirds. Eczema is not contagious or hereditary, although an inherited predisposition may be assumed in certain families.

A dyscrasia, such as rachitis, scrofula, or tuberculosis, cannot be regarded as a direct cause of eczema. It merely changes the nutrition of the tissues and increases the irritability of the integument and papillary vessels, so that eczema results from causes (heat, water, etc) which would otherwise be ineffective.

The *prognosis* is good, inasmuch as eczema never proves dangerous and may recover completely at any time. Whether an acute eczema will become chronic, whether relapses are to be apprehended,

etc., will depend upon the cause of the disease, the irritability of the skin, the ability to avoid harmful etiological factors, and, finally, upon the treatment.

The *treatment* of eczema is perhaps the most important chapter in practical dermatology. In no other disease of the skin is the physician able to exert a greater influence by the choice of remedies, the method of application, and by doing too much or too little. Nowhere does more importance attach to general therapeutic principles (*vide* page 74) than in the treatment of eczema. Stress should be laid upon three indications: 1. We must ascertain accurately in regard to every diseased part whether the inflammatory change is increasing or diminishing, whether it is acute or chronic. 2. We must know what change is to be effected by our remedy. 3. We must be able to control, at any moment, the effect of our remedies.

Acute eczema is treated, in general, by methods which relieve and prevent inflammation; chronic eczema, by methods which irritate and excite inflammation.

TREATMENT OF ACUTE ECZEMA.

In the developmental stage the main object of treatment is to avoid everything that will increase the inflammation and itching. The pressure and friction of underclothing, heat, sweating, must be avoided. Washings and baths are to be interdicted. The initial forms of acute eczema (eczema intertrigo and papulosum) may be rapidly relieved by such measures. Dusting powders are valuable to keep off sweat and irritation from the cutaneous folds which are the site of intertrigo. Any bland powder will answer, such as semen lycopodii, amylum tritici, oryzæ, talc. venet. pulv., or pulv. baptistæ, either singly or in combination, or with the addition of oxide of zinc, subnitrate of bismuth, or carbonate of magnesia. The addition of pulv. rad. ireos florent. will give the powder a faint perfume, but ethereal oils are not suited for this purpose. The following prescription may be used: amyl. oryzæ 100.0, talc. venet. pulv. 20.0, flor. zinci, pulv. rad. ireos florent. āā 5.0; or zinc. oxid., bismuth. subnitrat. āā 5.0, cerussæ 2.50, pulv. talc. venet. 100.0. Shoemaker recommends oleate of zinc as an agreeable and efficient dusting powder.

Upon exposed parts of the skin the powder is applied by means of Bruns' cotton or powder puffs; in intertriginous folds of the skin it is applied upon pledgets of cotton which are dipped in the powder, and by which the surfaces of the skin are carefully separated. The applications are changed as often as they become warm and moist.

In eczema papulosum the pruritus is often very violent and must be treated, because the scratching may rapidly aggravate the condition. We may apply spir. vini gallic., to which acid. carbolic.

(1 : 200) or acid. salicyl., resorcin, menthol, etc., has been added. For example : acid. carbolic. (or salicyl.) 1.00, spir. vin. gall. 150.0, spir. lavandul., spir. colon. āā 25.0, glycerin. 2.50. This is followed by the application of dusting powder. Brushing with tinctura rusci (olei rusci 50.0, æther. sulphur., spir. vin. rectif. āā 75.0 ; filtrat. ; adde : ol. lavandul. 2.0) acts still better.

If eczema vesiculosum, madidans, or impetiginosum has developed mild treatment must be continued during the acute stage.

In the most severe cases—*i.e.*, when various stages of acute eczema are found over the larger part of the body—it is best to undress the patient entirely and to put him to bed, covered simply with a sheet. The sheet and the patient's body should be freely strewn with dusting powder and the latter inserted between the folds of the joints, genitalia, etc. This is frequently repeated. If there is fever the proper diet is recommended and an acid may be given internally. Crusts over weeping places are burst with the powder ball, in order that the retained secretion may escape. It is only when there is very intense inflammation of the skin and violent pain that we employ applications of cold-water compresses or plumb. acet. basic. (10 : 500 aquæ). These must be kept at a low temperature by frequent changing. On account of the irritant action of water the application of Leiter's cooling apparatus is especially advisable in such cases. It consists of very flexible rubber or metallic tubing, rolled into concentric circles, and which can be fitted to different parts of the body ; through this a stream of water is allowed to flow. I have obtained better results of late years from compresses of alumin. acet. in the form of Burow's liquor (5–10 : 100 parts of water) and renewed every two or three hours. This is especially indicated in cases of intense œdematous swelling and abundant weeping, or in local, obstinately relapsing, acute outbreaks. This treatment has acted excellently in cases in which the abundant loss of serum from the moist surfaces, the intense itching and insomnia, cause exhaustion and have a very depressing effect.

Good effects are sometimes obtained, under similar circumstances, from compresses of thymol, ichthyol, thiol, or resorcin (one to two per cent). Non-irritating ointments may also be employed to advantage in this stage. For example : lanolin. 15.0, ung. emoll. 50.0, ol. amygdal. dulc. 2.50, aq. lavandul. 10.0 ; lanolin. 15.0, vaselin. pur. 75.0, zinc. oxid., bismuth. subnitrat. āā 5.0, spir. colon. 10 0 ; ung. emoll. 50 0, cocain. muriat. 1.0, zinc. oxid. 2.50.

We may also use protective applications to which zinc, bismuth, tar, resorcin, ichthyol, or thiol is added, according to the individuality of the case. For example, Pick's and Unna's glycerin-gelatin (gelatin. 50.0, aquæ 100.0, glycerin. 5.0), which may be made more or less flexible according to the proportion of glycerin. This not alone

acts as a protective covering, and according to the specific properties of the medicinal agents which have been added (zinc, tar, etc.), but it also acts favorably upon the hyperæmic skin on account of the uniform pressure which it exerts when it becomes stiff. We may also use Pick's liniment. exsiccans (page 81) and Lassar's zinc paste (zinc. oxid., amyl. oryz. āā 25.0, vasel. pur. 50.0, acid. salicyl 1.0); Unna's starch and dextrin pastes (the former consisting of 3 parts starch, 2 parts glycerin, and 15 parts water, boiled down to 15 parts; the latter consisting of glycerin, dextrin, and water in equal parts); finally, S. Kohn's epidermin may be added to any of these ointments or pastes.

According to the statements of Veiel, Unna, and Wolters, eczema acutum solare has been prevented, in some obstinately relapsing cases, by the wearing of yellow, blue, and, still better, green veils. Perhaps this is due to the absorption of chemical rays which are especially irritating to the skin. On the whole, however, the treatment with dusting powders is the best in acute eczema.

In hairy parts the process is allowed, without further interference, to resolve as far as the falling of the crusts and the stage of eczema squamosum. Then the tinctures and ointments which are useful in chronic eczema of the scalp come into play. In hairless parts the crusts may be removed by means of fats during the diminishing stage, and the course of the disease shortened by covering the still weeping surfaces with suitable ointments and a compress and bandage. The most serviceable are unguent. diachyli of Hebra (page 115), or my unguent. vaselin. plumbic. (empl. diachyl. simpl., vaselin. āā 100.0, liquef., misc.). The ointment is smeared thick on linen cut into pieces of proper size and kept in place by means of a flannel bandage; safety pins should be used for fastening. The ointment is renewed once or twice a day after the macerated masses of crusts and epidermis have been removed. Sometimes the skin swells acutely under unguent. diachyli, and we may then try unguent. vaselin. plumb. or unguent. boracic. (acid. boracici, glycerin. āā 5.0, paraffini, ceræ albæ āā 20.0, olei olivar. 50.0), zinc salve, or some other soothing ointment. Often none of these applications is tolerated, and the treatment to the squamous stage must then be continued with cold compresses, acetate of lead, Burow's solution, Lassar's paste, or starch.

After this stage different methods may be adopted. The most convenient is to apply fats several times a day to the raw, scaly surface, and then to dust powder upon it to conceal the redness. Suitable preparations are unguent. emolliens, glycerin cream (amyli puri 10.0, glycerini 40.0; coqu. misc.), ointments of white precipitate (1 : 40), oxide of zinc, subnitrate of bismuth (1 : 40), unguent. Wilsoni (ben-

zoes pulv. 5.0, axung. porc. 160.0 ; digere, cola ; adde : zinc. oxidat. 25.0 ; m. ; f. unguent.), pure vaseline, glycerin, etc.

As eczema squamosum itches and some of the remedies just mentioned irritate the skin, it is best to employ tar in this stage, unless these applications are given for cosmetic reasons—for example, in facial eczema, in order to enable the patient to leave the house. We employ the varieties of tar mentioned in the chapter on psoriasis. Great caution is requisite. The tar should not be applied to moist places. Even after the epidermis has been regenerated, and while the skin is still redder and warmer than normal, tar is very apt to cause fresh inflammation, especially in places which are in contact with other folds of skin (genital folds, pendulous breast). After a single application of tar we may be disagreeably surprised to find that the process begins anew with swelling and weeping. For the first few days it is therefore advisable to place the ointments over the tarred surfaces. It is only after the epidermis becomes browner and the hyperæmia diminishes and the skin remains cool that the tar may be applied alone. Even then it is well to prevent contact with adjacent parts by applying dusting powder.

As the regeneration of the epidermis becomes slower the tar-impregnated epidermis remains adherent and the surface appears uniformly brown. After this brown layer has been shed the spot appears white and smooth, or is still a little scaly. It is then made pliable by any of the bland ointments mentioned.

In this stage the process may also be rapidly terminated by a naphthol solution of 1 : 200 alcohol. The fluid is applied daily, at the most twice a day, for two to three days. If tolerated, the skin appears slightly browned, smooth and pale, and its use is then discontinued. If the skin becomes slightly reddened or the epidermis shows shallow fissures, the application must be discontinued. Similar effects are obtained from one-half- to one-per-cent solutions of salicylic acid or resorcin in alcohol.

The one-per-cent naphthol ointment, rubbed in once or twice a day and then dusted with powder, may also replace the final treatment with tar. Excellent effects are obtained in this stage from one or more applications of tinctura rusci (page 355).

TREATMENT OF CHRONIC ECZEMA.

The first indication is the methodical softening and removal of the crusts and of the dry, thick epidermic masses. The second indication is the relief of the chronic hyperæmia, which maintains the hyperplasia of the epidermis and the occasional exacerbations.

As we do not have to deal with acute hyperæmia, we may occasionally use active remedies, and even such as may give rise to a condition of acute eczema. It is known from experience that in the

active circulation of the blood and lymph which is associated with acute inflammation, thick calluses of epidermis are shed more rapidly and old inflammatory infiltrations of the corium are absorbed more readily.

The remedies employed for removing the epidermis and crusts are the well-known fats, especially cod-liver oil, also unguent. diachyli of Hebra, unguent. vaselin. plumb., and water. The oils must be rubbed in several times a day in large quantity, so that the crusts crumble and soften. At the same time the parts are surrounded with woollen stuffs in order to keep the oil upon them. The solid fats and ointments will act best if they are smeared thickly on linen or woollen cloths carefully fitted to the eczematous parts and kept in position with a flannel bandage.

A good substitute for the latter is Pick's emplastrum saponatum salicylicum (ten to twenty per cent. salicylic acid to one hundred empl. saponat.). These pieces of plaster adhere very well, and when pressed against the skin by calico bandages or tricot, as Pick suggested, they act excellently in softening the crusts and also in diminishing the inflammatory hyperæmia and infiltration. The plaster may be kept in position for one or more days, according to the indications in the special case. It is even preferable to ung. diachyl. and the other ointments which must be smeared upon cloths; also to Unna's plaster mull dressings, to Seabury & Johnson's gutta-percha mull, and the collemplastrum of Turinsky. The latter cause vigorous maceration, but artificial eczema is apt to develop on account of the india-rubber, and this is by no means counterbalanced by the addition of zinc, salicylic acid, etc.

Water may be employed in the shape of compresses or Priessnitz's packs, vapor, shower, and ordinary baths. Great efficacy also attaches to the use of india-rubber clothing in the shape of caps, gloves, jackets, stockings, or the adaptation of rubber bandages to the eczematous portions of the skin. But, as we have already stated, eczema is often produced on healthy portions of the skin when maceration occurs beneath india-rubber or from the use of water.

In order to macerate and remove the already softened morbid products, occasional washings with sapo. viridis, glycerin soap, spir. sapon. kalinus, naphthol soap, and naphthol-sulphur soap may be resorted to.

Very callous parts which are not softened by the remedies mentioned must be rubbed with concentrated acetic or hydrochloric acid, or they yield to the application of soft soap, which, smeared on flannel, is applied for twelve to twenty-four hours. They may also be cauterized with a solution of caustic potash five parts to distilled water ten parts.

Admirable effects are obtained from the combination of soft soap,

tar, sulphur, and fat in the modified sulphur ointment of Wilkinson (flor. sulph., olei fagi āā 10.0, sapon. virid., axung. āā 20.0, pulv. cretæ alb. 2.0). It may be used when there is considerable thickening of the epidermis, and even in the moist and pustular stages, especially in eczema capitis, mammæ, scroti, digitorum, palmare, and plantare.

The following statements may be made in regard to the method of treatment. We begin with macerating treatment, and continue this persistently for days and weeks until the eczematous skin has become flexible and smooth and no longer becomes sore after vigorous washing with soap, and until no moist points are observed. The skin is then either entirely healthy or it may still be hyperæmic (eczema squamosum). Tar is then applied in the manner described under the heading of acute eczema.

Eczema squamosum without notable thickening of the epidermis may be treated from the start by applications of tar. The tar is rubbed in vigorously in a thin layer by means of a brush. This relieves the itching most rapidly. If the skin is very thick, a mixture of ol. oliv. or ol. morrhuæ with ol. rusci or fagi (1 : 1 or 1 : 2) may be first employed. The modified Wilkinson ointment, used in a cycle of eight to twelve applications, acts very favorably upon old patches of eczema. Upon slightly diseased patches it will suffice to use applications of tinct. rusci, washings with solid tar soap or fluid tar soaps (ol. rusci 20.0, spir. sapon. kalin. 50.0, glycerin. 10.0), inunctions of tar ointment (ol. fagi 10.0, glycerin. 5.0, ung. emol. 50.0, bals. peruv. 2.50), tar with Hebra's glycerinum saponatum (page 80), with the previously mentioned pastes and liniments, carbolic salve (1 : 50), zinc and precipitate ointments, naphthol (1–2 : 100 ung. emolliens), potash cream. The latter is known as No. 1, 2, 3, or 4, according to the amount of potash (glycerin. 40.0, ol. rosar., ol. flor. aurant. āā gtt. 2, kali carbon. sol. 2.5 (No 1), 5.0 (No. 2), 10.0 (No. 3), 20.0 (No. 4).

I will now give a few directions in regard to the treatment of specially localized eczemata.

In eczema capitis the crusts are softened with olive oil, cod-liver oil, carbolized oil (acid. carbol. 1.0, ol. oliv. 100.0, bals. peruv. 2.0, or naphthol, resorcin, or salicylic acid 1 : 100 ol. olivar.), or a rubber cap. The latter is pressed down by a flannel bandage, never by an elastic band. The softened masses are washed off daily or every third or fourth day with spir. sapon. kalin. or some other soap. The patients should be told that the hairs will fall out freely and be removed during the manipulations. The hair is subsequently restored. After the stage of eczema squamosum we use applications of tinct. rusci, naphthol-alcohol (one-half per cent), later carbolic acid, salicylic acid, or resorcin with alcohol, and pomades of white precipitate or zinc with ung. althææ, etc. Lassar recommends his paste even in the moist stage. Applications of Wilkinson's ointment often act

excellently from the start. Cold douches and compresses (acetate of lead, Burow's solution) are to be recommended when the scalp is very much inflamed.

In eczema faciei impetiginosum the macerating plasters, ointments, and rubber masks must be carefully adapted to each part of the face, cotton pledgets placed in the folds, and the whole pressed down with a flannel mask or calico bandage. Tampons dipped in glycerin, oil, ung. emoll., or the like (aq. fortis, glycerin. āā 10.0, zinci sulph. 0.5) are placed in the nostrils. Obstinate rhagades of the nasal mucous membrane or folds of the ear are cauterized with the solid stick or emplast. sapon. salicyl. is applied. A salve of præcipit. rubri 0.15, ung. emoll. 10.0 is useful in eczema of the margins of the lids. The absorption of infiltrations of the lips is facilitated by pressure with emplastrum minii adustum or salicylicum. If weeping is no longer present we may employ tar, zinc or precipitate ointment, ung. Wilsoni, vaseline, zinc paste, glycerin cream, etc.

Chronic eczema of the vermilion border of the lips only yields, in many cases, to repeated cauterization with a solution of potash (5 : 10). This is also true of eczema of the mamma and nipple, whose integument softens more rapidly after treatment with soft-soap compresses, potash solution, sublimate-collodion (0.50 sublimate to 50.0 collod.), or acetic acid. In pregnant women I have never seen abortion follow such a plan of treatment.

Chronic eczema of the scrotum is treated according to the same principles, but it is more difficult to adapt the macerating applications. In old eczemata we will rarely succeed without cauterizing one or more places. When the entire scrotal integument is raw, nervous symptoms are not infrequent. The washings with soap, which are to be carried out twice a day, should be performed in the sitz bath. Tar is employed only when weeping no longer occurs after the application of potash solutions. An old scrotal eczema can rarely be cured in less than six to twelve weeks. Even after recovery the action of perspiration upon the scrotum must be prevented by the wearing of a suspensory and the use of dusting powder.

Eczema of the perineum and anus is treated in the same way. Ung. diachyli or, if this burns too much, ung. simpl., boracic acid ointment, salicylated plaster, etc., must be applied with the aid of a flannel T-bandage and a suspensory. If the rectum is fissured we apply suppositories of cacao butter 1.50, zinc. oxid. 0.15 ; or add ext. opii aquos. 0.02, ext. bellad. 0.02 ointment, with cocaine (one per cent) and cold enemata.

Eczema of the hands and fingers is conveniently treated with rubber gloves and fingers, or systematic applications of salicylated plaster, stripes of ointment, plaster mull, and washings with soap, so long as raw places, pustules, or rhagades are present. In obsti-

nate cases, especially in callous thickening of the palm and fingers, and when the vesicles are deeply seated, we may recommend hand baths (for ten minutes daily) of caustic potash 5 : 500 or corrosive sublimate 5 : 500. Immediately afterward the hands are washed with water, dried, and again covered with rubber plaster or ointment. The condition is often shortened very materially by applications of Wilkinson's ointment alternating with macerating treatment.

Circumscribed callous eczema of the palm of the hand may be softened by cauterization with acetic acid, citric acid, or potash (1 : 2), or traumaticin (india-rubber dissolved in chloroform), or by inunctions with a five-per-cent naphthol ointment.

Eczema of the umbilicus is treated with tampons dipped in ointments or acetate of lead, or with simple powder. If the redness and itching remain obstinate, tar is used. Very small eczema patches on the trunk and limbs are sometimes cured by applying corrosive sublimate (1 : 100 of alcohol or collodion).

The treatment of chronic general eczema depends upon the individual case. At one time it will be advisable to wrap the entire body in a rubber suit, at another time to apply cod-liver oil, Wilkinson's ointment, or Lassar's paste. Again, the different parts of the body must be treated differently, one with tar, another with ung. diachyli, another with powder, etc., according to the judgment of the physician.

We expect a positive cure from the proper use of local remedies in every case, even when it is believed to be due to other diseases, such as chlorosis, indigestion, chronic catarrh of the apices of the lungs, dysmenorrhœa, etc. In the latter event we also attach great importance to the internal treatment of the underlying disease. To scrofulous children we give cod-liver oil ; to chlorotic and dysmenorrhœic women, iron, arsenic and iron, or Fowler's solution. The following prescriptions are useful : Wilson's mist. ferro-vinoso-arsenicalis (liq. arsenic. chlorid. [Br. Ph.], syr. simpl. āā 10.0, vini ferri 60.0, aquæ fœnic. 80.0), one tablespoonful a day ; or sol. arsen. Fowleri 5.0, tinct. martis pomat., tinct. rhei āā 20.0, aquæ menth. 140.0, one to two tablespoonfuls daily ; or ferri albumin. 75.0, sol. Fowleri 2.0, twenty-five to thirty drops morning and evening ; or ferri citr. ammoniat. 5.0, arsen. alb. 0.04, ext. rad. gentian. ut f. pill. 50, two pills daily. The arsenical ferruginous waters of Roncegno, Levico, and Guberquelle may be taken in doses of two to four tablespoonfuls daily. In chronic pulmonary catarrh or dyspepsia we may order teas of chenopodium or lichen islandicus ; milk and whey cures ; feeble alkaline or ferruginous mineral waters ; in the summer, a trip to the country or mountains. After the recovery of the eczema, mild hydrotherapeutics and nourishing diet are indicated In such cases it is well to order strong wines and good beer.

We are never opposed, in eczema, to the ingestion of salted or spiced food, cheese, caviar, etc., because they increase neither the eczema nor the itching, nor do they produce the mythical "acridity of the blood." All these remedies, however, possess only an auxiliary action and are ineffective in the direct cure of eczema.

LECTURE XXVI.

PRURIGO.

CHARACTERISTICS—PRURIGO AGRIA AND PRURIGO MITIS.

MANY physicians still employ the term prurigo as synonymous with pruritus, and apply it to any skin disease which is attended with itching—for example, prurigo pedicularis, senilis, localis.

Hebra first used this term to designate a morbid process which differs materially from all other itching diseases and constitutes a malady *sui generis*. Neither is it justifiable to apply the term pru riginous to papular eczema or other papular dermatoses in contra-distinction to Hebra's prurigo.

Prurigo begins in earliest childhood and usually continues during life. Repeated eruptions of light-colored or pale-red, firm, violently itching epidermic papules, from the size of a millet seed to that of a pin's head, appear scattered over the body, but chiefly on the extensor surfaces of the limbs. The integument of the joint flexures is regularly exempt. The symptoms of prurigo are still further amplified by the sequelæ of the eruptions and by the peculiarities in their development and course.

The symptoms of prurigo are not presented by the new-born child, but appear at the age of eight to twelve months as an urticaria. This continues into the second year of life, with its characteristic appearance and disappearance of the wheals, itching, insomnia, and excoriations. Toward the end of the first or beginning of the second year, papules also appear, mainly on the anterior surface of the lower limbs, the sacrum, buttocks, and extensor surface of the upper limbs. The papules project very slightly, often can only be found on palpation, are pale or red, itch very violently, become larger on scratching and are injured thereby. The little drop of exuded serum and blood soon dries into a brown crust upon the tip of the papule, and remains after the latter has been absorbed.

Other symptoms follow as the result of the scratching, viz., excoriations in the shape of streaks, blood crusts, pustules and deeply spreading losses of substance, streaked and diffuse brown pigmentation, tearing out of the downy hairs, œdema and thickening of the legs, swelling of the inguinal glands, and eczema of all degrees.

With the end of the second year or beginning of the third year the typical symptomatology of prurigo is complete. When the patient is stripped it is seen that the morbid changes are most marked on the extensor surfaces of the limbs, and increase in severity from the forearm to the legs.

In the latter locality are found the most papules, usually excoriated and covered with a blood crust, in addition to numerous pustules and excoriations. The skin is dark brown in color, and scales like fine meal when scratched with the nail. On stroking the skin from the thigh downward, we have a distinct sensation of increasing roughness, dryness, and thickening. The lines and furrows over the knee are enormously developed. When the integument of the anterior surface of the thigh is picked up between the fingers, it is found to be unusually thick. In severe cases the integument of the legs may be so thick and rigid that it cannot be raised into a fold.

The changes extend in a milder degree to the dorsum of the foot. On the trunk there are often many scattered papules and excoriations, fewer upon the cheeks, neck, and forehead ; here we usually find scaly eczema. The skin of the popliteal space, bend of the elbow, axillary space, and groin is always white, smooth, flexible, perspiring, and free from prurigo papules. The group of enlarged inguinal glands completes the characteristic clinical picture. The disease continues thus to mature life, and even to old age, with a complete retention of the original type. A child of three years with prurigo will look like a miniature edition of a man of fifty suffering from the same disease.

The condition changes repeatedly, however, in regard to severity and attendant symptoms. As a rule the eruption and itching diminish in summer, and perspiration may even be produced in the pruriginous skin. Constant care of the integument has an undoubted mitigating influence. When treatment is abandoned there is an aggravation of the symptoms, particularly of the sequelæ, complications of the exudative processes, and mechanical injuries. These include pigmentation, which may even be blackish-brown (melasma), and the thickening of the skin ; on the legs it finally becomes firm, almost cicatricial, and cannot be raised into a fold. A further complication is eczema crustosum, which usually covers the pruriginous parts, but, as in all itching diseases, may also attack the unaffected parts, the joint flexures, face, and scalp ; the hairs then look dusty, lustreless, become thin, and fall out. Finally, there may be enlargement, and in rare cases even suppuration, of the inguinal glands.

There are two forms of the disease, a severe one, *prurigo agria s. ferox*, and a milder one, *prurigo mitis*. The former has just been described. The other form is milder, either as regards the number of papules and severity of the itching (so that the complications and

sequelæ are much less marked), or because only the legs (in exceptional cases only the arms) are affected.

The character of the disease as regards intensity is evident from the beginning, so that a child of five years with prurigo agria exhibits much more marked changes in the legs than a man of forty years who suffers from prurigo mitis. This distinction possesses special importance in regard to *prognosis*. The severe and even the moderate forms of prurigo in adults are incurable.. In early childhood prurigo mitis may be entirely cured by careful and persistent treatment. Even in later years prurigo agria may be improved to such an extent that the patient is free from it at times.

When left to itself prurigo exercises a marked effect upon the patient, both physically and mentally. The local processes lower the physical condition by the vital losses, nervous tension, insomnia, etc., so that the patients usually have a sallow, poorly nourished look. Attendance at school is often interfered with on account of the disease. The patient is avoided by his companions on account of the scratching, and he is usually unable to assume his proper position in life. It is only the well-to-do who are able, by constant care, to relieve the condition to such an extent that they can retain their position in society.

The *diagnosis* can hardly go astray. The brown, scratched, thickened, dry skin, with the papules and punctate crusts, the increase of the changes toward the legs, the enlargement of the inguinal glands, the unaffected white skin of the femoral triangle, the popliteal space, and the elbow, are so characteristic that they can be mistaken for nothing else.

The diagnosis is difficult when the eruption first appears and consists mainly of urticaria. Prurigo may also be overlooked when the eczematous signs are so prominent that they conceal the evidences of prurigo, and when parts which are not attacked by prurigo. such as the joint flexures, are also the site of eczema.

In ichthyosis nitida the skin is dry and the epidermis scaly in the same localities as those in which prurigo develops. But the other characteristic signs of prurigo—viz., papules, pigmentation, and thickening of the skin—are wanting, although moderate eczema is present. Pigmentation, papules, pustules, and eczema develop in all chronic skin diseases which are attended with intense itching, but they never present the typical localization of prurigo.

The results of *anatomical* investigations do not explain the peculiarities of the symptoms. The papules merely exhibit moderate cellular infiltration and serous exudation into the papillæ and the rete Malpighii. In places which have been the site of intense prurigo for many years, the findings are the same as in every chronic dermatitis —viz., thickening, proliferation of the rete layers; scattered pigment

deposits in the corium; more abundant cellular infiltration of the latter, especially around the vessels; here and there dilatation of the lymph spaces and some of the sweat glands from proliferation of their cellular lining; projections into the follicles as the result of outgrowth of the root sheaths; thickening of the musculi arrectores. In very old cases there is atrophic degeneration of the follicles and sebaceous glands.

With regard to the finer anatomical changes in recent prurigo papules, Auspitz and Caspary claimed that they consist of a proliferation of the rete, without inflammatory affection of the papillary vessels. Riehl, on the other hand, emphasizes the inflammatory processes in the papillary layer, but states that they have the greatest analogy, not with eczema papulosum, but with urticaria wheals. Hence he regards the disease itself as analogous to urticaria, especially in view of the fact that it always begins with urticaria. But he does not overlook the fact that this does not explain its peculiar localization, obstinacy, etc.

Auspitz's assumption that the prurigo papules were proliferation forms of the rete did not suffice for his interpretation of prurigo. He also assumed the concurrence of a motor neurosis in the shape of a spasm of the arrectores muscles, and of a sensory neurosis (pruritus). These indefensible views have already been overthrown by Riehl and Caspary. Leloir's statements concerning the degenerative changes in the nerve endings of the papillary body have not possessed much importance since S. Mayer showed that such changes are normal.

Hence the papules still remain the essential anatomical basis of the disease. Caspary regards them as epidermal papules, Riehl calls them urticarial, and I consider them analogous to the papules of eczema papulosum, *i.e.*, as due to inflammatory processes in the papillary tissues. It will be found, however, that these views do not differ very materially. In a certain stage—*i.e.*, after disappearance of the vascular dilatation and absorption of the exudation—every papule of eczema papulosum consists only of proliferating rete cells. I cannot regard the prurigo papules as true urticaria, because they are extremely small, never enlarge, and persist much longer than wheals. The important fact is that such papules constantly appear in certain localities, and that the process begins about the end of the first year of life.

Every other itching disease, even if it exhibits papules and, temporarily, the same localization, is not prurigo if it begins at another time—*i.e.*, at a later period or during the very first weeks or months of life. Such a disease is an eczema papulosum, and hence is curable. G. Behrend has reported the development of true prurigo after scarlatina in children of five years, and believes the disease may develop in later life from an impoverished condition of the

blood. With this statement I do not agree; the children had evidently suffered from prurigo at an earlier period.

Duhring's *prurigo hiemalis* is not a prurigo, but a pruritus which is due to the dryness and brittleness of the epidermis consequent on the coldness and dryness of the winter atmosphere.

Hutchinson's *summer prurigo* is also not a prurigo, but the condition is not entirely clear to me. He observed in a number of people that papules and nodules developed every summer, beginning in childhood and lasting into the twenties, upon the face, ears, neck, and arms. These itched violently, and the scratching produced ulcerations, losses of substance, and cicatrices.

It is probable that the itching sensation which arises in the papules of prurigo is due to the irritation of the papillary nerves by the suddenly developing, although extremely minute, inflammatory product (serum). But this does not explain why more considerable exudations, as in herpes or erythema papulatum, do not itch so violently; it does not explain the obstinacy of the papules and their peculiar localization. Prurigo is not a pure neurosis, because there are visible changes in the skin which keep pace with the severity of the symptoms.

On the other hand, in pruritus cutaneus, which is a true neurosis, no anatomical changes develop in the skin apart from the rapidly healing effects of scratching, even after the disease has lasted for years. From this standpoint, likewise, prurigo must be excluded from the pure neuroses.

With regard to the *causes* of prurigo, we are merely able to mention certain general conditions in which it occurs more frequently. For example, prurigo is much more common among the poor than among the well-to-do. Furthermore, it often develops in feeble, poorly nourished, scrofulous children. It must not be forgotten, however, that the disease is sometimes seen in splendidly nourished children, and that it is also apt to impair their nutrition very materially. The peculiar localization on the extensor surfaces of the limbs is probably due to the same congenital anatomical conditions as in ichthyosis. However, similar papules appear upon the trunk and face when the disease increases in severity. It appears to be more frequent in males than in females.

In some cases it seems to be due to hereditary predisposition. Not infrequently several children of the same family are attacked. There is much truth in Hebra's statement that tubercular mothers (and, according to my experience, those who are suffering, during pregnancy, from an exacerbation of chronic catarrh of the pulmonary apices) often give birth to children who acquire prurigo.

The disease is not due to external agents and it is not contagious. We have seen nothing in our numerous cases which would lead us

to believe that prurigo is transmitted directly from the parents to the children.

In the *treatment* of prurigo, sulphur, tar, soap, and naphthol are the most effective agents against the itching and the papular eruptions. Sulphur is used in the shape of sulphur soap, a solution of kali sulphuratum, or sulphur thermal waters. Tar is used pure or mixed with olive oil or cod-liver oil (ol. morrhuæ, ol. rusci āā). Resort may also be had to the various bland ointments and oils and to baths in the treatment of the prurigo symptoms, as well as of the accompanying eczema.

At the onset, and in mild forms in which urticaria predominates and there are only a few prurigo papules, it will suffice to wash the patient thoroughly every night with sulphur or sulphur-tar soap, or to leave him in the bath for an hour and then anoint with cod-liver oil, oil and tar, or fat. In more intense prurigo, sol. Vlemingkx is to be employed in protracted baths, as recommended for psoriasis.

A series of ten to twelve inunctions with Wilkinson's ointment will produce considerable improvement in prurigo agria and will at once cause cessation of the itching and secure sleep. The wearing of rubber garments also has good effects. It goes without saying that other remedies which are indicated in moderate itching, dry epidermis, and weeping eczema (carbolic acid, salicylic acid, 1 : 200 of alcohol, zinc salve, unguent. diachyli, etc.) may also be applied, either generally or upon certain parts of the skin

The natural sulphur thermal and also artificial sulphur baths are extremely useful in prurigo, but they must be sufficiently prolonged. Baths of corrosive sublimate (5.0 to 10.0 to a bath), alum, soda (1,000 to 2,000 grammes to a bath), iodine and bromine salts, cortex quercus, may be occasionally used to advantage, but as a general thing their action is feeble.

For the last ten years I have treated all cases of prurigo with naphthol and have had extremely satisfactory results. The itching diminished at once and soon disappeared, and the eczema also improved *pari passu*. This plan of treatment possesses merely the advantages of cheapness, convenience, and cleanliness, inasmuch as baths are unnecessary, the remedy has no disagreeable odor, does not soil the linen, and can be used conveniently. An ointment consisting of naphthol 5.0, ung. emoll. 100.0, is rubbed every night into the integument of the limbs, especially on the extensor surfaces, and then dusting powder is applied. For children under ten years I order a one- to two-per-cent ointment. Every other night the ointment may be washed off in the bath with naphthol-sulphur soap. If there are severe eczematous symptoms, with weeping and crusts, these are first relieved with salicylated plaster, Wilkinson's ointment, etc., before the naphthol treatment is applied.

The local methods of treatment are continued until the skin feels smooth and flexible and there are no fresh papules or itching. The treatment is then continued in a moderate manner every second, later every third day, and only stopped entirely when the skin has remained healthy for months.

Internal remedies do very little good in prurigo. In a few cases I have observed undoubted improvement from the administration of carbolic acid, 1.0 to 1.5 grammes daily in pill form. Temporary improvement can also be obtained from hypodermic injections of pilocarpin. muriat., 0.02 at a dose, or the administration of jaborandi.

Arsenic is useless. In those patients who are poorly nourished, sallow, scrofulous, good effects are obtained from the internal administration of cod-liver oil, either alone or in combination with iodine (iodin. pur. 0.1, ol. morrhuæ 100.0) or phosphorus (ol. morrhuæ 30.0, phosphor. pur. 0.01, gumm. arab., sacch. alb. āā 15.0, aquæ destil. 40.0, syr. simp. 15.0); also from general hygienic measures, such as a milk cure, trip to the country, the occasional use of iodine baths, etc.

LECTURE XXVII.

ACNE DISSEMINATA—ACNE VULGARIS—ACNE ARTIFICIALIS—TAR, IODINE, AND BROMINE ACNE—ACNE ROSACEA.

3. FOLLICULITIDES—ACNE FORMS.

THE forms of disease in this category—viz., acne disseminata, acne rosacea, and acne mentagra or sycosis—constitute a natural group of diseases on account of certain common factors, such as the localization in the face, the implication of the cutaneous glands, etc., but they are also distinguished as separate processes on account of many characteristics.

ACNE DISSEMINATA.

This consists of the formation of red, conical or hemispherical, painful nodules, which correspond to the follicles, and vary from the size of a pin's head to that of a pea or more. At the apex they carry a black comedo or pustule, or they contain pus in the interior. Pressure expresses the comedo, pus, and creamy fat, and this is followed by considerable hæmorrhage.

It is not difficult to see that each nodule corresponds to a sebaceous gland and its vicinity and is due to inflammation. They are found on the face, sternum, and back, much more rarely on other parts of the body, particularly the limbs. The palms of the hands and soles of the feet seem hardly ever to be affected.

The variety known as acne vulgaris (Fuchs) occupies the forehead, cheeks, nose, lobe of the ear, neck, sternum, and back, occasionally the borders of the lids and the conjunctiva (Arlt). Its forms include the previously mentioned nodules with a central comedo (acne punctata) or with purulent contents (acne pustulosa), or red, firm, painful nodules (acne indurata) ; they are disseminated (acne disseminata) or arranged in long rows like grains of wheat (acne hordeolaris). Numerous comedones are always present and the skin is greasy (seborrhœa oleosa).

During the chronic course of the disease (several months or years) the local signs are constantly changing, but, taken as a whole, the process maintains essentially the same character. New inflammatory nodules, pustules, and comedones appear, while the oldest abscesses burst or dry up, leaving shallow scars or temporary pigment patches.

They vary in number from a few to many hundreds in all stages of development. When closely aggregated the face is swollen and greatly disfigured by the red, fluctuating, and firm nodules, abscesses, comedones, and cicatrices.

Tumors sometimes form, from the size of a pea to that of a hazelnut, which are due to cystoid dilatation of the sebaceous glands. When opened they discharge tough, mucoid, fatty, rancid contents (molluscum atheromatosum). Some of these tumors persist for years and shrivel into hard, globular bodies after their contents have undergone thickening. In many places perifollicular abscesses develop and their pus surrounds that of the gland abscess proper. Finally, there may be hæmorrhagic undermining and destruction of parts of the skin occupied by large acne pustules, and this is followed by bridging cicatrices.

FIG. 30.—VERTICAL SECTION THROUGH AN ACNE PUSTULE.

a, epidermis ; *e*, cell infiltration in the corium surrounding gland and follicle and neighboring papillæ ; *b*, *c*, sebaceous gland, its contents fallen out to a great extent, the rest consisting of inflammation and fatty epithelial cells ; *d*, remains of hair follicle greatly atrophied.

The *anatomical* site of the inflammation is the cutis tissue surrounding the sebaceous glands and hair follicles and their common exit to the cutaneous surface (G. Simon, Virchow, Hebra-Kaposi, Biesiadecki). The changes correspond in degree to the clinical symptoms. In acne punctata the papillæ and upper layers of the corium surrounding the comedo contain distended blood vessels, serum, and exudate cells in their dilated meshes. In acne pustulosa purulent exudation is found in the excretory duct ; in the larger nodules and pustules there are extensive inflammations in the tissues surrounding the glands and follicles, a collection of blood and pus in the glandular cavity and hair follicle, separation of the root sheaths and purulent destruction of their epithelial cells. With increasing intensity of the local process the sebaceous gland is entirely lost in the suppuration while the hair follicle is still intact, thus distinguishing it from sycosis. In large acne abscesses, however, the hair follicle is also destroyed, and we find merely a large pus cavity, sometimes containing a hair, surrounded by highly vascular and infiltrated cutis. In such cases the local process can only terminate in cicatrization and obliteration of the follicles, while complete resolution is possible in acne punctata and superficial acne pustulosa.

The immediate *cause* of acne is the irritation of the tissues by the secretion which stagnates in the excretory duct or in the sebaceous gland (Virchow). This may result from mechanical obstruction (occlusion of the mouth of the follicle by tar in tar acne) or from a

functional disorder, inasmuch as the secretion is chemically changed or is excessive. The latter condition appears to obtain in acne vulgaris, because this begins usually at the period of puberty, when the function of the sebaceous glands increases with the more active development of the hairs on the body. It is more frequent in males and brunettes, who suffer from seborrhœa oleosa, than in females and blondes. Chronic dyspepsia and chlorosis appear to predispose to acne. It is popularly believed, but without foundation, that acne is due to the ingestion of highly spiced food and also to sexual continence. The disease usually subsides gradually at the period of complete maturity, in women about the age of twenty years, in men somewhat later. In exceptional cases it continues into the forties.

The *diagnosis* of acne vulgaris is easily made from the symptoms just described, from the presence of comedones, nodules, and pustules, and from their inflammatory character. Variola of the face and also a pustular syphilide may occasionally be mistaken for acne.

We apply the term *acne varioliformis* to a peculiar variety which usually appears at the capillary border of the forehead and neck in groups of flat papules and pustules. A few scattered lesions are found on the scalp ; more rarely they extend over the entire face and upper part of the chest. This is not to be mistaken for Bazin's acne varioliformis, which is identical with our molluscum verrucosum.

At the centre of the flat, firm, brownish-red papule, which is as large as a lentil, a flabby pustule forms, and soon dries into a crust which sinks below the level of the skin or is converted at once into a dry, parchment-like slough ; after this has fallen off a cicatricial depression remains. The picture vividly recalls that of variola (hence the name), and, on the other hand, there is great resemblance to syphilis corymbosa on account of the arrangement in groups, the dark color, and the central depression. On account of its obstinate recurrence the process lasts for years. Nothing is known concerning its etiology, but in some cases it appears to be associated with chronic disturbance of digestion.

C. Boeck and, later, Pick have described, under the term *acne necrotisans*, inflammatory nodular elevations, at least as large as a lentil, which exhibit an analogous localization and form. They are attended with capillary hæmorrhages and dry mummification of the upper cutis layers of the centre of the papules, so that firmly adherent sloughs are produced. After these fall off depressed cicatrices remain. These cases seem to me to belong essentially to the category of acne varioliformis.

As a variety of acne varioliformis or necrotisans I may mention *acne urticata*. In exacerbations which are repeated for years there

is a very acute development of pale-red, wheal-like, very hard elevations, varying from the size of a bean to that of a twenty-five-cent piece. They appear upon the face, forehead, nose, chin, cheek, scalp, or later upon the hands, arms, and legs, usually upon the extensor surfaces. This is attended with the most violent itching, burning, and pain. In two to four days, sometimes in a few hours, they undergo spontaneous involution. As a rule, however, the patient scratches or pierces them with the nails or a needle, and then compresses them, because no relief is felt until serum and blood escape from the swollen papillary and rete strata. Coagulation of the exudation and the regeneration of new epidermis rapidly take place, but the base and surrounding parts remain very hard and give rise to itching, burning, insomnia, and nervous restlessness. Hence the patient repeats the puncture and compression. At the end of one to two weeks the hardness disappears, and flat, brown, cicatricial streaks, corresponding to the injuries and suppurations, remain. In the few cases which I have seen the process has continued for fifteen to twenty years. The itching, pain, localization, suppuration, the insomnia due to the subjective distress, and the constant relapses make this disease one of the most annoying and depressing of all diseases of the skin.

The term *acne* or *folliculitis exulcerans serpiginosa nasi* may be applied to a disease which I observed simultaneously in three patients, one woman and two men of middle life. It occurred as an acute eruption on the tip of the nose, in which flabby papules, as large as a pin's head or a little larger, developed ; these rapidly underwent purulent or necrotic degeneration, resulting finally in numerous deep scars. A dense row of new papules, running the same course, then developed at the margin, and this continued until, within a few weeks or months, the cutaneous part of the nose was destroyed by the deep cicatrices. After scraping out the papular wall formed by the new eruption around the cicatricial part, relapses still continued until the process stopped at the level of the bony part of the nose. The curetted tissue proved to be vascular, flabby granulation masses with numerous giant cells. There was great resemblance to syphilis pustulosa, except that the papules were very flabby and vascular, while their original size and prominence, their rapid development and destruction, excluded the diagnosis of lupus.

Unlike the previously described folliculitides, the essential feature of this form is the development of vascular granulation tissue around the follicles, which rapidly undergoes necrosis. In this sense they resemble the rare case of folliculitis exulcerans in my clinic, described by Lukasiewicz. For two to three years such papules appeared upon the nates and limbs of an anæmic girl. They occurred in large numbers, in patches which increased, by peripheral ex-

tension, from the size of a quarter to that of the palm of the hand. Recovery was finally secured by successive applications of the thermo-cautery.

In a man, finally, I have seen, in combination with acne pustulosa, the subacute development of numerous non-suppurating, spongy, vascular papules throughout the face. They could only be removed by the sharp spoon. The term *acne telangiectodes* is not inappropriate for this form.

This does not exhaust the entire series of possible folliculitides. General acute eruptions of folliculitis which were etiologically incomprehensible have been observed, such as Barthélemy's "acnitis" (!), or the acne cornée of French writers, or forms accompanied by papillary or epidermoidal hyperplasia, which will be discussed in the chapter on keratoses.

Acne cachecticorum (Hebra) occurs in marantic, scrofulous individuals, and is often combined with lichen scrofulosorum. It occurs abundantly on the trunk and lower limbs, less upon the face. It consists of flat, flabby, livid-red papules and pustules, from the size of a pin's head to that of a lentil, which are very similar to syphilitic eruptions. They are distinguished from the latter by the absence of a firm infiltration and by the fact that they never give rise to characteristic ulcers; at their worst, to flabby, hæmorrhagic, superficial tissue necroses.

They are due to impaired nutrition, which leads to a combination of follicular disease with hæmorrhagic exudation into the tissues, and often also to scorbutus. In one case I observed acne cachecticorum as the result of mental depression in a well-nourished individual. It disappears after improvement of the causal condition, but may last for years.

Next follow those forms of acne which are produced artificially by irritation of the sebaceous glands by certain drugs, which either enter the mouths of the glands from without, as in the case of tar, or enter the circulation from within and are excreted by the glands (occasionally tar, also iodine and bromine).

Tar acne—acne picealis, acne ex usu picis—has already been discussed in our remarks on the tar treatment of psoriasis. It results in the development of numerous reddish-brown papules, from the size of a pin's head to that of a pea, whose centre contains a black speck, the particle of tar occluding the mouth of the follicle. In addition there are firm nodules (which may even attain the size of a hazelnut), abscesses, furuncles, and black comedones. They are situated mainly on the extensor surfaces of the lower limbs, which are plentifully provided with hair follicles.

Acne is also produced by various tar products, such as benzine and creosote. These may be rubbed into the skin or finely diffused

in closed rooms and irritate the skin directly, or they may be inhaled and then excreted through the skin. Endemic tar acne has been observed repeatedly in tar-distillation factories and in mills in which the looms were smeared with such oils. An analogous condition is the acne due to chrysarobin and pyrogallic ointments.

Iodine acne follows the internal administration of iodide of potassium and sodium, sometimes after a small dose. It appears first in the face and is often associated with other symptoms of iodism. The pustules are conical, with a bright-red base; exceptionally they are hæmorrhagic (Fournier) or are surrounded by a vesicular wall (T. Fox). This variety is distinguished from acne vulgaris by its acute onset, the coincidence of many similar acne pustules, and the absence of the pigmentation and cicatrices incident to a chronic course. Adamkiewicz demonstrated the presence of iodine in the contents of the pustules. It disappears after the discontinuance of iodine treatment.

Bromine acne has been known to physicians since the use of bromides became frequent in medication. I have seen a marked case in a nursing infant of six months, who received the bromide through the mother's milk. In this variety larger and smaller nodules and pustules develop, as in ordinary acne, sometimes attended with febrile symptoms. If there is a cumulative action of the drugs, infiltrations may form, as large as a quarter or a dollar, from the close approximation of numerous acne pustules. Above these the epidermis is raised into closely aggregated pustules, resembling eczema bullosum, herpes, or pemphigus. They project one to two lines above the level of the skin, and, after evacuation of the individual pustules, look like a honeycomb or break down into foul ulcers. There may also be dark brownish-red, diffuse, moderately hard infiltrations, from the size of a dollar to that of the palm of the hand, which become depressed in the centre and thus resemble syphilitic nodules still more closely. Finally, there may be warty and nodular excrescences on an infiltrated base. If the administration of the bromides is continued these lesions may be produced incessantly for many months, or even one to two years, and may extend over the greater part of the body. They disappear, leaving either brown pigmentation or cicatrices. Neumann has shown that they are due to deep inflammatory infiltration of the cutis, destruction and degeneration of the glands and follicles. They are undoubtedly due to the irritation exerted by the bromine, which is excreted through the skin or sebaceous glands. P. Guttmann has demonstrated the presence of bromine in the pustular contents.

The prognosis of these artificial acne forms is favorable, inasmuch as they disappear spontaneously after the discontinuance of the drug.

There are considerable cicatricial changes, however, at the site of the deep infiltrations.

The *treatment* of acne vulgaris, when properly carried out, is always crowned with success. In the first place, all the glandular and subcutaneous abscesses must be opened with a sharp-pointed bistoury and their contents expressed. The knife must often enter very deeply, and ten to fourteen sittings are sometimes required to evacuate the bulk of the abscesses. The hæmorrhage is considerable, but can be checked by compression with cotton. Cold compresses may be applied after each sitting. Hæmorrhagic, flabby infiltrations are scraped with a sharp spoon, fringes of skin are removed with scissors.

At the end of ten to fourteen days, when the fluctuating nodules have been removed and the swelling of the skin relieved, and only small nodules remain, we may begin a plan of treatment which, in cases of moderate severity, may be initiated at once. This is carried out as follows: 1. Mechanical expression of comedones with the squeezer and incision of beginning abscesses. 2. Constant, vigorous washings with soap (toilet soap, solid or fluid glycerin soap, soft soap, spirit. sapon. kalin., iodine-sulphur soap, naphthol and naphthol-sulphur soap), combined with vapor and douche baths 3. The methodical application of such remedies as will cause rapid exfoliation of the epidermis as well as the lining cells of the sebaceous glands, thus relieving the latter of their contents and causing contraction (increase of the feeble tonus). For this purpose we use sulphur pastes, irritation by means of sulphur, soft or naphthol soaps, tincture of iodine, iodine-glycerin, emplast. hydrargyri, and cold douches. 4. Protective measures, ointments, water, powder are employed for cosmetic purposes.

The method of treatment may be carried out according to the following plan:

At night the face and back are washed with one of the above-mentioned soaps, then the skin is vigorously rubbed and pressed, so that the comedones are removed mechanically. Then a douche and drying. Next, a sulphur paste is rubbed in with a brush and allowed to remain over-night—for example: lact. sulph. 10.0, spir. vin. gallic. 50.0, spir. lavand. 10.0, glycerin. 1.50; or sulph. citrin. 10.0, spir. sapon. kalin. 20.0, spir. lavand. 60 0, bals. peruv. 1.50, spir. camphor. 1.0, ol. bergamot. gtt. 5; or lact. sulph. 10.0, potass. carb. 5.0, spir. sapon. kalin. 20.0, glycerin. 50.0, ol. caryoph., ol. menth., ol. ror. mar. āā 1.0. Sig.: To be applied after being well shaken. Instead of such pastes, to which naphthol (one per cent), carbolic or acetic acid, etc., may also be added, we may rub in simple soapsuds or the suds of sulphur or naphthol-sulphur soap, and allow them to remain on the skin over-night. Sol. Vlemingkx cauterizes delicate skin and should

be employed only against acne of the back. The action of such remedies is increased by covering them with flannel. In the morning the paste or soap is washed off and a protective ointment applied to the rough and reddened skin—for example, ung. Wilson. or zinc. oxid. 20.0, ung. emoll. 100.0, ol. resedæ 2.0, ol. rosar. gtt. 5 ; or bismuth. subnitrat., zinc. oxid. āā 5.0, ung. emoll. 50.0, ol. naphæ gtt. 4 ; or cold cream 50.0, zinc. oxid. 5.0, glycerin. pur. 1.50, tinct. benzoes 1.0. The ointments are rubbed in a thin layer until they disappear, and then one of the well-known dusting powders applied. Ointments and powders which contain lead and mercury are contraindicated during treatment with sulphur and when there is an abundant secretion of fat, because lead and mercury sulphides produce brown patches on the skin. In addition to the other dusting powders, we may recommend the so-called ladies' powder : pulv. lapid baptistæ, talc. venet., amyli oryzæ āā 30.0, zinc. oxid. 10.0, ol. neroli gtt. 2, ol. rosar. gtt. 4 ; also eau de princesse (Hebra) : bism. carb. 10.0, talc. venet. pulv. 20.0, aq. rosar. 70.0, spir. colon. 3.0. The moist precipitate is brushed in the skin.

Tincture of iodine or iodine-glycerin (iodin. pur., kali iodidi āā 5 0, glycerin. 10.0) is applied twice a day for three to six days. After exfoliation of the brown scale the skin is usually red and scaly, and is then treated exclusively with the cosmetic remedies until we may again enter upon a new course of irritant treatment with sulphur, iodine, etc. According to the severity of the case, recovery will be complete in six to twelve weeks after four to eight repetitions of the treatment.

Acne due to tar, iodine, and bromine requires symptomatic treatment, the application of cold when the inflammation is violent, and of lead and zinc salves in ulcerative destruction of the bromide eruption. I have seen rapid resolution of firm bromide infiltrations and excrescences after the use of emplast. hydrargyri, but have often been compelled to remove them with the thermo-cautery. This may also be recommended, in conjunction with precipitate ointment (5 : 50) and spir. sapon. kalin., in acne varioliformis.

A disease in horses, known to veterinarians as "horse-pox," consists of an acute eruption of flat vesicles on an inflamed base, upon the back, shoulders, and in the mane. This has been studied by Diekerhoff and Grawitz, who call it acne contagiosa. So far as I have seen, the disease does not resemble the acne of man, but is more like impetigo contagiosa.

ACNE ROSACEA.

Acne rosacea (gutta rosea) is a chronic disease of the hairless parts of the face, especially the nose, cheeks, glabella, and chin, and

sometimes extending to the side of the neck. It is characterized by the production of bright-red to dark-red patches, which pale under the pressure of the finger, and are either uniform or traversed by distinct vascular twigs ; there are also red, soft elastic nodules, or even large tubercles and outgrowths.

We recognize three grades of the disease. The *first grade* consists of a uniform, diffuse redness of the tip of the nose and its immediate vicinity. The patients labor under the mistake that the nose has been frozen. In some the redness extends diffusely over both cheeks, ears, and chin. After a time newly-formed sinuous vessels are found. After extreme changes of temperature these patches become darker and cause a feeling of heat and burning. The process may remain in this stage for many months, or even years, and may then disappear completely, or it may develop into the higher grades.

In the *second grade* bright-red, firmly elastic, non painful nodules, from the size of a lentil to that of a pea, gradually develop upon erythematous patches. The nodules are either isolated or collected in dense masses, and their surface is covered with sinuous vessels. They are found on the cutaneous part of the nose, the chin, glabella, and cheeks.

In the *third grade* round and irregularly shaped, tumor-like, lobulated outgrowths of soft, elastic consistence develop upon the nose. The integument is traversed by abundant vessels, which may even be as large as a crow quill, and contains comedones and acne pustules. The organ may attain colossal dimensions ; the lobes may hang down to the upper lip and assume the most bizarre shape (rhinophyma). Another variety develops as a uniform hyperplasia of the cartilaginous part of the nose, which becomes wider and projects with a long, snout-like tip.

The smaller nodules of the second grade, as well as the lobulated new formations of rhinophyma, consist of new-formed, gelatinous connective tissue which is capable of organization into firm connective tissue, but may also undergo shrinking and absorption in the recent growths. In addition to this, the investigations of H. Hebra have shown that the essential anatomical basis of acne rosacea is distention and hypertrophy of the sebaceous glands, the new formation of superficial vessels in the skin, and even dilatation of the ascending corium vessels and their branches.

The *diagnosis* is not difficult, even if acne vulgaris is also present. Acne rosacea of the second grade may be mistaken for lupus or nodular syphilide. The acne nodules can be distinguished from syphilis by their extreme vascularity, soft consistence and compressibility, and the absence of cicatricial and ulcerative involution.

The *etiology* of acne rosacea is manifold. The first and second

grades often develop in females at the period of puberty and at the menopause, more rarely in the intervening period, and in connection with disorders of the sexual organs. In young people the causes are chlorosis, dysmenorrhœa, sterility, and diseases of the uterus and its appendages; in older people the cause is the physiological process of sexual involution. In exceptional cases it occurs in women who are sexually healthy. Chronic dyspepsia appears to predispose to acne rosacea in both sexes.

A well-known etiological factor, especially of the highest grade of the disease, is the excessive use of alcoholic beverages. In wine-bibbers the nodules are usually bright red; in beer-drinkers we find a more cyanotic rhinophyma; in whiskey-drinkers the nose is dark blue and smooth. It also occurs in persons who have resorted excessively, for years, to cold-water cures. Finally, the condition is observed in those who are exposed a good deal in the open air (drivers, engineers, sailors, masons, etc.).

Physiologically the process depends upon a paretic condition of the finest cutaneous vessels in the most peripheral parts of the body,

FIG. 31.—HEBRA'S PUNCTURE NEEDLE.

associated with sluggish circulation in these parts. Hence it is occasionally due to the same causes which give rise to chilblains, etc.

The *prognosis* of the first and second grades is the more favorable the more readily the cause can be removed, for the reason that under such circumstances spontaneous recovery may take place. This cannot be expected in rhinophyma.

The *treatment* of the first and second grades must be directed against the causes and the local changes. The former indication is met by the treatment of diseases of the female genitalia, by the administration of bitters, iron, arsenic, milk and whey cures, etc., in chlorosis, etc. The slighter grades will improve under general treatment.

The local treatment aims to cause the disappearance of the diffuse redness, the telangiectatic vessels and nodules. The flattening of the red nodules is readily effected by the application of emplast. hydrargyri or the methodical application of sulphur paste, tincture of iodine, and iodine-glycerin, mentioned in the therapeutics of acne vulgaris. The iodine-glycerin is brushed on the skin eight to twelve times in three to four days, and then covered with gutta-percha paper. The sulphur paste and gray ointment may only be employed at night. During the day, and whenever the irritation causes red-

ness and scaliness of the skin, the cosmetic salves and powders already mentioned may be applied. In intense diffuse redness with telangiectases and large, firm nodules we must resort to methodical scarifications in repeated sittings, in order to cause obliteration of the vessels. We make numerous shallow, parallel incisions with a fine scalpel, or puncture with the puncturing needle, or tear the vessels by scraping with the sharp spoon. E. Veiel has devised an instrument for scarification, consisting of six parallel lancets which are fixed in a handle, and whose position can be changed by means of a screw. For scarification I prefer a fine scalpel or Vidal's scarification lancet; for puncturing I employ Hebra's puncture needle (Fig. 31), a strong, double-cutting, lance-shaped instrument. In puncturing we make numerous punctures rapidly following one another; in scarifying we make close, parallel incisions and then cross-incisions. The hæmorrhage, which is often considerable, is checked by compression with cotton. The application of a solution of silver nitrate or iron chloride to the open vascular surfaces is not advisable. After scraping, the surface is covered with tissue detritus, which soon becomes discolored, but is exfoliated in a few days after the use of cold compresses, ung. simpl., or emplast. hydrargyri. According to the severity of the disease, these methods must be repeated for weeks or months.

The deformity of rhinophyma can be relieved only by excision and by decortication, removal of the nodules and outgrowths in layers by means of the knife, or constriction of the prominent nodules by surgical methods. Severe hæmorrhage from the large veins is to be expected, and should be treated by a compress and bandage of aseptic cotton.

LECTURE XXVIII.

SYCOSIS—SIGNIFICANCE, PATHOLOGY, AND TREATMENT—SYCOSIS PARASITARIA—IMPETIGO—ECTHYMA—IMPETIGO HERPETIFORMIS.

SYCOSIS.

ACNE mentagra, folliculitis barbæ (Köbner), is a chronic disease of those parts of the skin which are covered with thick hairs. Inflammatory papules, nodules, and pustules, each of which is perforated at the tip by a hair, make their appearance, and extensive inflammatory infiltrations with suppuration, crusts, and occasionally papillary excrescences, are also formed.

The most frequent sites are the bearded parts of the face, more rarely the eyebrows, the vibrissæ of the nasal mucous membrane, the hairy part of the axillæ, the mons veneris, and the scalp.

In sycosis faciei a few red, inflammatory, painful nodules and pustules appear upon one or more parts of the cheeks, each being perforated by a hair. If this is removed its root sheath is seen to be thickened, vitreous, and appears to have absorbed pus. Not infrequently a little drop of pus escapes from the opened follicle. This process is converted into sycosis by its course. A few of the suppurating nodules open, are covered with crusts, and result in cicatricial destruction, while fresh nodules appear in the neighborhood and the disease becomes chronic.

The process thus spreads for a number of years over the cheeks, chin, and upper lip. The cheek appears irregularly swollen, nodular, covered here and there with crusts, with scattered or closely aggregated pustules, or red and scaly or moist. Most of the hairs are loose and easily pulled out. The numerous cicatrices and the ragged appearance of the beard increase the deformity. In addition there are sometimes larger abscesses and blood boils. A special peculiarity is the presence of red, moist, easily bleeding, papillary outgrowths, whose viscid secretion dries into thick crusts; these patches vary from the size of a quarter to that of a dollar, and they project two to four millimetres. These are found between the lip and chin, at the angles of the chin, and rarely on the cheeks. The hairs in such localities are loosened.

Sycosis runs an extremely chronic course. Ten to fifteen years may elapse before the process has extended over both cheeks and to the hairy part of the temple. Some cases advance more rapidly.

In the eyebrows the process is isolated, or associated with blepharadenitis and sycosis of the rest of the face. Sycosis of the nasal mucous membrane is usually combined with a similar affection of the upper lip. In the axillæ, on the mons veneris and the scalp, sycosis usually starts from an eczematous inflammation. But sycosis capillitii, with chronic nodular and pustular eruptions and painful infiltration of the cutis, also occurs in rare cases as an idiopathic disease.

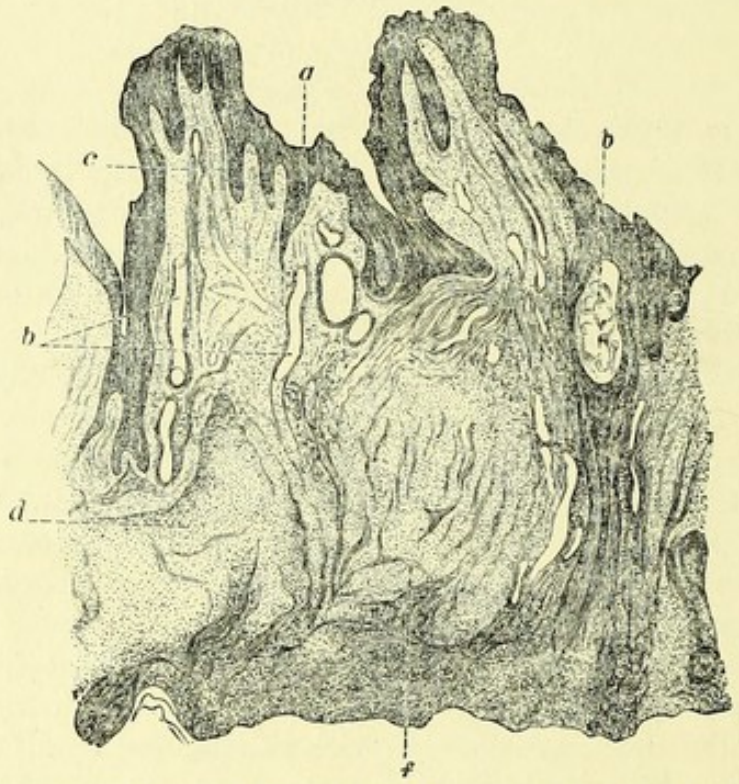

FIG. 32.—DERMATITIS PAPILLARIS CAPILLITII. LONGITUDINAL SECTION. (LOW POWER.)

a, hypertrophied epidermis over the dentated, projecting papillæ, *c*; *b*, enormously distended vessels; *d*, cell infiltration of the corium.

I must here refer to a peculiar form of disease which I have described (1869) as *dermatitis papillaris capillitii*. Papules as large as a pin's head appear upon the back of the neck, at the border of the hair, at first isolated, later closely aggregated. These coalesce into scar-like, keloidal, projecting, very firm, pale or red plaques, upon which the hairs appear tangled and matted together, while other parts appear entirely bald. The hairs are pulled out with difficulty, break off in the act, and appear twisted and tortuous. The nodules creak when incised, and the cut surface bleeds from numerous points. Here and there a small pustule is seen.

As a rule the process spreads up the occiput toward the vertex.

In one case I found it confined to the latter situation. During the spread of the disease on the occiput, papillomatous, readily bleeding vegetations develop; they are two to three centimetres in height, furnish a stinking secretion, and are covered with crusts. They are partly destroyed and undermined by the intercurrence of abscesses here and there. These structures are extremely vascular, papillary excrescences, and have the same microscopical anatomy as granulations. In the course of years they are converted into sclerotic connective tissue (Fig. 33, *a*), causing extensive atrophy of the hair follicles and baldness; in other places a few hairs remain, tangled and matted.

The process has been described by Alibert as pian ruboid and identified with syphilis, while Rayer calls it sycosis papillitii. The latter view is adopted by Hebra, who calls it sycosis framboësiaformis. I think I have proven that not alone is the condition unconnected with syphilis, but that it does not originate in follicular pustules—and hence is no sycosis—and is an idiopathic inflammatory process. The acne-keloid of Roget, Bazin, etc., is undoubtedly identical with this disease.

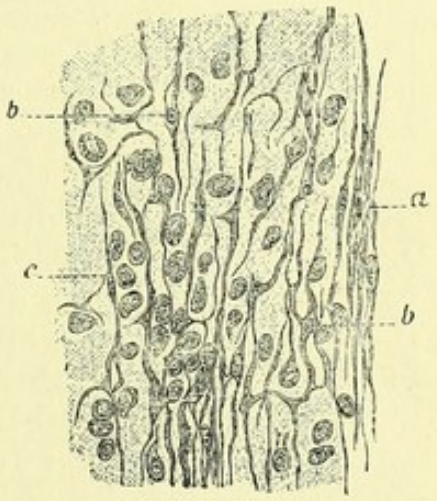

FIG. 33.—FROM THE INFILTRATED CORIUM IN DERMATITIS PAPILLARIS CAPILLITII.

a, round cells; *b*, mono- and polynuclear cells; *c*, the same, undergoing transformation into fibrous tissue.

Sycosis parasitaria (Bazin) is similar in its outward appearance to ordinary sycosis, but is characterized by the acute development of mushroom-like, fissured proliferations on the cheeks (Köbner, Kaposi, Lewin) or scalp (Auspitz, Lang), often combined with red, scaly circles. It is due to a fungus corresponding to herpes tonsurans, and will be discussed with the latter affection.

The *diagnosis* of sycosis is not difficult. The inflammatory infiltration and pustulation, the loosening of the hairs and thickening of their root sheaths, the chronic course as shown by the juxtaposition of recent or cicatrizing pustules, cicatrices and bald spots in the beard—all these symptoms sufficiently characterize the disease. But lupus and a nodular, ulcerating syphilide of the cheeks, lips, and nasal mucous membrane also exhibit crusts, cicatrices, and even papillary proliferations. The character of the latter must be taken into consideration in doubtful cases. In lupus the differential data are furnished by the primary papules, which do not disappear under the pressure of the finger; in syphilis, by the sharply defined, firm infiltration of the rim or by a characteristic, painful ulcer.

The *prognosis* of sycosis is favorable, because the disease is curable, and even its unimpeded course produces only local disturbances. Spontaneous recovery occurs only after the lapse of years and after extensive follicular obliteration. Sycosis of the nasal mucous membrane and the scalp is the most difficult to cure.

Ordinary sycosis is sometimes due to local eczema, inasmuch as folliculitis sets in with the long-continued or increasing inflammation. This holds good particularly in regard to sycosis of the nasal mucous membrane and upper lip, and sycosis of the scalp, axillæ, and mons veneris. Sycosis of the beard usually develops idiopathically, without any demonstrable *cause*.

Pliny maintained that mentagra was due to contagion. According to him, the disease was carried from Egypt to Rome, and was there spread epidemically by kissing. It is more than probable that this mentagra really consisted of mucous patches. However, this possesses merely an historical interest. Sycosis in the modern sense was generally regarded as non-contagious. The question of contagion has again been raised since Gruby (1842) reported a fungus in mentagra and Bazin established the existence of a sycosis parasitaria. Köbner has shown that the special form, sycosis parasitaria (which really belongs to herpes tonsurans), is alone contagious, but that this is not true of ordinary sycosis or folliculitis. The latter is usually idiopathic and is perhaps excited by certain anatomical conditions.

G. Simon and Wertheim have shown that every sycosis nodule is an abscess of the hair follicle. If the hair is pulled out, the root sheath is swollen and purulent, and a drop of pus escapes from the cavity. The interfollicular tissue, cutis, and papillæ exhibit inflammatory infiltration, and the latter occasionally become papillomatous. The inflammation is perhaps excited by a more vigorous reproduction of the hairs, the newly formed hair at the bottom of the follicle pushing out beside the old one and irritating the walls mechanically. In fact, two hairs are often found in the same follicle, and sycosis is more frequent when the beard is very thick. Wertheim states that the irritation is due to the fact that the transverse diameter of the hair is relatively too large for that of the follicle.

In sycosis parasitaria a fungus, the trichophyton tonsurans Malmsten, is found between the elements of the removed hairs and their root sheaths.

In the *treatment* of sycosis both patient and physician must be exact and persistent, and even an extensive process, which has lasted many years, may be entirely cured in three to twelve weeks.

Very moderately developed sycosis of the beard can be cured without removing the latter. The few pustules must be opened, the diseased hairs removed, and the spot thoroughly smeared with simple ointments until the excoriations have healed.

When the disease is more extensive and older, the beard must be taken off. It is first cut short. Then ung. diachyli is smeared on linen and bound down with flannel, salicylated plaster, or rubber cloth upon the cheeks and upper lip, in order to soften the crusts. At the end of twelve to twenty-four hours they may be washed off with soap, and the beard is then shaved.

The diseased surface now appears diffusely reddened, infiltrated, covered with numerous pustules, weeping or bleeding in places, and moderately painful on pressure. The next step is epilation, by which the diseased hairs are removed and the escape of pus from the follicles is made possible. The patient is placed in a good light. With the fingers of the left hand the physician makes a part of the integument tense, grasps the epilating forceps with the thumb, index and middle fingers of the right hand, and removes each hair in its natural direction. He supports the hand with the little finger, and places each extracted hair upon the skin without removing the hand. The operator thus has the advantage of retaining the direction of traction and removing twenty to thirty hairs in rapid succession. After the blood and pus have been wiped off and a brief rest given to the patient, the epilation is continued. On the first day the sitting is short, because many patients become excited or even faint away. In the following sittings no difficulty will be experienced. The pain is very slight, because the hairs are already loose. After epilation the part is washed, and, if there is severe inflammation, cold compresses or liq. Burowii (ten per cent) applied for a couple of hours, and then ung. diachyli or ung. vaselin. plumb.

These manipulations are repeated daily. The epilation is regularly advanced, not done here and there. The well-epilated patch will look better even on the following day, and the patient will acquire confidence in the treatment. In this way general epilation of both cheeks may be completed in two to three weeks. When the skin is entirely soft, no pustules are present, and the hairs, which always grow again, are firmly inserted, then the sycosis is cured. It is then merely necessary to apply simple emollient and protective salves and powders. The beard must continue to be shaved for at least a year, or the sycosis will return with its growth.

In many cases other measures are also necessary. Firmly infiltrated places must be punctured or scarified before they flatten and fade; freely bleeding patches must be scraped with the sharp spoon; larger abscesses must be opened. When there is an obstinate recurrence of numerous pustules, or diffuse firmness of the skin continues, it is advisable to apply sulphur paste or ung. Wilkinsoni, as in acne, or to wash with iodine-sulphur soap, or to excite acute inflammation by the application of soft soap for twelve hours, tincture of iodine, iodine-glycerin, or corrosive sublimate 0.50 : 100.0 aq. destil. Then

the above-described treatment with salves and epilation is again indicated.

The vegetations referred to are removed by cauterization with acetic acid, a paste of acetic acid 10.0, lact. sulph. 2.50 ; a salve of cupri acetat. 0.30, ung. simpl. 10.0 ; or with concentrated muriatic acid followed by dusting with calomel or by scraping with the sharp spoon.

In sycosis vibrissarum the pustules must be opened, the hairs removed, emollient salves placed in the nostrils, as in eczema of this part, and rhagades are cauterized.

In sycosis capillitii we attempt to soften the pustules by oils, a rubber cap, washing with spir. sapon. kalin. ; and only when these measures prove unsuccessful do we proceed to shaving and epilation. During the day a wig is worn on the scalp, which has been covered with ointment or rubber cloth. Sycosis of the axillæ, eyebrows, and mons veneris is treated in a similar manner. Wilkinson's ointment is especially adapted to these regions.

In dermatitis papillaris capillitii, emplastrum hydrargyri is the only remedy which has produced any effect. The bulk of the papillary and keloid-like growths must be removed with the scissors and knife. The free parenchymatous hæmorrhage is checked with simple cotton tampons, the additional application of nitrate of silver 1 : 1, or the Paquelin cautery.

In sycosis parasitaria, epilation of the diseased hairs, with application of sublimate solution or acetic-acid-sulphur paste, will prove successful.

A few remarks in regard to

PUSTULAR ERUPTIONS

naturally follow at this time. Since the time of Willan many writers have applied the terms impetigo, ecthyma, psydracion, phlyzacion to an eruption of a few pustules or to extensive disease with numerous pustules, particularly when localized on the lower limbs.

For us it is sufficient to remember that pustules—*i. e.*, epidermic vesicles with purulent contents (phlyzacion, impetigo)—and firmer purulent nodules (ecthyma) and furuncles occur wherever diffuse or circumscribed acute inflammation and exudation occur in the upper part of the corium and in the papillary layer. They are idiopathic in traumatic and caloric inflammation of the skin ; as a result of scratching in eczema, prurigo, scabies ; after inunctions of mercurial ointment ; in certain affections in which the inflammatory factor is found in the skin itself, as in acne and sycosis. They are found symptomatically in variola, glanders, syphilis, and metastatic processes. In all these conditions the pustules are only local

symptoms and constitute an increase of the inflammation, which has reached the stage of suppuration. We are not opposed, therefore, to the use of the terms impetigo and ecthyma to indicate the temporary character of an eruption, if this consists in an outbreak of pustules; but, in addition, we should employ the designation of the primary process or the exciting cause—for example, ecthyma pustules of the lower limbs in consequence of prurigo or clothes lice, or impetigo as the result of eczema or head lice. Such impetigos and ecthymas are not independent diseases. Only one form is a disease *sui generis*—viz., that to which Hebra applied the term

IMPETIGO HERPETIFORMIS.

Fifteen cases have been observed in our clinic. All occurred in pregnant women, usually in the last months of pregnancy, and terminated fatally, with the exception of the cases which will be mentioned later.

I have seen one case in a male, a cachectic young man of twenty years. In this case the disease had begun in the fold between the scrotum and thigh, with symptoms which resembled eczema intertrigo. It was so characteristic, however, that I made a diagnosis of impetigo herpetiformis despite its occurrence in a male. The further course was the same as in the female patients, and death occurred at the end of two months. A second case, in a boy of eighteen years, was reported by Pataky, Geber's assistant.

The following symptoms were observed in all the cases:

Little pustules, as large as a pin's head, with opaque, later greenish-yellow contents, develop in the groin, at the umbilicus, the breasts and axillæ, later in many other parts of the body. They are situated on a red, moderately swollen base, and are at first confined to patches from the size of a lentil to that of a penny. In one or two days they dry into a dirty-brown crust, while similar ones appear immediately around them in a single, double, or triple circle; the desiccation of the latter enlarges the central crust. The lesions thus spread, like herpes circinatus, over large areas and coalesce with adjacent ones. Beneath the detached crusts the skin appears red and covered with new epidermis, or it is devoid of epidermis and weeping, as in eczema, infiltrated and smooth, or papillary; it is never ulcerated. At the end of three to four months almost the entire integument is involved; it is swollen, hot, covered with crusts, and contains excoriated and fissured surfaces which are still surrounded, here and there, by circles of pustules. In some of the cases the mucous membrane of the tongue, palate, velum, and posterior pharyngeal wall contained circumscribed gray patches with a central depression. In the boy of twenty years mentioned above there were found at the autopsy groups of pustules and little ulcers upon

the folds of the œsophagus ; these were most numerous near the cardiac extremity of the stomach. The disease is accompanied by continued or remittent fever, with intercurrent chills and high temperature prior to the fresh eruptions, dry tongue, vomiting, and delirium. In addition to the characteristic eruption, I observed erythema læve and urticatum in the young man just mentioned and in one female patient.

The nature of this peculiar process has been freely discussed in the last few years. All writers, even including Duhring, no longer place it in the latter's category of dermatitis herpetiformis, but regard it as an independent disease. I cannot regard the dermatitis herpetiformis of Duhring (any more than the opinion previously expressed by him, and also, particularly, by Brocq) as a well-defined clinical entity, but as a purely theoretical formula which includes processes with entirely distinct causes and significance. As in the case of erythema, herpes iris and circinatus, the various forms of pemphigus, concerning the cause of which we know practically nothing, so in impetigo herpetiformis, it is necessary to hold fast to these clinical differences.

The *prognosis* of impetigo herpetiformis is bad; among the fifteen cases under our observation thirteen terminated in death. Two women apparently recovered permanently ; in another a relapse and death occurred a few weeks after recovery ; in still another the disease went on to recovery in two successive pregnancies, but terminated fatally in the third.

Parturition did not affect the course of the disease in any of these cases. In one case the autopsy showed endometritis and peritonitis ; in the others no explanatory lesion was found. In the young man mentioned above, the autopsy revealed extensive tuberculosis of the peritoneum and chronic pachymeningitis with serous infiltration of the brain. In the cases observed during the last few years numerous bacteria and cocci were found in the contents of the pustules.

On account of the almost exclusive occurrence of the disease in pregnant women, it must be regarded as analogous to other skin diseases which are excited by uterine conditions—for example, pemphigus hystericus, pemphigus gestationis, certain forms of erythema and herpes—and which must be regarded as neuroreflex dermatoses.

There will always be a certain analogy between these processes, impetigo herpetiformis, and certain others which clinically may be very different. With Hallopeau's " Dermatite pustuleuse chronique en foyers à progression excentrique " such an analogy exists, I do not wish to deny. In this connection C. Heitzmann and Fr. Schwarz reported two interesting cases in which impetigo herpetiformis had run its course and then a rapidly fatal pemphigus developed ; fur-

thermore, Pataky's case, in which vesicles and hæmorrhagic vesicles appeared as in herpes iris ; finally, my case, in which erythema urticatum was associated with the pustular eruption. In favor of the interpretation of the disease as a metastatic pustulosis (Neumann) there are only two findings, including that of the young man with tuberculosis of the peritoneum.

The theory that we have to deal with an infectious disease is not to be discarded entirely, especially in view of the pustular eruption, which reminds us of metastases, the high fever, and the fatal course. Hitherto, however, we have been unable to obtain any objective support for this theory. Schwarz, Du Mesnil, Marx, I. and others have found cocci, bacteria, and vibriones in the pustular contents, but this is not very significant in view of the fact that many micro-organisms occur normally in the upper layers of the epidermis.

The *treatment* consisted in all cases of antiphlogistic applications, later of soda and simple continuous baths, dressings of simple salves, carbolic acid, and tar, in addition to remedies directed against the fever and the general enfeeblement of the organism.

LECTURE XXIX.

4. VESICULAR ERUPTIONS.

PEMPHIGUS.

DEFINITION OF PEMPHIGUS—GENERAL DIVISION INTO PEMPHIGUS VULGARIS AND FOLIACEUS—GENERAL SYMPTOMATOLOGY—SPECIAL FORMS OF PEMPHIGUS AND THEIR PATHOLOGY, ANATOMY, DIAGNOSIS, PROGNOSIS, TREATMENT.

PEMPHIGUS is characterized by the repeated outbreak of vesicles upon the integument and the adjoining mucous membrane. It is a chronic affection. Hence this does not include the disease previously described (page 258) as acute pemphigus.

The great difference in the symptoms has led to a division into numerous varieties. No less then ninety-seven were enumerated by H. Martius.

If we hold fast to the most essential symptom—*i.e.*, the appearance of vesicles upon the general integument—it cannot be questioned that all cases may be divided into two categories. In one the individual vesicles pass through a typical course of development and resolution, which terminates in complete restitution of the epidermis at their base. This form we call pemphigus vulgaris. In other cases this regeneration does not take place. The epidermis, starting from the originally affected spot, is progressively detached, so that the corium is laid bare, red and weeping, in a centripetally advancing zone. These symptoms represent pemphigus foliaceus.

PEMPHIGUS VULGARIS

is characterized by well-developed, tensely filled vesicles. In the majority of cases the process runs the following course :

The disease begins with febrile symptoms—chill, high temperature, accelerated pulse, nausea, vomiting, etc. The fever also accompanies later eruptions with a continued remittent, sometimes typical intermittent type. It falls gradually with the subsidence of the vesicular eruption, and increases suddenly before each new eruption.

In the majority of cases the general integument presents bright-red patches, also a few wheals, which develop into erythema an-

nulare, gyratum, figuratum, urticatum, and return repeatedly in different parts of the body during the entire duration of the vesicular eruption.

The vesicles appear in part upon the erythema patches and wheals, in part upon apparently unchanged skin. Their size varies from that of a bird shot or a pea to that of a hen's egg or even larger, and their number varies from a few to a hundred or more. They are irregularly scattered (pemphigus disseminatus), but are very often symmetrical on both sides of the body, both as regards number and localization. In places they may be closely aggregated (pemphigus confertus, pemphigus en grouppes). In rare cases three to five vesicles are ranged around an older central one (pemphigus circinatus), and, after healing of the central vesicle, they form circles and later sinuous lines of vesicles (pemphigus gyratus, serpiginosus).

Each vesicle retains its original size, or increases either by inclusion of adjacent vesicles or by its own growth. It runs a typical course. The clear, watery or wine-yellow, sometimes slightly hæmorrhagic contents (pemphigus hæmorrhagicus) soon become cloudy; in one to two days they become cloudy like pus, the top of the vesicle dries, or it ruptures and then dries into a crust with the exudation and blood. After this has fallen off the skin appears covered with young epidermis and bluish red over a disc-shaped space corresponding to the base of the vesicle. Later this spot exhibits brown pigmentation for a few weeks.

The process continues in this way, increase of the fever and of the erythematous and vesicular eruptions alternating with remissions. Where the vesicles and crusts are in close proximity and exudation is retained beneath the latter, the skin is hot, œdematous, painful; lymphangitis is present and is complicated with adenitis. Among the subjective symptoms may be mentioned more or less violent burning and itching at the site of the vesicles, pain and tension in parts closely covered with vesicles and crusts or excoriated by their detachment, insomnia, anorexia, thirst during exacerbations. These symptoms, with the fever and direct vital losses, reduce the patient considerably.

The disease terminates at the end of two to six months. The fever ceases, the vesicles are no longer produced, sleep and appetite return, and the patient rapidly recovers. There may be only a single attack, but usually new attacks recur at intervals of a few months or a year, and then the disease is ended. Otherwise the eruptive periods subsequently follow each other very closely and the disease becomes a continuous pemphigus (pemphigus diutinus). The form of pemphigus just described corresponds to the *pemphigus vulgaris benignus* of the author and the *pemphigus idiopathicus dispersus infantum* of Schuller.

The disease may exhibit a much milder form. Its course may be brief and apyrexial, and only a few vesicles may appear during the eruptive periods; or the vesicles may appear for many years, even for a lifetime (pemphigus diutinus), but only a single one or very few at a time. In the extremely rare pemphigus localis a few vesicles appear upon the bluish-red, cool skin of a circumscribed part, usually the fingers, toes, or nose.

In *pemphigus vulgaris malignus* the dangerous character is expressed by various factors: an extremely large number and constant reproduction of the vesicles, continued fever, rapid exhaustion (pemphigus cachecticus infantum of Schuller); or it may be converted into pemphigus foliaceus, inasmuch as the corium remains red and bare at the site of the vesicles or is covered with a grayish-yellow croupous exudation (pemphigus crouposus), with or without firm infiltration of the cutis and destruction of its upper layers (pemphigus diphtheriticus). Finally, there are forms in which, upon the exposed cutaneous surfaces of the axillæ, groin, or other parts of the body, fungoid, partly necrotic proliferations develop in a luxuriant manner and discharge a viscid secretion which sometimes has a rancid odor (Hebra, myself). These either remain stationary for a long time or they rapidly spread in a serpiginous manner (pemphigus papillaris vegetans of Neumann). All these forms may terminate in recovery, but they usually end fatally.

A fatal termination is especially frequent in *pemphigus vegetans*. In this variety, groups of vesicles, from the size of a lentil to that of a penny, develop upon a red base without any demonstrable cause and without prodromata. The vesicular contents become cloudy in one to two days, and during this time the base is elevated like a wheal. New vesicles form at the periphery, and at the centre, after the epidermis has fallen off, a moist, red or grayish, raw surface remains visible; this is due to proliferation of the epidermis and outgrowth of the papillæ. In this way the patches grow to the size of the palm of the hand or even larger; their edges unite with those of adjacent lesions into serpiginous lines. In the centre we often find that the process ends in flattening and the formation of new epidermis with dark pigmentation, so that only the serpiginous margins are left. Recovery may occur in such cases. In the majority of cases, however, the patient dies in a few months from the exhaustion due to the great loss of serum, or from nephritis, pulmonary œdema, etc.

Papillary vegetations in pemphigus do not warrant an absolutely unfavorable prognosis; they may originate from any form of pemphigus, especially from pemphigus circinatus and from those forms which are localized in the joint flexures. But in those cases in which

the vegetations spread serpiginously and do not acquire a new epidermis, the termination is almost always fatal.

In these cases the first plaques appear usually on the alæ nasi, the lips and surrounding parts, the soft palate, and around the anus. These are very easily mistaken for syphilitic plaques (condylomata lata). The same lesions are soon exhibited upon many other parts of the body, and in a few weeks we will find, in addition to the above-described papillary, moist, raw surfaces, the lesions of pemphigus foliaceus et crouposus, even large, isolated pemphigus vesicles. Death occurs at the end of a few months.

Pemphigus pruriginosus is also a grave variety. The violent itching which characterizes it is not alone a very annoying and debilitating symptom (on account of the disturbance of sleep and the nervous irritability), but it also exhibits an entirely distinct clinical history. Vesicles are very rarely formed, because the patients at once scratch the urticarial wheals upon which the vesicles are beginning to rise. On the other hand, we find all those lesions which are common after protracted itching and scratching (pruritus cutaneus and prurigo), viz., excoriations, scaly and crusty eczema, brown pigmentation in streaks and patches, dryness of the skin. All these appearances are scattered irregularly over the body.

PEMPHIGUS FOLIACEUS.

This disease is characterized by flaccid bullæ, whose scanty contents accumulate in the dependent parts and soon become cloudy. A new formation of epidermis does not take place at the base of the bullæ. This is owing to the fact that, from the original boundary of the bullæ, the separation of the epidermis advances peripherally. It is thrown into folds, like the layers of short dough ("patisserie feuilletée"); hence the term "foliaceus" used by Cazenave. In this way the corium is soon denuded over surfaces as large as the palm of the hand. As in eczema rubrum, it appears red and moist, and the scanty secretion dries here and there into thin, varnish-like crusts which are easily torn. Delicate layers of epidermis also form in certain places, but they are soon removed, either mechanically or by fresh exudation. After the lapse of many months or years the process occupies the entire body. A vesicle is nowhere visible, because the epidermis is not thick enough to be protruded. It is torn at once. The general integument is traversed by irregular tears, composed of small segments of a circle, while the enclosed areas are partly covered with crusts, or they are moist, or brownish red and dry, or they look like parchment. The hairs on the scalp are thin and have fallen out in great part, the eyelids are ectropic, the nails thin and brittle, the individual emaciated. The patient is in great pain while lying down or turning, on account of the tearing of the loose covers of the

vesicles and the crusts. The fever varies ; at first hardly noticeable or intermittent, at a later stage it becomes continuous.

Next to pemphigus crouposus and vegetans, pemphigus foliaceus is the most severe form of the disease. It almost always ends fatally, although, thanks to the advances in therapeutics, in recent years we have succeeded in a few cases in securing recovery or temporary improvement.

Pemphigus foliaceus develops as such from the start or from pemphigus vulgaris after the latter has lasted many years. It often develops out of pemphigus circinatus, because the fresh detachment of the epidermis at the boundary of the central bullæ interferes anatomically with the regeneration of the epidermis in the centre.

Bullæ also develop upon the mucous membrane of the mouth, nose, pharynx, and larynx (pemphigus mucosæ) in pemphigus vulgaris as well as in pemphigus foliaceus crouposus and vegetans. Their epithelial covering is soon macerated, turns gray, is cast off, and leaves sharply defined, bright-red or gray discs. So long as they remain scattered and rapidly acquire new epidermis, they are simply annoying on account of the pain to which they give rise. There is danger of suffocation when the bullæ are situated on the epiglottis. The condition becomes extremely grave when the bullæ run the same course as in pemphigus foliaceus of the skin ; the epithelium of the isthmus faucium, posterior wall of the pharynx, and the epiglottis is diffusely detached, the mucosa is brownish-red and dry and looks as if varnished, the patient is unable to convey food or drink across the rigid mucous membrane, breathing is disturbed and the voice enfeebled or lost.

Pemphigus bullæ have also been observed on the conjunctiva palpebrarum. In one case, which I diagnosed as pemphigus crouposus and which was subsequently reported by Borysikiewicz, complete blindness followed as the result of gradual adhesion of the croupous conjunctiva palpebrarum to the bulbar conjunctiva. Mader's case terminated in the same way.

Pemphigus of the mucous membranes sometimes occurs separately or as a forerunner of pemphigus of the integument, but as a rule it is associated with the latter. There is no doubt that the pemphigoid disease of the mucous membranes may extend deep into the air passages, but this is probably very rare and may, as in Mader's case, be mistaken for croupous bronchitis.

Anatomically the bullæ of pemphigus are distinguished from those of herpes, eczema, etc., by their extremely superficial situation. Their roof is formed of the uppermost layers of the horny cells ; their base, of the rete, whose few ascending meshes are soon ruptured when the bulla becomes full, so that later it appears to possess only a single chamber. The epidermic prolongations from

the mouths of the follicles often hang as conical appendages from the under surface of the roof of the bulla. The corresponding papillæ are infiltrated with serum and traversed by wide spaces (Haight). It is evident, then, that in pemphigus only the uppermost layer of epidermis is lost, and no loss of substance or cicatrix develops, despite long duration of the disease. After pemphigus leprosus, however, cicatrices are left over, and Steiner has seen them in one case of pemphigus vulgaris. In pemphigus pruriginosus the bullæ are usually situated deeply, as in herpes iris and chronic urticaria. In a few cases Bärensprung, Hebra, and I have seen, upon the hands, arms, and trunk, hundreds of delicately grouped, pearly-white milium granules which did not disappear for weeks and months after the healing of the pemphigus vesicles. In pemphigus foliaceus et cachecticus I have observed, toward the end of life, numerous furuncles on the abdomen and deep-seated ulceration in other places.

In pemphigus vegetans Neumann and Riehl demonstrated intense proliferation and conoidal outgrowth of the rete and hypertrophy of the corresponding papillæ. This tallies with my findings (in 1869) in a case which probably belongs to this category, but was then regarded as syphilis vegetans (framboesioides).

Great attention has been paid to the anatomical and chemical structure of the contents of the bullæ, in the hope of finding a materia peccans. But the statements do not even agree in regard to the reaction of the fluid, much less in regard to its chemical constituents. The majority agree that these contents exhibit essentially the properties of the blood serum, a neutral or feeble alkaline reaction, precipitation of molecular albumin on boiling, scanty cellular elements in the clear fluid, more pus corpuscles and often red blood globules in the cloudy fluid. Heinrich states that the fluid has an acid reaction, due to free acetic acid. Among the older investigators, F. Simon and Raysky found albumin, phosphates, lactate of soda, chlorides, and cholesterin; Folwarczny and Schauenstein found leucin and tyrosin; Malmsten found crystals of uric acid; Bamberger and Beyerlein, free ammonia; E. Ludwig found urea, paraglobulin, and serum albumin. In fourteen cases of pemphigus in my clinic Lukasiewicz and Gollasch reported a marked increase of eosinophile cells as compared with the contents of bullæ due to other causes; in severe cases there was pronounced leucocytosis.

The results of urinary examinations, even in the same patient, vary to an equal extent. Raysky, Heller, and Hillier noted considerable diminution of urea in a few cases. Jarisch found nothing abnormal. Examination of the blood did not disclose any notable abnormality. Many of the findings, such as the diminished number of blood globules, must be attributed to the anæmia and impaired

nutrition. This is also true of the usual post-mortem findings. Hebra mentions anæmia of the muscles, flabby lungs and heart, serous infiltration of the brain. The waxy degeneration of the liver and spleen which is occasionally found (Hertz) is also to be regarded as an expression of the cachexia. The complications and immediate causes of death in some cases included pneumonia, tuberculosis, follicular ulceration of the intestines, acute Bright's disease.

The *causes* of pemphigus are involved in great obscurity. This is due in part to the relative infrequency of the disease and to the slight agreement of different cases with one another. From 1865 to 1877 there were treated in our dermatological service 103 pemphigus patients (79 males, 24 females) among a total of 30,362 skin cases and 278,295 patients in the entire hospital. Nothing positive could be learned concerning general causal factors, such as nationality, occupation, mode of life, etc. One-fourth of the cases occurred in females. Age appears to exert a decided predisposing influence, because pemphigus is much more frequent in infants and the newborn than in adults.

Contagion has not been demonstrated hitherto, either clinically or experimentally. Heredity of the predisposition is rare, but is undoubted. A man of twenty-two years, who had suffered from pemphigus since early childhood, stated that his mother, sister, maternal uncle, and half of the latter's children had suffered from the disease. Toward the end of the last century and since that time it has been repeatedly claimed that pemphigus was produced by chemical or mechanical disturbances of the urinary secretion. The excretory products, which were retained in the blood, were supposed to be excreted by the vicarious action of the skin and there gave rise to the pemphigus vesicles. This notion has received a certain measure of support from the fact that urea, uric acid, and free ammonia were sometimes found in the contents of the bullæ, and also that there may be a coincidence of pemphigus and renal disease (Riegel). Steiner saw a combination of pemphigus and periodical hæmaturia, and Beyerlein observed, after an attack of scarlatina in a boy aged nine years, a general outbreak of bullæ (whose contents had an alkaline reaction, due to free ammonia) associated with acute Bright's disease and uræmia.

The disease is sometimes undoubtedly connected with hysterical conditions (pemphigus hystericus), in so far as these depend upon anomalies of the female sexual organs. In some women the disease appeared regularly with each pregnancy and disappeared after parturition (Hebra); in Köbner's case it appeared twice shortly after delivery (herpes gestationis of Bulkley). Steiner has seen an outbreak of pemphigus during pyæmia and after variola.

In leprosy, Boeck and Danielssen observed pemphigus with cica-

trices at the site of the bullæ (pemphigus leprosus). In some of these cases a few vesicles developed upon anæsthetic patches either spontaneously or after pressure; in others they continued to develop for years as prodromes of the leprous disease proper.

In all these cases a relation between pemphigus and the nervous system may be assumed. It seems to be due to reflex irritation from the genital system through the medium of the sensory and vaso-motor (or trophic) nerves; or, as in leprosy, from direct irritation of the peripheral vaso-motor or trophic nerves by the leprous infiltration. In many cases of fatal pemphigus anatomical changes have been found in the spinal cord or sympathetic system (Marianelli, Jarisch, Schwimmer-Babes, Ferraro); pemphigoid eruptions have been observed during chronic myelitis (Chvostek, Déjérine, Brissaud, etc.); and, finally, traumatic and spontaneous neuritides (Weir Mitchell) are also accompanied by an eruption of bullæ. Schwimmer has even seen zoster, followed by general pemphigus, after contusion of the shoulder joint.

The relations of pemphigus to diseases of the nervous system are sufficiently evident, but hitherto we have only been able to recognize coincidences and not a relation of cause and effect. On the one hand, the bullæ following neuritis do not constitute pemphigus in the clinical sense; and, on the other hand, the lesions of the spinal cord found in pemphigus are extremely variable in regard to their character and anatomical site. Moreover, even this coincidence is very rare. In nine cases of fatal pemphigus N. Weiss and I examined the cord carefully and found anatomical changes (diffuse sclerosis) in only a single one. These changes were uniform throughout the gray and white matter, and the patient was a cachectic individual who had also suffered from cirrhosis of the liver and cancer of the tongue. Cases of myelitis rarely exhibit an eruption of vesicles, and still more rarely do they present the symptoms of pemphigus. Hence pemphigus may be referred to a neuropathic cause in only a few concrete cases.

Some cases convey the impression of an infectious disease by the sudden, unexpected onset and the rapid, grave course. This is particularly true of pemphigus crouposus and vegetans, which in a few months carries off a patient who had been in blooming health. Hence a search has been made for specific bacteria. In the contents of the bullæ and in the urine Paul Gibier found bacteria arranged like a rosary; Spillman found bacteria in the contents of the bullæ, the urine, and the blood in febris bullosa; Demme found diplococci in the contents of the vesicles and in the blood, and made pure cultures. Inferences concerning the etiological value of such findings are not yet warranted, especially in view of the large variety of spores and bacteria which have been found in the normal epidermis.

We are therefore compelled to say that even typical pemphigus may be due to various causes, apart from the cases in which the eruption is merely another manifestation of an allied anatomical or vaso-motor disorder of nutrition.

In a man of twenty-two years, who had suffered from prurigo since childhood, pemphigus vulgaris of the skin and mucous membrane with severe symptoms developed and lasted upward of a year. The prurigo disappeared during the course of the pemphigus and returned after its cessation. In two cases of lichen ruber planus I have also seen an eruption of bullæ which recurred for several weeks. In these instances the latter must be regarded as an expression of an increase in the local inflammatory process.

Syphilis is also a cause of pemphigus (pemphigus syphiliticus). It occurs as a symptom of hereditary syphilis, either congenitally or three to six weeks after birth The skin is ulcerated at the base of the bullæ. In adult syphilitics, bullæ rarely form over ulcerating nodules.

The forms of pemphigus last mentioned may be called sympto matic, while much the larger number are idiopathic—*i.e.*, their cause is unknown.

The *diagnosis* of pemphigus has become more difficult in the last few years, on account of the tendency to include it under Duhring's dermatitis herpetiformis. Some writers even doubt the existence of pemphigus. I need not repeat that the symptoms ascribed to dermatitis herpetiformis—polymorphous erythemata (papulatum, urticatum, etc.) with more or less violent itching—are among the constant prodromes of pemphigus bullæ. It is also said that a general picture of pemphigus is wanting, as is shown by the great variety of forms described, viz., pemphigus vulgaris, foliaceus, pruriginosus, etc. It must be remembered, however, that although such forms often occur as types, yet in the majority of cases almost all these types may occur at various times. For example, the first eruption may be pemphigus vulgaris with many erythemata, then comes a period with much urticaria, violent itching, and small vesicles (pemphigus pruriginosus), then an eruption in the shape of pemphigus circinatus, then pemphigus serpiginosus, and finally pemphigus foliaceus, croupo-sus, and a fatal termination. This very variety in the number and intensity of the eruptions belongs to the characteristics of pemphigus, together with the chronic, relapsing course and the danger to life.

The diagnosis is made by observation of the symptoms already described for the various forms of the disease. It is easiest when the chronic recurrence of the symptoms may be inferred from the presence of numerous bullæ, crusts, and pigment patches. Even then mistake is possible, because these appearances may be produced artificially, in a measure, by the daily application of blistering remedies

(cantharides, mezereum). This is done occasionally by the insane and by malingerers. During the first period of acute eruption the diagnosis should always be made guardedly, because it may be mistaken for urticaria bullosa, erythema bullosum, herpes iris and circinatus, impetigo faciei. In extensive, far-advanced forms and in general pemphigus foliaceus we must differentiate from eczema rubrum (pemphigodes), psoriasis universalis, pityriasis rubra; pemphigus pruriginosus must be distinguished from pruritus cutaneus, prurigo, chronic urticaria. We should also keep in mind all those affections in which bullæ occasionally develop—for example, over gangrenous or anæsthetic integument—also those diseases in which the formation of crusts and detachment of the epidermis are repeated in a chronic manner.

The *prognosis* can only be given in regard to the eruption present at the time, not in regard to the course and termination of the disease. Pemphigus vulgaris generally warrants a favorable prognosis, pemphigus foliaceus and pruriginosus a doubtful or unfavorable prognosis, because the latter forms usually exhibit a steady advance to a fatal termination. In no case, however, does pemphigus vulgaris furnish any data which will enable us to foretell its future course, hence the prognosis must be given with great caution and should refer only to the immediate future. When the bullæ are tense, not too numerous, and appear slowly (pemphigus benignus, hystericus, solitarius), and the disease runs an apyrexial course in well-nourished young people and infants, it offers a favorable outlook. When there is an abundant and constant recrudescence of flabby bullæ, with continued fever, loss of strength, and marasmus, the prognosis is unfavorable.

A few data which may be useful in judging the further course, duration, danger to life, and the fatal termination, are supplied by the results of our own experience.

In twenty-one years (1866–1887) I saw at the hospital two hundred and ten cases, and altogether more than three hundred cases. Among one hundred and eighty-two cases which were carefully studied there were thirty-five deaths (with autopsy)—*i.e.*, the mortality under our observation was seventeen per cent. The remaining eighty-three per cent can only be regarded as temporarily "improved" or "cured," and the majority of them terminated fatally. In my estimation not ten per cent of the cases really recover permanently.

In the *treatment* of pemphigus, whether symptomatic or idiopathic, we do not possess a single specific or direct curative remedy. When a causal affection may be assumed or demonstrated (pathological conditions of the female sexual organs), suitable measures are indicated. Otherwise we are restricted to symptomatic local and

general therapeutics. During the first part of the eruptive period, and when only a few bullæ are present, the application of dusting powders is sufficient. When the bullæ are in close proximity they should be punctured in order to relieve the feeling of tension. Patches which are covered with crusts and denuded, in parts, of epidermis are covered with indifferent ointments as in eczema. Cold compresses and general wet packs are suitable in cases of markedly inflamed integument, high fever, and extensive eruption. The continuous bath is an invaluable remedy in pemphigus foliaceus; it relieves the pain, diminishes the fever, and, by causing a return of sleep and appetite, carries the patient through the period of eruption, in which he would otherwise perhaps succumb. In this way we kept one patient alive for four years. At one time, not including shorter periods, he passed eight months, day and night, in the bath, to his great advantage. Protracted tar baths, also sulphur-tar (unguent. Wilkinson.), are very useful and sometimes curative in pemphigus pruriginosus. Baths of alum, corrosive sublimate, tannin, and sulphur are also useful in pemphigus vulgaris.

The febrile symptoms and accidental complications are treated by internal remedies, such as quinine, acids, iron, opiates, etc.

DERMATITIS EXFOLIATIVA NEONATORUM.

A disease known as dermatitis exfoliativa neonatorum (Von Ritter) must here be mentioned on account of differential diagnosis. Clinically it resembles pemphigus foliaceus, with which it has been identified by G. Behrend. It appears during the first few days of life, occasionally not until the second week. In the mildest grade it occurs as diffuse redness of the general integument, with fine desquamation of the epidermis. This is more marked in the face and the joint flexures, but also attacks the trunk. It gives rise to rhagades at the angles of the mouth and eyes and the nares. The buccal mucous membrane also exhibits epithelial opacities and exfoliation, occasionally vesicles. As a rule the exfoliation ceases at the end of one or two weeks, the cutis gradually growing pale and the epidermis becoming smooth and flexible. Again, the body grows cold, in children who were cachectic, from the start, and death takes place either with or without complications. In the higher grade of the disease the integument over large areas of the face, trunk, and limbs appears loosened by serous effusion, as after scalding. In places the skin tears, and then there is moderate escape of serum and formation of crusts. Regeneration of the epidermis and recovery are possible, but as a rule a fatal termination occurs within a few days. In the highest grade of the disease a moderate serous exudation beneath the loosened epidermis forms flabby bullæ, as in pemphigus foliaceus, while in the remaining parts the epidermis is

simply loosened and here and there is thrown into loose folds. These children usually die. I have repeatedly observed these forms of disease, and agree with Von Ritter that the affection is different from pemphigus foliaceus, while I do not believe that it is a pyæmic process. I regard it as an exaggeration of the physiological exfoliation of the epidermis in infants.

In a case of this kind Riehl detected a fungus growing into very narrow, long mycelia, and regards it as the cause of the disease. The diseases described under the names pemphigus epidemicus and pemphigus contagiosus neonatorum are perhaps due to such mycotic agents (pages 259 and 349), unless it turns out that they are produced by a micrococcus, which Almquist claims to have observed and to have inoculated.

The *treatment* must carry out the same indications as in ichthyosis sebacea—*i.e.*, the excessive loss of heat due to the general loss of epidermis must be combated by applications of poor conductors of heat (fat, cotton), the nutrition must be stimulated by artificial means, and the life of the child maintained until the epidermis is restored.

There are cases of general redness and exfoliation of the skin which are individualized with difficulty. They may be acute or chronic, and be attended with mild or severe symptoms, while they do not fall into the category of other cases of a similar nature (erythema toxicum, pityriasis rubra, psoriasis, lichen ruber, pemphigus foliaceus universalis). Besnier has recommended that the term "erythrodermia exfoliativa" be applied to such cases until their pathological character has been cleared up.

Vidal and Brocq believe that they have taken a step in this direction in describing an independent morbid process under the term "dermatitis exfoliatrice généralisée," or "Erasmus Wilson's disease." This develops suddenly, without any demonstrable cause, from the region of the joints. It is a diffuse, peripherally spreading, erythematous inflammation of the skin, which becomes general in a few days to a few weeks, and leads to exfoliation in large scales. It terminates in recovery in two to three months, sometimes not until the lapse of five to twelve months. Alopecia of the scalp, occasionally of the hairs of the body, opacity and loss of some or all of the nails, have been constantly observed, but the hairs are gradually restored. Severe cases are attended with gastric symptoms, diarrhœa and vomiting, fever, albuminuria, iritis, paraplegia, and in some cases by a fatal termination.

I have no personal experience with such a disease, but I may state that there are chronic relapsing erythematous and erysipeloid

dermatitides which are due to autoinfection (from sacculated pleuritic and peritonitic exudates and pus foci, arthritides, and other organic diseases), and which perhaps belong in part to the process described by Vidal and Brocq.

CLASS V.

CUTANEOUS HÆMORRHAGES.

DISEASES OF THE SKIN DUE TO EXTRAVASATION OF BLOOD.

LECTURE XXX.

SIGNIFICANCE AND ANATOMICAL CONDITIONS—CLINICAL FORMS OF CUTANEOUS HÆMORRHAGES—INVOLUTION—IDIOPATHIC AND SYMPTOMATIC FORMS—CONTUSION—INJURY—PURPURA SENILIS, VARIOLOSA, RHEUMATICA, SIMPLEX, HÆMORRHAGICA—SCORBUTUS—HÆMOPHILIA—HÆMATIDROSIS.

We have repeatedly spoken of cutaneous hæmorrhages in the discussion of small-pox, zoster, and erythema nodosum. In these and in some other processes the hæmorrhages constitute more or less grave complications; in others they are the essential symptom of disease.

In cutaneous hæmorrhage there is a free escape of blood from the capillaries and finest vessels. A rupture (rhexis) of the walls of the vessels may often be assumed or demonstrated. In other cases the red blood globules may pass through the uninjured walls of the vessels (diapedesis). Or there may be merely an effusion of bloody serum into the tissues; this presupposes a chemical decomposition of the blood within the vessels.

Rupture of the vessels may follow external mechanical violence (blow, compression, puncture) or the inability of the walls of the vessel to resist the internal blood pressure. This does not occur in general increase of the blood pressure, as in fever and in certain cardiac lesions, but may follow interference with the return flow of blood in certain regions – for example, in the capillaries of the head during an attack of coughing or an epileptic paroxysm, or in the lower limbs in varicose veins. A similar condition results from the relative increase of blood pressure owing to diminished power of resistance of the capillary walls—for example, when the epidermis, the protecting cover of the papillary vessels, is removed, as in the formation of bullæ; or when the nutrition of the vessel walls has suffered, locally in inflammatory foci, or in the dependent parts of the body when the general nutrition is impaired. In this category

belong the hæmorrhages occurring from diminished atmospheric pressure, as in ascending high mountains (hæmorrhages from the nose, lungs, conjunctiva, tips of the fingers), during balloon ascensions, local diminution of pressure by means of cupping, etc.

The hæmorrhage takes place between the layers of epidermis, into the meshes of the papillæ and corium, more rarely into the glandular cavities and the subcutaneous cellular tissue. The tissue elements are either merely separated or they are torn apart. Cutaneous hæmorrhages generally appear in quite sharply defined patches and streaks of varying size, in the shape of (1) *petechiæ*, bright-red or livid patches, from the size of a pin point to that of a finger nail, projecting very slightly or not at all above the level of the skin, and not disappearing under the pressure of the finger ; (2) *vibices* which look like streaks ; and (3) *ecchymoses* which vary from the size of a dollar to that of the palm of the hand. More rarely the hæmorrhages form small papules (*lichen hæmorrhagicus*) corresponding to the mouths of the follicles, or tumor-like, firm, or fluctuating swellings (*ecchymoma*). Finally, they may form superepidermic accumulations in the shape of hæmorrhagic bullæ. Free extravasation of blood, when the epidermis is intact, from the sweat glands or hair follicles (*hæmatidrosis*) is the rarest form of cutaneous hæmorrhage.

The hæmorrhagic patches continue in their original shape and size until the blood has passed through its physiological changes and has been absorbed. Changes in shape and size occur only as the result of fresh adjacent hæmorrhages. The patches change in color from bright red to bluish red, later to yellowish green and brown. These changes of color depend upon chemical changes in the extravasated blood, particularly in the hæmatin. The latter is given up by the red blood globules and colors the surrounding fluids, the clots of fibrin, and the tissue elements. After the latter are absorbed the hæmatin remains in the shape of orange-yellow or rusty-brown granules, either scattered or gathered into little clumps. The red blood globules may retain the hæmatin and shrivel into little clumps of granules, or the hæmatoidin is deposited in the shape of yellowish-red to ruby-red, rhombic prisms and plates (Virchow). Such pigments may disappear entirely if the hæmorrhage is slight and superficial. Permanent brown pigmentation sometimes remains after deeper and more extensive hæmorrhage into the corium. If the extravasation has accumulated in a large cavity the blood serum is first separated from the fibrin. This gradually shrivels after disappearance of the serum and removal of the blood globules to surrounding parts ; the fibrin is then absorbed. Finally, very extensive extravasations are encapsulated like cysts.

In regard to the exciting causes, cutaneous hæmorrhages are

idiopathic or symptomatic. In addition traumatic hæmorrhages are distinguished from those which occur spontaneously. The latter are generally known as purpura.

Idiopathic hæmorrhages are due to traumatism which mechanically ruptures the vessels and tissues, or to local obstructions to the circulation. The former variety includes contusions which are produced by violent pressure of the skin against a hard body, by a blow, etc. It occurs most frequently in those parts in which the cutis is particularly liable to injury—as, for example, over bony prominences. After pinching, hæmorrhage often takes place from the papillary body into the epidermis in the shape of a hæmorrhagic bulla. This soon bursts, or it dries with its fluid contents into a rusty-brown, crumbly mass which is cast off in one to three weeks. When the contusion is more severe the skin is raised into a red, firm, painful swelling, which sinks in at the end of one to two days and disappears after the well-known changes of color. The blood is diffusely extravasated, infiltrated. In very severe contusions the blood collects in a large cavity—ecchymoma. Gradual absorption may take place in the manner already described. Otherwise acute, painful inflammation of the surrounding tissues develops and results in an abscess. After this is opened the hæmorrhagic contents and the mechanically detached and necrotic shreds of tissue are cast off. More rarely the original pain subsides, and a tumor gradually forms which fluctuates in the middle and crepitates at the periphery. A firm wall is felt around the hæmorrhage, and the absorption of the latter is impeded by the development of a fibrous limiting membrane which itself secretes fluid. A hæmorrhagic cyst of this kind may remain permanently.

Contusions do not differ in appearance from certain spontaneous hæmorrhages. Their diagnosis is often necessary for forensic purposes, and is based upon the congruity of the hæmorrhagic patches with the assumed sources of injury and the time at which they are said to have been produced. Their position in the most projecting parts of the body, the history of a painful sensation at the moment of their development, aid in the diagnosis. The spontaneous hæmorrhages of purpura are distinguished by their simultaneous occurrence in places which are not commonly exposed to injury (flexor surfaces), and by the presence of very small, petechial patches in addition to the ecchymoma-like patches. Erythema nodosum s. contusiforme (the latter term is used on account of the resemblance to contusion nodules) is characterized by the predominant localization on the legs and the hyperæmia over the fresh lesions.

The *prognosis* of contusions is favorable, as a rule, and depends, so far as regards duration, upon the intensity and extent of the traumata. With the exception of those which are complicated with

inflammation, contusions do not require treatment. The popular method of compressing fresh contusions with the finger or a coin is entirely rational, because this distributes the extravasation over a larger area and thus facilitates its absorption.

Injuries with fine puncturing apparatus, such as the stings of insects, needles, give rise to hæmorrhages into the integument, because the blood cannot escape through the narrow puncture. Insect or leech bites are also aided by suction. The most frequent form is the flea bite—purpura pulicosa. These are punctate or as large as a pin's head, and immediately after their development are surrounded by a zone of injection. It is only after the latter fades and disappears that the central hæmorrhagic point is recognizable. After lying in a bed which is infested with fleas, the skin may be covered with flea bites and simulate purpura simplex. The diagnosis is assured by the uniformity of the points, their aggregation in places where the folds of the underclothing are more closely applied to the body, and the presence of injection zones. Similar petechiæ also occur in the Oriental plague, so that flea bites have become important of late in differential diagnosis.

Local circulatory disorders which cause abnormal increase of the blood pressure in a definite capillary district often give rise to hæmorrhages, the more readily when the supporting tissue of the papillary vessels is loosened and the epidermis is thin or has been removed. This category includes the local hæmorrhages in acute inflammatory and exudative processes following stasis in the capillaries in herpes, eczema, from granulating wound surfaces ; also the frequently recurring hæmorrhages in the legs as the result of varicose veins. They occur more readily when the veins are filled by standing or walking, or when their supporting tissue has become flabby on account of advanced age, severe disease, or childbed. Willan's purpura senilis seems to refer to this form. So long as the epidermis is intact and the cutis is sufficiently elastic, the frequent ecchymoses merely result in patches of brown pigmentation. But if the integument of the legs loses its elasticity and mobility on account of complicating inflammation, ulceration, and cicatrization, the hæmorrhages are apt to cause necrosis of the tissues and to produce ulcers which are healed with difficulty.

The sudden change in the circulatory conditions is probably the cause of the purpura which I have seen in the new-born, soon after birth, in the shape of numerous petechiæ resembling flea bites. Upon microscopical examination of the skin the extravasations were found in the upper layers of the corium, and the deep vessels contained infarctions of red blood globules (purpura neonatorum).

All these various forms of hæmorrhage require no *treatment*, because they undergo physiological absorption. Complicating inflam-

mation and pain are relieved by the application of cold; hæmorrhages in the legs are best relieved by the horizontal position.

Symptomatic hæmorrhages form part of a morbid condition of the general organism, or of vascular innervation, or of disease of an internal organ. This class includes the fatal purpura variolosa, which is due in part to the chemical decomposition of the blood caused by the specific blood-poisoning, in part to the changes produced by the former in the vaso-motor nerve centres (page 184). A similar interpretation is to be placed upon the petechiæ and livid patches of the skin in the Oriental plague, snake-poisoning, and septicæmia.

I have recently seen purpura of the limbs, eyelids, and trunk as prodromes of cerebro-spinal meningitis.

An allied condition is exhibited in the hæmorrhages, usually confined to the legs, which are observed in marasmic individuals who are suffering from tuberculosis, cancer, or intestinal affections. In these cases Kogerer has found thrombosis of some of the vessels, and regards this as the probable cause of the local hæmorrhages, *per rhexim*. This category also includes the occurrence of petechiæ after the use of iodine (iodisme pétéchial of Fournier), after the inhalation of benzoinated vapors (T. Fox), or during ergotism (Lailler).

Other forms of purpura present a more independent type and peculiar symptomatology. This is particularly true of

Purpura rheumatica (peliosis rheumatica of Schönlein, rheumatokelis of Fuchs). After slight fever or merely malaise, anorexia, poor sleep, and mental depression, drawing pains are felt in the joints of the knees and feet, with or without demonstrable swelling and exudation. After a few days bright-red patches (hæmorrhages), which soon grow livid and vary in size from that of a pin point to that of a lentil or even larger, appear upon the integument of the legs, in smaller numbers upon the thighs, the buttocks, the abdomen, occasionally upon the forearms. In mild cases the pains in the joints cease with the appearance of the extravasations of blood, and the latter disappear in ten to fourteen days. The latter usually reappear in two to three relapses inside of three to six weeks, together with a return of the joint affection and the fever, and the disease is then at an end. In some cases, however, the relapses may recur for three to six months, or even for years. I have observed periodical hæmorrhages from the kidneys during this disease. In one case they preceded the cutaneous hæmorrhages by six months, and in another case albuminuria attended the purpura, which lasted several years. In one girl a fatal termination was due to hæmorrhagic destruction and gangrene of the velum and the laryngeal mucous membrane. Henoch, Bohn, Lewin, etc., also mention hæmorrhagic

and other complications of internal organs, particularly of the alimentary tract (see page 221).

Apart from such cases the prognosis of purpura rheumatica is favorable, but nothing can be foretold with certainty concerning the duration of the process.

The diagnosis is not difficult if we bear in mind the connection of the rheumatoid pains with the hæmorrhages and their peculiar localization.

The etiology of peliosis rheumatica is as obscure as that of erythema multiforme. It is more frequent in young females, and particularly during the spring and autumn. The real agent which produces the change in the peripheral vessels (angioneurosis), so that their walls become suddenly and temporarily permeable to the blood, is entirely unknown.

The treatment is confined to sedative local applications (cold, opiated ointments and plasters), horizontal position, and rest. If the hæmorrhages return obstinately we may resort to ferri sesquichlorat. 0.5 to 150.0 aq. cinnamom., ext. secal. cornut. 0.1 at a dose in pill, ergotin 0.05 internally in divided doses, or hypodermically (ergotin 1.00, aq. destil. 10.0, half a syringeful).

Purpura simplex begins with moderate fever and general malaise, or without any noticeable disturbance of the general condition. Then hæmorrhages, in the shape of patches and streaks or wheal-like elevations with hæmorrhagic discoloration (purpura urticans of Willan), develop irregularly in different parts of the body; later they are found mainly on the lower limbs and hands. The process generally runs its course in ten to fourteen days.

In *purpura papulosa* (Hebra), or lichen lividus (Willan), there are hæmorrhagic, projecting papules which correspond to the follicles and are perforated by the hairs. They are located frequently on the legs of cachectic, senile, scrofulous individuals, in whom all inflammatory products on the dependent parts of the body are apt to be infiltrated with hæmorrhages.

Purpura hæmorrhagica — morbus maculosus Werlhofii — is placed, as regards intensity, midway between purpura simplex and scorbutus. It usually begins with general symptoms of depression and febrile movement. Hæmorrhagic patches, from the size of a lentil to that of the palm of the hand, appear upon the integument, the face usually escaping. The hæmorrhages also follow slight injuries to the skin. A characteristic symptom is the occurrence of hæmorrhages from the nasal, buccal, and pharyngeal mucous membrane, intestinal and renal hæmorrhages, and hæmoptysis. These conditions may be attended with high fever, syncope, and collapse, and may rapidly end in death. The majority of cases run a favorable course. Although purpura hæmorrhagica is sometimes regarded as

due to poor nutrition, this does not hold good of all cases, inasmuch as it often develops in previously healthy and vigorous individuals. It is generally sporadic, rarely endemic.

The term *scorbutus* is applied to purpura when there is early loosening and hæmorrhages of the gums, with a dirty-gray coating and a fœtid odor from the mouth, and when the cutaneous hæmorrhages are not only more extensive than in purpura hæmorrhagica and simplex, but also involve the subcutaneous cellular tissue, the muscles and fascia. In places we find ecchymoma-like, painful, firm or fluctuating extravasations which lead to gangrene, exposure of the bones, ulcers with a bloody base. Scorbutus occurs only as the result of poor or insufficient nourishment, a lack of meat, salt, fresh air, exercise, in sailors, convicts, etc. According to Uskow inflammation of the deeper vessels of the mucous membrane causes local disturbance of the circulation and hence hæmorrhages. Kretschy found the coloring matter in the urine increased prior to the appearance of the hæmorrhages, so that he assumes that the process begins with destruction of the red blood globules, and regards this as the cause of scorbutus.

In the last-mentioned forms the prognosis will be the more favorable the less rapidly and frequently the hæmorrhages appear, the more superficial their situation, and the less the impairment of general nutrition. In purpura due to septicæmia, grave jaundice, glanders, variola, scarlatina, and other infectious diseases, the prognosis does not depend upon the number and the repetition of the hæmorrhages, as such, but upon the intensity of the primary affection.

The treatment is not concerned with the already existing hæmorrhages, because they undergo spontaneous absorption. The chief aids are derived from nourishing diet and a stay in well-oxygenated air.

Hæmophilia (bleeder's disease) is characterized by the readiness with which slight mechanical causes, blows, or injuries cause considerable ecchymoses and free hæmorrhages which are checked with great difficulty. This predisposition is sometimes hereditary in certain families, and is found particularly in children and young people.

Hæmatidrosis, which means the occasional spontaneous oozing of arterial blood from the sweat glands, has already been mentioned (page 118). The affected parts are usually the eyelids, cheeks, back of the hand, and inner surface of the thigh. Messedaglia and Lombroso, who observed this symptom in a patient suffering from various neuroses, and hence regarded vascular paralysis as the cause of the spontaneous hæmorrhage (hæmatidrosis paralytica), obtained good results from the internal administration of belladonna.

CLASS VI.

HYPERTROPHIES.

CUTANEOUS DISEASES CHARACTERIZED BY INCREASE OF VOLUME.

LECTURE XXXI.

GENERAL REMARKS ON HYPERTROPHY—ANATOMICAL AND CLINICAL DIVISION ACCORDING TO THE IMPLICATION OF THE PIGMENT, OF THE EPIDERMIS AND THE PAPILLÆ, AND OF THE CUTIS AS A WHOLE—PIGMENT HYPERTROPHY—ANATOMICAL SEAT—NÆVUS—LENTIGO—EPHELIS—CHLOASMA—ADDISON'S DISEASE—MELASMA—APPENDIX: ICTERUS, ARGYRIA, TATTOOING.

By hypertrophy we mean those morbid conditions of the skin which manifest themselves as an increase of volume of the organ, or of some of its parts, which transcends the physiological limits. The condition presupposes an excessive formation of the normal tissue elements by reason of unduly increased local nutrition—hyperplasia. The augmented volume is based partly upon an enlargement of the several tissue elements (true or elementary hypertrophy), and partly upon their multiplication (numerical or quantitative hypertrophy). In this respect, therefore, it is at the same time a neoplasia in which there is a new formation of elements analogous to the physiological—*i.e.*, homœoplasia. The latter is not uniformly the case, for up to a certain degree of hypertrophy the organ and its elements may remain within physiological limits with reference to their quality and function; but when the hypertrophy becomes excessive the texture and function of the organ or its elements may deviate materially from the normal.

Hypertrophy of the common integument affects, either exclusively or largely, one form of its anatomical elements—pigment, epidermis, papillæ, glands—or else several or all of its constituents. In accordance therewith its clinical manifestation is very variable. In the present chapter we shall deal with hypertrophy of the pigment, of the epidermis, and of the papillary body.

PIGMENT HYPERTROPHY—HYPERCHROMATOSIS.

This appears as a coloration of the common integument more intense than the normal tinge, in the form of punctate or lentil-sized and larger spots, which may exceed in size the palm of the hand; they are sharply demarcated and range in color from various shades of brown to a grayish black, or these tints may be diffuse, and do not disappear under the pressure of the finger. The normal pigmentation according to race, individuality, and topography of the skin must be taken into consideration. The peculiar color of the skin is due to pigment deposited in the form of yellowish-brown granules in and between the cells of the inferior layers of the rete. In the light-complexioned Caucasian race the granules are sparse, more numerous in brunettes than in blondes, and hence such a skin looks whiter,[1] though with a reddish cast which is more or less pronounced according to the quantity of blood present. In general, however, the pigment is found more closely packed in certain regions, such as the areola of the nipple and on the genitals, which accordingly are darker in color. This applies also to negroes and the colored races in general, but even in them the pigment granules are not black. (Negroes are usually born with a light skin; from and after the sixth week of life the cutaneous pigmentation develops rapidly.) Pathological pigmentation likewise is based only on the increase and closer aggregation of pigment granules in the cells of the rete.

It is true, pigment is found also in the corium at all points which are normally or pathologically more deeply colored. This, however, contributes but slightly to the dark tint, though it is intimately connected with the process of pigmentation.

Until recently it was generally taught that all cutaneous pigment was ultimately derived from the blood, from the vessels of the papillæ and the superficial corium; that the red corpuscles, after their disintegration, whether within the vessels or after their diapedesis in inflammatory conditions, hæmorrhages, etc., give up the hæmatin derived from their hæmoglobin to the surrounding structures.

We can gain a pretty clear idea as to the path taken by the hæmatin into the mucous layer from the investigations of Demiéville, Langhans, Riehl, Ehrmann, and Quincke.

It can be demonstrated in most forms of hyperpigmentation that red blood corpuscles which have left the vascular channels, either singly (physiologically) or in masses (in microscopic or macroscopic hæmorrhages, in hyperæmia and inflammation), and have entered the tissue, are taken up, partly as such, by connective-tissue and lymph cells, to which they give up their hæmatin on their disintegra-

[1] The plausible explanation of this fact has been given by Kromayer (page 19), not by Max Josef, as a misprint makes it appear.

tion; partly, however, they have previously undergone chemical decomposition. It is, therefore, the lymph and migratory cells, or fatty granular cells (Philippson's mast cells), or large caudate cells (Ehrmann's chromatophores), projecting, according to this author, with protoplasmic processes into the layers of the rete, which carry with them the pigment derived from the red corpuscles in granular form or solution. A portion is given off again to the rete cells; another is added perhaps directly to the existing pigmentation of the mucous layer; while the cells are transformed into rete cells or remain in the interepidermal lymph spaces, undergoing degeneration and leaving their pigment there.

In the subepidermal zone of the papillæ and in the subpapillary portion of the corium we see such pigmented, rounded, caudate, and multipolar cells, partly isolated and discrete, partly arranged in networks and layers along the vessels and their most minute ramifications, often completely enclosing them.

These conditions, however, vary widely in the presence of physiological or pathological formations of different characters. Reported investigations show it to be pretty certain that the escape of red blood corpuscles is not continuous and uniform either in physiological or in pathological forms, but that it occurs only at intervals. Considering that the epidermis reforms and desquamates continually, while the pigmentation of the rete remains constant, the explanation of physiological pigmentation still lacks completeness with reference to the source and modus of the process. This remark gains in force by the fact that under normal conditions we find pigmented migratory cells in the corium in small numbers only. This circumstance, together with the findings in a number of pathological processes—*e.g.*, in acquired and congenital achromatia associated with certain irritative hyperæmias, etc.—which cannot be explained by a hæmatogenous origin of the pigment, long ago led me to think it probable that in the case of physiological pigment formation we would be justified in assuming a metabolic or functional autochthonous origin for the pigment from the basal rete cells (or in pigment tumors, from their cells); further, that such cells may lose this function, either accidentally or through inherited and congenital factors. At least for certain dyschromasiæ we would have to ascribe to the protoplasm of the epithelial cells a certain vital quality by which they are capable of retaining the pigment carried to them, because under some conditions they lose this quality temporarily or permanently, as in vitiligo. Moreover, the assumption of a loss of productive power is more probable on physiological grounds, as is also the productive power when nutrition is normal.

This theory has recently been materially strengthened by a number of developmental investigations by Jarisch, although Ehrmann

has attempted to deny the corroborative bearing of his painstaking researches. Ehrmann's opposing arguments, however, utterly fail to weaken the force of such clinical factors as I have adduced in support of the probability of an autochthonous pigment formation from the basal rete cells or their nucleus (Jarisch). Nevertheless a final settlement of the question as to the origin of the cutaneous pigment is still lacking. Until the question is definitely decided we must consider both of the above-mentioned sources of pigment formation as active. In my opinion the hæmatogenous origin of the pigment is more probable for some processes, the cellular origin for others.

As stated above, we must emphasize the presence, in conjunction with the scattered pigmented migratory cells found in all the more deeply colored areas of the skin, of trabeculæ of cells and nuclei along the course of the vessels of the subpapillary layer and the papillæ, often ensheathing these vessels and compressing them here and there so as to obliterate them (Demiéville). The trabeculæ exhibit at some points a homogeneous or else a fibrous intermediate substance. The latter forms at the same time the basis for the combination of pigment hypertrophy with connective-tissue hypertrophy, or the transition of the one into the other—*e.g.*, of simple pigment moles and warts into the corneous and verrucose moles of a different type.

The conditions here represented explain, first, why it is that every considerable and long-continued afflux in the papillary vessels, such as occurs in acute and chronic hyperæmia, inflammation, and vascular new formation. causes a more abundant deposit of pigment in the mucous layer and a deeper coloration of the skin ; second, why it is that in every intense pigmentation—whether acquired, resulting from inflammation, or congenital, as in pigment moles and warts—we can always demonstrate by the microscope a pigment deposit in the corium as well as in the epidermis. In other dyschromasiæ this explanation will not suffice, for the reason that the exudation of blood is lacking.

The pathological pigmentations of the skin which come under observation are either congenital or acquired.

Congenital pigment spots are called pigment moles, nævi pigmentosi (nævi materni). Their color varies from pale to dark brown and black There are several varieties : *nævus spilus*, the pigment mole with smooth, pliant surface, the skin being otherwise unchanged; *nævus verrucosus*, the mole with a rough, warty surface, often set with thick, bristly, dark hairs (*nævus pilosus*) ; *nævus mollusciformis, lipomatodes*, a prominent or even tumid mole. In the latter formations we find extending from the subcutaneous cellular tissue into the corium a gelatinous tissue deposit which looks

yellowish white in sections—*i.e.*, young connective tissue with delicate fibrils and numerous cells.

Pigment moles vary in size from that of a dollar or the palm of the hand ; they may even occupy whole regions of the body. Their outlines are very irregular, and thus they may resemble the shape of an animal (mouse), a fur rug, etc., and be ascribed to maternal impressions during pregnancy. When large, their direction generally corresponds with the course of the cutaneous nerves ; the mole sometimes being, like zoster, unilateral and parallel to the intercostal nerves or to the cutaneous nerves of the extremities ; or else, being sharply demarcated at the level of the umbilicus, surround the pelvis and upper thighs like bathing tights (Hebra's case) corresponding to the lumbar and sacral plexus—the nerve nævi of Th. Simon, (Bärensprung's nævus unius lateris). The assumption that such nævi are caused by trophic nerve influences has of late years become, as it were, an article of the pathologic creed, and has even been cited as a proof of the existence of trophic nerves and the neuropathic cause of many cutaneous diseases. There is, however, no actual proof of such a connection, though theoretically it appears quite plausible.

To me the matter appears in a different light. During the development of the embryo the tissues differentiate themselves everywhere, including the rudimentary extremities, into vessels, nerves, etc. As the extremity develops further and assumes its proper spiral direction forward and inward (Voigt), all the parts—papillæ, nerves, vessels, hairs, and connective-tissue trabeculæ—take the same course which is also that of Langer's cleavage lines, and thus we can understand that any marked alteration in shape and color of these tissues likewise follows this direction. Naturally the course is also that of the nerve, but it does not by any means prove a causal connection between the developmental anomaly and the nerve alteration. When the nævus is small its correspondence with the course of a nerve cannot be discovered, but the parallelism becomes more marked in proportion as its size increases.

If, then, such a part undergoes a local arrest or excess of development—whether one or all forms of tissue (papillæ, vessels, pigment, connective tissue) share more or less in this anomaly—it is clear that all these faultily developed formations take the same direction or correspond to the course of the nerves. Indeed, it is usually the case that every extensive nævus at various points presents different predominant characteristics—here more of a vascular, there more of a pigment, verrucose, or connective-tissue growth. This is certainly true when the nævus is general, as in the condition known as ichthyosis hystrix. In other cases, it is true, only one kind of tissue seems to be developed in the main ; for instance, the vessels in angiomatous, or the papillary layer in pigment and verrucose moles.

Even where a positive nerve alteration is found it corresponds with the type described, appearing as it does as a connective-tissue hypertrophy upon the nerve, a neuroma. This matter will be more fully discussed in connection with elephantiasis telangiectodes and fibroneuromata, all of them congenital pathological forms.

In explaining these general conditions in congenital nævi expressive of hypertrophy, I intended to show that the term nerve nævus is merely a conventional one, applying to the external appearance and not to the neuro-pathological element. On the other hand, there are acquired dyschromasiæ which undoubtedly depend upon a nervous influence, but in the sense of a reflex or sympathetic dystrophy, as in chloasma uterinum. Congenital pigment spots must be looked upon as analogous to unilateral heterochromia in animals (Hebra), as congenital development anomalies.

Pigment moles very rarely undergo involution after birth. On the contrary, most of them enlarge and persist unchanged through life, or at most grow darker under certain conditions (pregnancy).

Acquired pigment spots, in general called *chloasmata*, are of idiopathic or symptomatic origin.

Among the idiopathic pigment spots *lentigo* and *ephelis* (freckles) represent spontaneous formations. Lentigines are uniformly yellowish or dark-brown spots, ranging in size from a pin's head to a lentil and larger, disc-shaped and sharply demarcated ; they appear singly or in numbers scattered over the body, between the second and sixth years of life, undoubtedly on an embryonal base, and persist to advanced age. Ephelides are in general smaller than the former, pale brown, irregular, jagged, and unevenly tinged. Their most frequent seat is the nose, the adjoining skin of the face, and the forehead ; but on a delicate and fair skin (red-haired persons) they are also numerous on other parts of the face, the neck, chest, inner surfaces of the extremities, the dorsum of the hand, the nates, and penis—a fact which is sufficient to prove that they are independent of the influence of the sun. They appear first between the sixth and eighth years, become paler in winter and darker in spring ; they disappear completely in advanced age.

There is, however, no material difference between the truly congenital pigment spots and those arising in early life, either anatomically or in their course. As regards the anatomy, it has been stated above that the papillary body and the corium are involved in the mole, both by the deposition of pigment and by varying degrees of hyperplasia of one or all forms of tissue.

With reference to the course, it should be noted that both the fœtal pigment spots and those appearing in early life generally increase in number, intensity of pigmentation, and bulk (circumference and thickness). In proof of this we need but look at many persons,

particularly women. In girls of ten to fifteen years we discover here and there on the face lentigines and moles the size of a poppy seed; at twenty to thirty years these persons begin to complain of the disfigurement caused by the numerous pigment deposits, and in the fourth or fifth decade they have developed into good-sized warts. Even in advanced age there is no cessation in the transformation of the elements constituting the nævus, and then it happens frequently enough that the forms of lower organization—the epidermal tissues—predominate quantitatively over the more highly organized connective-tissue formations and take the place of the latter. Thus arises epithelial cancer, a destructive formation which frequently develops from warts and pigment moles.

Next in order to the congenital or early-life pigmentoses we have as idiopathic hyperchromatoses those pigmentations—mostly temporary, but often also permanent—which remain after local inflammatory exudative processes, such as eczema, psoriasis, pemphigus, and after hæmorrhages. Other pigment spots are artificially produced by local influences which cause intense and frequent hyperæmia of the papillary layer and the corium, and as a consequence darker pigmentation.

In accordance with their etiology we distinguish:

Chloasma traumaticum, produced by mechanically induced hyperæmia of the skin. To this class belong the dark staining of the skin resulting from long-continued pressure, as about the waist from straps, over the sacrum from trusses, and particularly the pigmentations due to scratching. The latter are among the symptoms of all itching skin diseases, scabies, prurigo, eczema, and urticaria, and appear as brown streaks or diffusely stained surfaces of a yellowish-brown, sepia-brown, and black tint (melasma). The pigmentation is more extensive and dark, and even melanotic, in proportion to the frequency with which the same region has been rendered hyperæmic by scratching, or injured by finger nails so that hæmoglobin has been directly effused. The color, too, is more intense in chronic diseases, such as prurigo and pemphigus pruriginosus, than in urticaria or scabies, and is especially dark in long-standing pediculosis corporis, when the skin is often to a great extent, especially over the sacrum and nucha, of a grayish black. This is, however, not a ground for calling this form melanosis, melasma cutis, or melanoderma, as if it were a separate disease, like the pityriasis nigra of some authors, which arises when the darkly pigmented skin shows a branny desquamation in consequence of eczema or cachexia. Inasmuch as the location of such scratch pigments coincides with the parts subject to itching and scratching—in pediculosis, chiefly the nucha and sacral region; in prurigo, the extensor surfaces of the lower extremities; in general prurigo, the greater portion of the sur-

face in scattered regions—and as, furthermore, the intensity of the color shows the order of involvement and the relative age of the excoriations, it will be evident that the scratch pigmentation, when correctly interpreted, furnishes a material help in the objective diagnosis.

Chloasma caloricum is the term applied to the brown coloration acquired by the face, neck, chest, arms, hands, and all parts of the skin exposed to the sun and the free air—so-called sunburn or tan. It frequently ensues after several hours' march in the sun, as in tourists and dwellers in cities. Chlorotic persons are less subject than healthy individuals. But the long-continued influence of raw and cold winds has the same effect, for which reason this chloasma occurs in all persons who are much in the open air, as hunters, soldiers after a campaign, sailors, coachmen, masons, etc. The pigmentation from this and the former cause disappears when the subjects are for some time removed from such influences.

Chloasma toxicum results from the influence of some specific irritating substances, such as sinapisms, cantharidal plaster, cortex mezerei, which are still in favor among physicians. The pigments, however, sometimes remain for life—a fact which must certainly deter the physician from applying a vesicant to the breast or face of a female patient, if he cannot make up his mind to eschew such applications altogether.

Chloasma symptomaticum is found as a concomitant symptom or sequel of some diseases of internal organs or the entire system, in the shape of limited local spots or a diffuse and general darkening of the skin. The most frequent and best-known form is *chloasma uterinum*, sometimes also called chloasma hepaticum (Alibert), from the similarity of the color to that of the liver. At times it occupies only a few places, at other times the whole forehead up to the border of the hairy scalp, in the shape of a yellowish-brown to dark-brown, uniform or irregularly streaked staining. It occurs besides on the eyelids, in the neighborhood of the angles of the eye, on the cheeks, the upper lip, and the chin, in streaks. It is quite frequent in sterile or unmarried women and those suffering from various irregularities in the sexual sphere—dysmenorrhœa, displacements, neoplasms of the uterus, ovarian affections, and hysteria—in some women also during gestation. After the menopause the chloasma disappears. The darker pigmentation occurring in the areola of the nipple and in the linea alba during pregnancy is also connected with the processes in the uterus.

Chloasma cachecticorum has been observed by us located in the face, precisely as in chloasma uterinum, in boys affected with lichen scrofulosorum. Otherwise this term is applied to the general dark discoloration of the skin occurring in marasmic persons after

malaria, in drunkards, in senile atrophy, and in the cancerous cachexia.

The pigmentation characterizing *Addison's disease* represents merely one of the symptoms of the general affection known by this name, which is still enigmatical. In 1855 Addison described the malady, stating that the constitutional and local changes found in it are the result of disease of the suprarenal bodies. The skin of the face, trunk, and extremities gradually assumes a diffuse olive-brown, sepia, or bronze-brown color—*teinte broncée*, bronzed skin disease—and even the mucous membrane of the lips, cheeks, and palate has a diffuse dark to blackish-brown tint. With this there occur also disturbances in other organs: diarrhœa, constipation, vomiting, symptoms of gastric and intestinal catarrh, pains in the abdominal organs, palpitation, anæmia, weak pulse, states of psychic depression, neurasthenia, general debility, emaciation, and with these symptoms or in coma, sopor, or suddenly as in shock, death ensues.

Thus far it has not been possible to prove the connection between the disease of the suprarenal bodies and the symptoms of Addison's disease, for in many cases in which the malady was pronounced those organs were not found affected (Overbeck); in others degeneration of the suprarenal bodies existed without such symptoms, and experimental extirpations and irritations of these bodies (Nothnagel) were not followed by pigmentation. Neither do the theories concerning the embryological and functional importance of the suprarenal bodies (hæmatopoietic glands or nervous formations, ganglia) furnish an explanation of the concurrence of their degeneration with the dark pigmentation or of the origin of this pigment, as has been recently shown by G. Lewin (1892) in a critique of the available literature. This much, however, seems certain, that, since such concurrence has been demonstrated, not every disease of the suprarenal bodies, but only their caseous degeneration, appertains to Addison's disease.

In recent years, as may be gathered from the latest publications by Nothnagel, the tendency is becoming more and more manifest, in view of the insufficiency of all former attempts at an embryologico-anatomical, hæmato-chemical, and physiological explanation, to trace the entire symptom-complex together with the pigmentosis in Addison's disease to an organic or functional disturbance of the abdominal nerve plexuses and ganglia (sympathetic, splanchnic, cœliac plexus, and semilunar ganglion), although even here the marked inconstancy of the findings and an equally marked divergence in their interpretation stand in the way of evolving and determining a theory, even partly satisfactory.

With reference to the derivation of the cutaneous pigment in

Addison's disease, aside from the views entertained regarding the formation of pigment in general, it has been claimed that the suprarenal bodies in particular furnished the pigment in some way, either by the decomposition of the blood corpuscles or by the formation of a special protein substance which subsequently changes into pigment (Averbeck, Riesel, Burger).

The anatomical investigations of Nothnagel and Riehl have shown that in Addison's melanosis the same conditions prevail as in melanosis from any other cause : pigment granules in the rete cells, pigmented migratory cells in the papillary body and the superficial corium, and this throughout, most plentifully along the blood vessels. If we bear in mind, in addition, that Riehl found in one case of Addison's disease a definite affection of the smallest cutaneous and papillary vessels—*i.e.*, of their adventitia and media—by which their permeability to red blood corpuscles is necessarily increased and microscopical hæmorrhages become possible, we must side with Nothnagel in ascribing the pigment in Addison's disease likewise to the red corpuscles which have escaped from the vessels and given up their hæmatin to the migratory cells and epithelia, in the way described above with reference to physiological and pathological pigmentation in general.

As the more remote cause of the vascular alteration and the diapedesis of red corpuscles which leads to the deposit of pigment. Nothnagel, as well as Jacoby, points to the abdominal nervous system, in the sense stated above, as producing reflexly by trophic influence the pigmentation in Addison's disease in the manner in which other pigmentoses are caused—*e.g.*, in that of pregnancy, chloasma uterinum, dependent on the nerves of the sexual organs.

As a symptomatic pigmentosis mention should also be made of "pigment syphilis."

Under this and similar designations (syphilide pigmentaire, tâches syphilitiques, syphiloderma pigmentosum) have been described, mainly by French and English authors and some of other nationalities (Monneret, Hardy, Pillon, Gibert, Bazin, Lancereaux, Fournier, Tanturri, Schwimmer, Atkinson; Drysdale, G. H. Fox, Duhring, and others), brown pigmentations of the skin which, in the opinion of most of these writers, arise like the specific exanthemata as the direct effect of the syphilitic diathesis and are said to be equally influenced by antisyphilitic treatment and so made to disappear. The spots are described as dirty-brown or brownish-yellow discolorations which appear isolated, reteform by coalescence, or diffuse ; they do not desquamate, occupy chiefly the neck, shoulders, face, and thorax ; occur usually early in the disease, last months and sometimes years, and usually yield with astonishing rapidity to the treatment instituted against the syphilis.

The pigmentations thus referred to have in part unquestionably merely the importance of the above-mentioned cachectic discoloration of the skin, since constitutional syphilis implies a variety of cachexia; hence they are not a direct effect of the specific virus, like a syphilide, for instance. Moreover, the fact that these pigment spots disappear with the cure of the syphilis makes this interpretation all the more permissible.

To a considerable degree, however, these dyschromasiæ reported as pigment syphilis may be local phenomena, and to a certain extent the negative side of the morbid picture first described by O. Simon (1880) and later reported by Neisser (1883) as *leucoderma syphiliticum.*

This dyschromasia, which, strange to say, has been so long overlooked, but which has recently been often described, appears in a considerable number of recent syphilitic patients, especially females, at the time of the involution of a maculo-papular syphilide, in the shape of discs ranging in size from a lentil to a penny, of a clear white color, bordered by normal or more commonly darkly pigmented skin. They are most numerous and crowded together in the cervical and nuchal regions, and discrete on the trunk and extremities. Tortora illustrates a case of "sifilide pigmentaria areolata" in a female patient, in whom the white spots cover the neck, trunk, and extremities equally closely, while the interstitial, more darkly colored bridges represent a continuous network. Similar cases have been seen by us and others.

In all these cases, however, the white spot, the loss of pigment, constitutes the essential and primary feature of the affection; while the darker color, when present, is secondary. Leucoderma syphiliticum, therefore, does not belong among the pigment hypertrophies, but to the pigment atrophies.

The decoloration starts from punctiform spots and always advances centripetally until it reaches the size of a penny, while the brown bridges become correspondingly narrower until they disappear when the white fields coalesce. As Riehl has correctly described it, the pigment atrophy originates directly from a roseola spot or a papule, at first forming usually a light ring about the latter, while the papular portion still appears darkly colored.

The white discs, or the larger spots resulting from the confluence of the latter, which are notched with pigmented cogs and streaks, persist for many months. Then their borders gradually become indistinct and faded, and the spots finally become unrecognizable, partly through the bleaching of the darker tinge at their borders, partly through the reappearance of the normal pigmentation within them.

According to Neisser's attempted explanation, the decoloration is

said to result from the rapid exfoliation of the pigmented rete cells in consequence of an inflammatory process; but in my opinion Riehl's interpretation is more plausible. According to the latter there is associated with the resorptive process to which the product of the syphilitic efflorescence is subject an absorption of the pigment situated in the deep rete cells, and a displacement occurs toward the peripheral zones of the various efflorescences. The active agents in this displacement are the migratory cells which have been mentioned above, after Ehrmann and Riehl, as the supposed intermediary carriers of the pigment

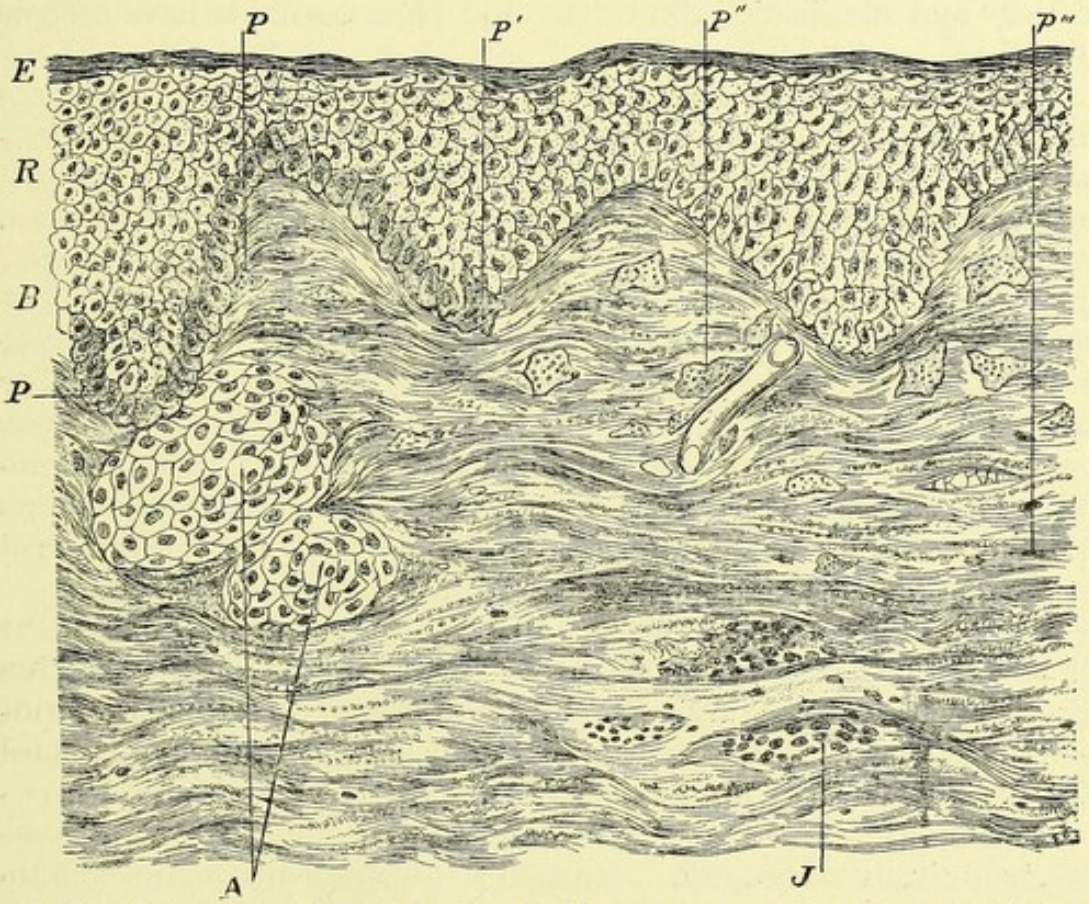

FIG. 34.—SECTION OF A PORTION OF SKIN AT THE BORDER BETWEEN A DECOLORED AND DARKLY PIGMENTED LEUCODERMA SPOT (RIEHL).

E, epidermis; R, prickle-cell layer; B, basal-cell layer; P, pigmented to P', thence to P''' non-pigmented; P', pigment-bearing migratory cell, insinuating itself with its processes between the rete cells. Numerous pigmented migratory cells, P'', in the corium; at P''' a heap of pigment granules corresponding to a migratory cell. A, transverse, J, longitudinal section of a blood vessel surrounded by cellular infiltration.

between the blood vessels and rete. In leucoderma syphiliticum Riehl found such pigment-bearing cells in the subpapillary corium, beneath those portions which showed a non-pigmented rete, and he believes that these migratory cells here carry off the pigment by the same route by which they supply it in the physiological process (Fig. 34).

Therefore we must interpret leucoderma syphiliticum, not in the

sense given by the authors first mentioned, as a true specific product of syphilis, but as an accidental sequel of the local infiltration caused by syphilis (roseola, papule), and, even with this limitation, not as a hyperpigmentosis, but as an achromatosis, analogous to vitiligo, to be discussed hereafter. Neither can this phenomenon be looked upon as a characteristic and diagnostic landmark of syphilis, but at most as an occurrence to be noticed in connection with other noteworthy symptoms of syphilis.

Alone, leucoderma has merely the importance of analogous decolorations, which start also, as a rule, at points where other inflammatory and neoplastic infiltrations and pigment warts have become absorbed.

The *diagnosis* of chloasma and of pigment spots in general is not difficult. The appearance of the color, its persistence under the pressure of the finger, its deep location, the impossibility of detaching it by superficial scratching, and the absence of other tissue alterations, such as redness or desquamation, prevent mistaking them for similar manifestations—*e.g.*, the brown spots of pityriasis versicolor. The special diagnosis of the pigmentation with reference to its importance and cause, and its prognosis, whether permanent or transient, may be deduced from the symptomatology.

Altogether different from the true pigmentoses are those dyschromasiæ of the skin which arise from the deposition of coloring matters in the cutis proper (not in the epidermis), partly such as are formed in the body, partly those introduced from without. These are :

The *icteric* coloration of the skin. It is based on the deposition of bile pigment in all layers of the cutis, and appears as a lemon- or grayish-yellow (icterus niger), diffuse staining. The duration, intensity, and curability of the condition depend upon the fundamental disease. The accompanying itching is often very great and hard to overcome.

Argyria, a slaty-gray, bronze-like (whence the term "teinte broncée") or bluish-gray discoloration of the skin and mucous membranes, due to the deposition of granules of silver in the cutis, which has been observed in persons who have taken silver nitrate by the mouth for a considerable time—*e.g.*, for epilepsy or dysentery. Since Zöllner (1795) published his observations on the disease quite a large number of similar cases have been reported. Argyria has also occurred a few times after cauterizing the pharynx with this drug, and local silver staining on the conjunctiva after applications of the caustic stick. Withal it is remarkable that argyria has never been observed, not even locally, after the countless cauterizations of the skin with silver nitrate which have been made, for instance, in lupus. It has not yet been proved from which chemical combination (soluble albuminate of silver ?) the granules of silver are precipitated after its

absorption. The influence of light is certainly out of the question with reference to the reduction, as the deposit occurs in internal organs likewise. Careful anatomical examinations (by Frommann, Riemer, Neumann, and others) have shown that the granules of silver are deposited, not in the epidermis, but in the connective tissue of the skin, most densely in the limiting layers adjoining the rete and the cells lining the glands. In the internal organs, too, it is always the connective tissue which encloses the precipitated silver (Weichselbaum). The condition is permanent and incurable.

Differing from this form in its origin is the "local argyria of occupations" (Lewin), reported almost at the same time by Blaschko and Lewin, in which "the occurrence of metallic silver in the skin of workmen in silver" (Blaschko) is due to the forcing-in of particles of silver which are detached in the mechanical processes to which the metal is subjected (filing, turning, polishing), and which, by reason of their momentum, apparently penetrate the epidermis and remain in the corium. They give rise to bluish-black points and spots, situated chiefly on the dorsal surface of the hands and fingers, more rarely the skin of the face and the chest when exposed during the work. In their histological examination both authors found metallic silver in the form of granules and in lines joined in a network, whose position and configuration correspond to the elastic fibres so beautifully represented by Baltzer, Unna, and Lustgarten by means of Victoria blue. The picture is like that obtained in microscopic sections stained with silver after Recklinghausen's method. Lewin thinks, moreover, that the network of elastic fibres and bands thus marked corresponds at the same time to the walls of a network of lymph channels. In this form, too, we must assume a reduction analogous to that in argyria caused by ingestion; the silver being first changed into a soluble salt, from which it is reduced and deposited.

By the introduction of finely divided colored or dark bodies into the corium, various colorations of the skin are produced which likewise have nothing in common with the pigment. The best known of these is:

Tattooing, which is done here by laboring men and sailors on the arms, by some of the South Sea islanders and Burmans on extensive regions of the body and in strange figures and lines, as in the case of the tattooed man from Burmah described by me and illustrated in Hebra's Atlas. The procedure is as follows: A single needle or a bundle of them, or an instrument resembling a drawing-pen, is pushed into the skin so as to draw blood, in lines corresponding to the figure designed; immediately afterward colored substances are rubbed in—charcoal powder, gunpowder (blue), cinnabar (red, used by surgeons after plastic operations), or vegetable dyes such as indigo. These

remain in the cutis tissue, where they become encapsulated. The gray color from powder burns results in a similar manner.

The *treatment* of spontaneously receding pigmentations is unnecessary, but it is often called for in persisting pigmentations, especially ephelides and chloasma uterinum.

Treatment here aims to remove the deepest layers of the rete, in which the excess of pigment is deposited. Oleum sinapis, cantharides, mezereum, sulphuric acid are not suitable for this purpose, because, as has been stated, pigment hypertrophy is particularly apt to occur in the newly formed rete after their influence. The following are to be recommended : hydrochloric and acetic acids, borax, potassium, sodium (soaps), tincture of iodine, sulphur pastes, and particularly sublimate. Where a rapid effect is desired—as, for instance, in numerous freckles or chloasma uterinum of the face—the latter is uniformly covered with pieces of linen fitting close together, the patient assumes the horizontal position, and the cloths are dampened with a sublimate solution (hydrarg. chlor. cor. 0.50, aq. dest. or alcohol 50.0) and thus kept moist for four hours. Great burning and tension result, and the epidermis is raised in a bulla, which is pierced at its lower edge so as to collapse. Under a powder dressing the epidermis crust falls within a week, and the newly formed integument is white and unpigmented. The same measure may be effective for nævus and lentigo. A similar result is produced by tincture of iodine and iodized glycerin, sulphur pastes in the course of from six to twelve applications, or soft soap spread upon flannel and applied for twelve to twenty-four hours, after which measures the entire epidermis separates and is detached with the contained pigment, or else it is discolored as in the application of chrysarobin (Leloir).

The pigments disappear slowly, accompanied by reddening and desquamation of the epidermis, after daily repeated washing with spiritus sap. kalinus, painting with dilute acetic or hydrochloric acid, or other slightly irritating substances. For instance : emuls. amygd. 100.0, tinct. benzoini 5.0, hydrarg. chlor. cor. 0.05 ; or, veratrin 0.1, aq. naphæ 50.0 ; or, naphthol (spir. sap. kal. 50.0, naphthol 2.0, glycerin. 1.0) ; or, aquæ cosmeticæ orientalis (aq. dest. 6 litres, hydrarg. chlor. cor. 35.0, album. ovor. no. 24, succi citri fruct. no. 8, sacch. alb., 300.0) 5 ad 100 aq. fragor. ; or, an ointment which is spread upon linen and applied over night, prepared according to the following formula : hydrarg. ammoniat., borac. venet. āā 5.0, ung. emoll. 50.0, ol. rosar., ol. naphæ āā gtt. 5; or, acid. salicyl. 2.0, ung. emoll. 40.0 ; or, acid. borac., ceræ alb. āā 5.0, paraffini 10.0, ol. amygd., 30.0. Ointments of pyrogallic acid and chrysarobin also destroy pigment, but their own staining power prevents their employment on the face. Should the skin become red and scaly, cos-

metic ointments and face powders are applied. Some of these have been mentioned before, but I add a few here, with the warning to avoid a combination of mixtures containing sulphur, lead, and mercury. White face powder: bismuth. carb. basici 10.0, talc. venet. pulv. 20.0, baryt. sulph. præcipit. 30 0, ol. rosar., gtt. 2. Liquid cosmetic: bismuth. carb. basici 10.0, talc. venet. pulv. 20.0, aq. rosar. 70.0, spirit. colon. 30.0; of this, as of Hebra's *eau de princesse*, the white sediment is painted on, and wiped off when dry. Cosmetic ointment: bismuth. chlor. præcip. 5.0, baryt. sulph. præcip. 10.0, ceræ alb. 3.0, ol. amygd. rec. grm. 7.

Nævi pigmentosi can also be removed by scraping with a sharp spoon, as well as by electrolytic, punctiform, or superficial cauterization, for which well-known procedure special methods have been devised by Voltolini, Gärtner, and Lustgarten. The spots named, freckles and lentigo, usually return again; only chloasma disappears permanently whenever its cause (sexual affection) is removed. Tattooing of pigment moles is useless, since no color has yet been found that corresponds with the tint of the skin. Sherwell claims to have been successful with dipping the tattooing needles into twenty-five-per-cent chromic acid or fifty-per-cent carbolic acid. In nævus pilosus the hairs must also be epilated or destroyed by electropuncture. Nævus verrucosus et lipomatodes can be removed only by thorough cauterization and excision.

LECTURE XXXII.

HYPERTROPHY OF THE EPIDERMIS AND PAPILLÆ.

STRICTLY speaking, hypertrophy of the epidermis cannot be described separately from that of the papillæ, since both, as a rule, exist in combination.

While the physiological process of the regeneration of the epithelium is far from being elucidated in every particular, this much is certain, that the material for the construction of new epidermis cells and for the nutrition of the entire cell layer is furnished by the papillæ or its vessels. In pathological processes the epithelial layer doubtless multiplies from its own elements by division of the cells and nuclei of the prickle-cell layer (Fig. 6).

As regards the internal process in the division of the cells and nuclei, especially of the epithelium, investigators were satisfied until recently with a simple explanation suggested by the common observation of nuclei constricted until there remained only a filiform connection, and of double nuclei. From the division of the nucleus thus effected investigators partly argued an analogous division of the cell protoplasm, with a daughter nucleus in each half of the cell thus produced; partly they proved it objectively by the observation of germination and constricting furrows, as was first done by Stricker.

Since the time of the publications of Frommann (1865 and 1867) and Heitzmann (1873), however, our knowledge of the internal structure of the cell and nucleus, as compared with that dating from M. Schultze, has increased and altered. A like change has come over the former idea of a simple mechanical bipartition of the cell during proliferation, thanks to the work done under improved optic apparatus and staining methods and perfected modes of examination by a large number of investigators, such as Strassburger, Schleicher, Klein, Arnold, Stricker, Retzius, Pfitzner, Spina, and others, but especially by Flemming and Rabl.

According to them the cell protoplasm consists, not of a homogeneous, finely granular mass (M. Schultze), but of two substances differing chemically, structurally, and biologically—namely, the filamentous mass (miton) and the interfilamentous mass (paramiton); and the nucleus, which must certainly be kept apart from the cell

substance, consists of a filiform nuclear framework, nucleoli, and nuclear fluid (Flemming). Besides, they differentiate chromatin, the substance of the cell nucleus which takes the stain but does not coincide with the filamentous material, and the unstained achromatic substance which represents the envelope completely surrounding the nucleus. The chromatic filament framework likewise extends to the cell border, though it forms there an envelope with numerous open meshes.

As the investigations into these most interesting details are far from having reached a final conclusion, I shall describe the present state of our knowledge of cell division in the words of C. Rabl, taken from his exhaustive paper on this subject.

Rabl states: "The indirect or 'karyokinetic' (Schleicher) cell division is associated with a metamorphosis of the cell nucleus. This consists in the formation of a figure composed of filaments, the figure of nuclear division, or nuclear figure. The figure of nuclear division is composed of the achromatic figure or nuclear spindle and of the chromatic figure. The achromatic figure is built up of those substances of the nucleus (Flemming), or perhaps of the cell body (Strassburger), which do not take the specific nuclear stains, and represents a bundle of filaments, mostly spindle-shaped though sometimes cylindrical, which join the two poles of division of the cell. From the ends of the spindle, rays extend into the cell substance. The chromatic figure is built up of the nuclear substances which take the stain, the nucleoli, and the framework filaments, and during the division undergoes a regular series of transformations. First the entire chromatic substance arranges itself into a filament, which passes through the nucleus in dense irregular coils; as the filament gradually shortens and thickens, the coils become less numerous and the convolution as a whole becomes looser. Then the filament divides into separate sections or segments, which probably split early longitudinally into equal halves (Flemming). All these various formations are comprised under the general term of convoluted form of the mother nucleus. . . .

"The further development proceeds thus: the segments of the filament contract toward the equator of the nucleus and arrange themselves around the centre of the achromatic bundle of filaments. During this step they assume the form of fillets arranged with their loops toward the centre—*i.e.*, the middle point of the axis of division—the free ends of the fillets directed outward. In this way the figure becomes star-shaped. Flemming therefore calls this stage the stellar form of the mother nucleus. . . .

"From the stellar form, according to Flemming, the chromatic figure changes into the stage of rearrangement or equatorial plate; this is effected by the sister halves of each fillet, which have resulted

from the longitudinal division, separating, one migrating to one, the other to the opposite pole (further details are given by Heuser). . . .

"The two halves of the equatorial plate now separate by advancing toward the poles. . . . Then the polar end of each filament bends in the shape of a hook, while the former curve straightens. In this way fillets are again formed, whose limbs are at first of unequal length and whose loops again turn toward the poles of the nuclear spindle. The two halves of the chromatic figure thereby again assume a stellar form, for which reason Flemming calls this stage that of the daughter stars or the stellar form of the daughter nuclei.

"Then there are formed, by the partial combination of the fillets of the daughter stars, the daughter coils or convoluted form of the daughter nuclei. This form then again changes into the framework of the resting nucleus."

Therefore every daughter nucleus in its development repeats in an inverse order the stages of the mother nucleus.

Flemming furnishes the following scheme of the main phases of the nuclear division :

Mother nucleus (framework, rest).	Daughter nucleus (framework, rest).
1. Convoluted form (spirem).	5. Convoluted form (dispirem).
2. Stellar form (aster).	4. Stellar form (diaster).
3. Phase of rearrangement (metakinesis).	

From his own studies, which apply chiefly to epithelial and glandular cells, we may select Rabl's conviction as of special importance —namely, that, with reference to the number of the primary fillets and those resulting from the division, there exists for each kind of cell a definite law of numbers, which is twenty-four for the epidermis cells of the floor of the mouth of the larva of the salamander ; furthermore, that in the embryonal cells the number of fillets is larger than in completed forms ; that Rabl agrees with Strassburger in applying the term cell to the cell substance together with the nucleus, to which latter the functions of nutrition and multiplication are entrusted ; that each cell possesses a nucleus, or at least has had one in its early age, and has always resulted from a mononuclear cell (by the above-described mode of division) ; that the filamentous mass is connected with neighboring cells by filamentous processes ; and that, finally, Rabl is not willing to give up the idea of "protoplasm," despite the distinction which is now necessary between the constituents of the cell and nucleus here discussed.

The following illustrations of the figures of cell division, taken from Rabl's paper, refer to the epithelium of the floor of the mouth and of the branchial layers of Salamandra maculata. Rabl acknowledges his indebtedness to Flemming for their mode of preparation.

We can observe the above-described karyokinetic figures in all

the more active forms of proliferation and regeneration of the epidermis, whether due to pathological processes or experimental irritations (Giovanini), and also in the cells of proliferating neoplasms, as, for instance, sarcoma.

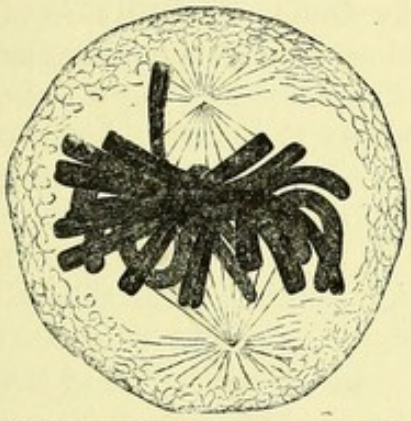

FIG. 35.—TERMINAL STAGE OF THE MOTHER STAR WITH DISTINCT LONGITUDINAL DIVISION OF THE FILLETS.

FIG. 36.—FIRST STAGE OF THE DAUGHTER STARS.

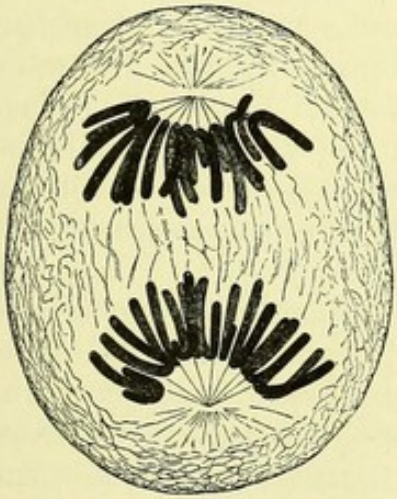

FIG. 37.—SECOND STAGE OF THE DAUGHTER STARS.

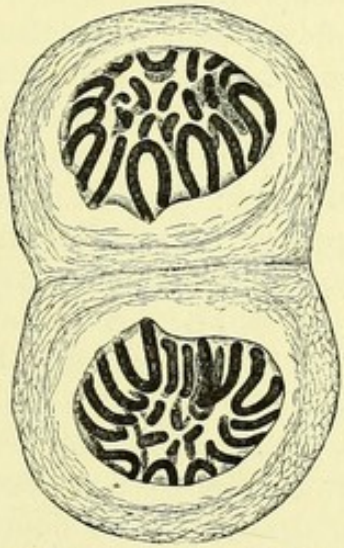

FIG. 38.—DAUGHTER COIL AFTER COMPLETED DIVISION OF THE CELL BODY.

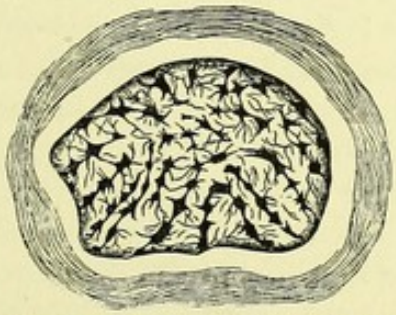

FIG. 39.—DAUGHTER COIL IN TRANSITION TO THE RESTING STAGE.

The papillæ participate in the proliferative process of the rete, not only by a more ample supply of plasma, but also by a contribution of round and spindle cells (migratory bodies), which reach the

mucous layer from the papillæ (Biesiadecki, Pagenstecher), as may be seen in Fig. 21. This condition of increased nutrient supply leads at the same time to a hyperplasia of the papillæ, and therefore an enlarged and histologically changed papillary body invariably corresponds to a hypertrophic rete. It is true, under certain conditions, the hypertrophy of the epidermis predominates markedly—namely, when a process of cornification is developed early, whereby the epidermal layer proper acquires great thickness.

If we follow Lebert in grouping together the similar forms characterized by an excessively thick epidermal layer, we may distinguish those without and those with (pronounced) papillary hypertrophy.

KERATOSES WITHOUT PAPILLARY HYPERTROPHY—PURE KERATOSES.

Callus (callositas, tyloma, tylosis) is the term applied to circumscribed, flat thickenings of the epidermis, of a dirty-white to yellowish-brown color and horny appearance, which are firm, dry, tough or brittle, and on which the normal lines and furrows of the skin are partly obliterated, while the tactile sense is blunted.

When detached from the skin tyloma represents a translucent, yellowish-white scale which may be slightly concave below or be convex on both the upper and lower surfaces. In sections it appears homogeneous ; it is thickest in the centre (two to five millimetres) and tapers off toward the periphery. It consists of horny cells superimposed parallel to the surface of the skin ; in the deeper layers the nuclei can still be recognized.

The extent, form, and location of the callosities correspond to their special *cause*. This in most cases is an external one—artificial tyloma—either oft-repeated pressure against a point subject to counter-pressure from a bony prominence, or else the frequent action of lye, mineral acids, or chemically irritating substances in general. Callus from pressure is found chiefly on the sole of the foot and the heel, caused by coarse shoes ; also under trusses, corsets, belts, and most commonly on the palm of the hand from certain occupations. At this point they correspond in their extent and location to the tool usually handled, and hence are met with in carpenters, from the use of the plane, at the fold between thumb and index finger ; in shoemakers, on the palm and the bends of the finger joints, where they are often fissured by the thread, on the right thigh from beating leather, over the tuber ischii from the pressure of the wooden stool ; in hatters, on the ball of the thumb and over the whole palm, due to rolling felt ; in tailors, on the right palm from handling the goose, and tylosities pierced with holes on the tip of the left index finger ; in musicians, on the finger tips of the left hand from the pressure against the strings. Callus due to lye is found in the palm of house

servants; due to the effects of mineral acids, in metal workers, gilders, etc. Hence it is not at all difficult to tell from the location of the callus the occupation of the person.

While tylosities have the advantage that they protect the underlying skin from injury by the pressing object or tool, yet they reduce the local tactile sensibility to a minimum, so that, for instance, fingers with callus become useless for fine feeling and handling. Moreover, when they occupy the whole palm, extension of the fingers is rendered impossible. Finally, they are troublesome by painful fissures, which often extend deep into the corium. Portions of the skin covered with callus are permanently hyperæmic and liable to become inflamed; at such points the eruptions of the exanthemata, variola, psoriasis, and scabies are more intense.

When the exciting cause is removed for some time the callus diminishes and disappears. Thus the amount of activity of the laborer can be deduced from his hand. At times, too, there appears under the callus an inflammation with suppuration of the corium, which is very painful owing to the tension of the integument, by which the callus is detached. Under such circumstances it is advisable to make an early incision, so as to prevent the danger of lymphangitis, erysipelas, and deep tissue necrosis.

A spontaneous development of tylosis can also be at times observed—for instance, on the glans penis; or in the palm of the hand and the dorsum of the fingers of persons who handle no tools, as clerks and ladies. Such keratoses of the palm I have seen developing within a few months, spreading, and spontaneously disappearing again after three or four years; but more frequently they persisted permanently.

The *diagnosis* of tylosis is not difficult when the callus is smooth throughout, tapers at the margins, and betrays its external cause by its form and location. When fissured and sharply limited, callus of the palm and sole is not so easily differentiated from eczema, psoriasis, scabies crustosa, lichen ruber, ichthyosis of these parts, and syphilis of the palm and sole. Under such conditions it is necessary not only to call to mind all the local characters peculiar to the processes named, or to exclude them, but also to inspect the rest of the skin for more marked symptoms of such diseases.

Corn (*clavus*) is a thickening of the horny layer analogous to callus, which does not lie flat upon the rete, but appears wedged into the skin by means of a central conical projection of its lower surface. The corn with its cone consists altogether of superimposed horny cells, between which the remains of hæmorrhages are sometimes found.

Due mostly to pressure from the shoes, corns appear over the joints and lateral surfaces of the toes and other projecting bones of

the foot. Pressure from without forces the cone of the corn against the skin, thus giving rise to violent pain, as is well known. Under the projecting cone the cutis with its papillæ becomes in time atrophic, even the meshes of the corium may be forced apart and be pierced by the corn, while the adjoining skin with its papillæ shows inflammatory infiltration and hypertrophy (Rokitansky).

There may be developed spontaneously, sometimes in the course of hyperidrosis, on the palm and sole, single or numerous corns, as we have occasionally observed, and, their limits being in contact, form an extensive callus. They interfere greatly with movement; stinging, burning pains radiate from the foot up above the knee and often lead to the erroneous diagnosis of gout, though a close inspection will show the presence of tylosis glabra or verrucosa.

The *treatment* of callus and corns consists in their softening and enucleation. The former is effected by warm baths, local fomentations, cataplasms, covering with impermeable dressings, rubber cloth, traumaticin (solution of rubber in chloroform), applications of green soap, cauterization with potash solution (1 : 2), acetic acid, citric acid, emplastrum domesticum (lithargyri adustum), mercurial plaster, emplastrum saponatum salicylicum (ten to twenty per cent), simple or salicylated rubber plaster. Enucleation is performed with knife and scissors, after which possibly bleeding vessels of the hypertrophic papillæ may be cauterized. Protective rings of leather, rubber, or wadding have merely a prophylactic value.

Cornu cutaneum represents an outgrowth of the skin which in form, color, and consistence bears close resemblance to the horn of an animal. Such formations have been observed in variable form and size : cylindrical, conical, pointed, provided with a broad extremity, laterally compressed, corrugated longitudinally and transversely, sharply angular, measuring from a few millimetres up to twenty-five centimetres, hook-shaped or curved like a ram's horn. They are attached by a broad base to the surface of the skin or are embedded in a depression of the skin with sharp margins. They may be single or multiple, sometimes quite numerous, as in Baetge's case, on one individual—on the scalp, the eyelid, the auricles, the tip of the nose, the lip, glans penis (Hebra, Pick), on the trunk and extremities, flexor and extensor surfaces. They sometimes develop within a very short time and persist for many years ; occasionally they fall off and recur in the same place. Now and then a cornu cutaneum changes into epithelial cancer. Some older observers (G. Simon) claim to have found in cornu cutaneum a cortical and medullary substance and a peculiar tubular structure composed of vessels (Virchow). The fact is that from the cutis a group of hypertrophic papillæ with dilated vessels projects, sometimes slightly, sometimes high up into the substance of the horn ; the latter, however, consists

of columns of epidermis cemented together longitudinally, each erected on the several papillary groups. The appearance of transverse sections varies according as they include papillæ in the lower sections or higher up where papillæ are absent. The several columns often show a concentric arrangement of the epidermis cells resembling cancroid corpuscles, or a cellular structure from the desiccation of the elements. When the horn is detached its base often shows depressions fitting the hypertrophic groups of papillæ. It is certain that cornu cutaneum develops upon pre-existing hyperplastic papillæ, as in Pick's case upon condylomata. Even when the horn is seated in a depression, as in a dilated follicle, or demonstrably in an atheromatous cavity, its basis is formed by papillary outgrowths (Rindfleisch), though in that case the epidermal investment of the glands and hair follicles contributes to the agglomeration of epidermis. Thus on the abdominal wall of a young man I have seen and removed numerous similar outgrowths which had formed in a few weeks from an atheroma.

Cutaneous horns, therefore, are essentially accumulated or agglutinated warts, and the only thing strange about them is their appearance, not their origin. Lebert, Hessberg, Bergh, Wilson, and Lozes have treated the subject in detail.

Cornu cutaneum is removed by simple detachment and cauterization of the papillary basis. Where an atheromatous wall is present this must likewise be cauterized, enucleated, or scraped, or simply expressed.

Cornu cutaneum forms a distinct transition to warts or

KERATOSES WITH PAPILLARY HYPERTROPHY.

WART (verruca) is the term applied to every roundish, uneven, nodular outgrowth of the skin which corresponds to the popular meaning of the word. Many warts are congenital (verruca congenita), but usually they do not appear until the later months of life. This variety is generally darkly pigmented and covered with hair —*i.e.*, it is a nævus verrucosus et pigmentosus. It varies in shape, size, and location; often it follows the course of the nerves (papilloma neuroticum), extends unilaterally over some regions of the body, or, as in a case described by me, over the whole body, always in the course of the nerves. I have above (page 414) given my views on the latter point, that the nerves have nothing to do with the abnormal formation. The warts either persist through life or disappear gradually. Most warts develop during later years (verruca acquisita) and are either permanent (verruca perstans) or temporary (verruca caduca). The most frequent form is that of the common warts (verrucæ vulgares). They range in size from a pin head to a pea, may represent a flat elevation (verruca plana) or be hemispherical. They

are firm, slightly sensitive, yellowish-white prominences of the skin, with smooth (verruca glabra), nodular (acrothymion), fissured, or fasciculated surface. They may appear slowly or rapidly, acutely or subacutely, singly or in large numbers, on the hands, feet, auricle, in the face or on the hairy scalp, usually in young persons, may persist for months and years, and vanish spontaneously. Some remain for many years or during life. The cause of their origin is unknown. The popular belief in their infectiousness is quite unfounded.

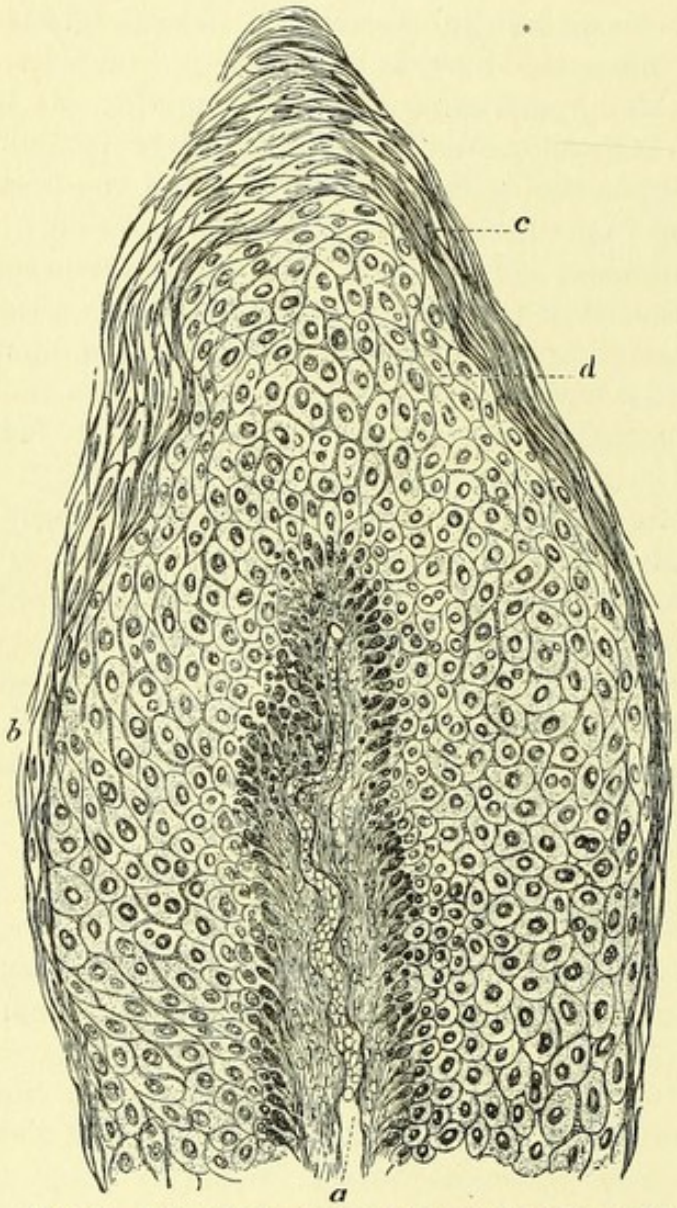

FIG. 40.—VERTICAL SECTION OF A VILLUS OF CONDYLOMA ACUMINATUM. (HIGH POWER.)

a, papilla with vascular loop ; *c*, horny layer of the epidermis ; *d*, rete layer with many proliferating binuclear prickle cells, mingled at the height of *b* with round cells, which may be derived from the cell infiltration of the papillæ (migratory cells).

Verruca senilis is a flat, micronodular, dirty-brown deposit on the trunk, face, and arms of old people, varying in size from a lentil to a penny. It can easily be scraped off with the finger nail, and has for its base a bleeding, moderately hypertrophied papillary body. Epithelioma frequently develops from these warts.

Condylomata acuminata are filiform, fissured, or blackberry-like nodular, multiple outgrowths, situated on an otherwise normal, non-infiltrated skin. They owe their origin to the specific irritation of the latter by gonorrhœal secretion. They are soft, succulent, bright red and weeping when located on mucous membranes or in places where their surface is subject to maceration, as at the introitus vaginæ and on the inner layer of the prepuce ; or dry and hard when their covering epidermis can undergo cornification. They proliferate most luxuriantly on the coronal sulcus, glans,

and prepuce ; in women on the introitus vaginæ, the external genital surfaces, the mucosa of the vagina and intravaginal cervix, the perineum, and the rectal mucous membrane as far as the internal sphincter.

Although the acuminated condylomata are due to the irritation of the skin and mucous membrane by blennorrhœal secretion, and by contact stimulate neighboring portions of the skin to a like proliferation, yet their direct transmission (apart from blennorrhœa) to other persons has thus far failed ; for Kranz's experiments with direct contagion have scarcely succeeded, and Zeissl's report of contagion by coition does not exclude simultaneous transmission of blennorrhœa.

The *anatomical* conditions in all these forms of warts are essentially the same : simple or dendritic outgrowths of vascular loops which fill the greater portion of the correspondingly enlarged and formed papillæ, and upon the latter an enormously developed and proliferating rete mucosum (Fig. 40). Wherever the rete is very thick and the process of proliferation very active and rapid, as in non-cornified acuminated condylomata, karyokinetic figures of division of the cell nuclei can be seen very plainly with moderate powers (Hartnack No. 8). Upon the thick rete we find in addition, in dry warts, a considerable layer of horny cells. In the papillæ and the adjoining corium we find, corresponding to the activity of the vegetation, often a marked cellular infiltration, which, when long continued, leads to the formation of fibrous tissue. For this reason the base of old condylomata usually appears as a cicatrix of firm connective tissue.

The structure differs in those warts which may be termed *verrucæ filiformes, pendulæ*—filiform or pediculated, club-shaped, soft, smooth appendages with normal epidermis, which appear and persist on the delicate skin of the neck, eyelid, and the female breast, often in large numbers. These verrucæ mollusciformes consist of an outgrowth of connective tissue protruding from the depth of the skin and provided with a vessel in the pedicle. Hence this variety represents a small fibroma molluscum.

The importance attaching to molluscum verrucosum (s. contagiosum, condyloma subcutaneum, etc.) has been explained above (page 136 *et seq.*).

Warts require operative removal by the curette, scissors, ligature, cauterization with sesquichloride of iron, fuming nitric acid. acetic acid, sulphur paste, Plenck's solution (hydrarg. chlor. cor., aluminis, cerussæ ; camphoræ, alcoholis, aceti vini, āā 5.00), or of plaster of mercury and arsenic (Unna). Acuminated condylomata of the mucous membrane can also be dried up by basic acetate of lead, pulv. frond. sabinæ, burnt alum, resorcin paste (resorcin 10–30, glycerini, vaselini āā 25.0), etc.

LECTURE XXXIII.

Owing to its special group of symptoms, the disease known as

ICHTHYOSIS

occupies a peculiar position among the keratoses.

Ichthyosis is an affection developing in earliest childhood and usually persisting through life. It is characterized by a skin which is rough and generally dry, and appears covered with thin scales and laminæ or thick plates of epidermis or horny warts.

The low grade of the disease—*ichthyosis simplex*—shows a typical character and various degrees of intensity. The former manifests itself particularly in the peculiar localization, which exactly corresponds to that described for prurigo—that is to say, the extensor surfaces of the extremities are mainly affected with the disease in an intensity increasing from the arm to the leg, while the skin of the popliteal region, the groin, and the axilla is normal in quality, suppleness, and perspiration.

In the mildest grade the extensor surfaces of the arms and thighs are set with pinhead-sized, pale-red nodules crowned in the centre with a small mass of scales. When this is scraped away a coiled-up hair appears. These nodules give the skin a rough, uneven appearance and sensation to the touch, and represent the affection known as *lichen pilaris*. This condition, however, in a slight degree, is found on the outer side of the arm and thigh of every man, especially about the time of puberty when the lanugo hairs begin to sprout more energetically. But in ichthyosis, lichen pilaris is constantly present from childhood on, and occupies often not only the extremities but the entire trunk, so as to present the appearance of a permanent cutis anserina. The condition observed by T. Fox in one case, named and figured by him as "cacotrophia folliculorum," seems to agree with the latter. I have seen a few instances in children, five to ten years of age, of such a lichen pilaris of the capillitium, which had existed from birth and resulted in a sparse growth of thin, brittle lanugo hairs.

Of more frequent occurrence is that form in which the surface of the skin of the extremities is covered with dirty-white or grayish polygonal lamellæ of epidermis, ranging in size from a lentil to a

penny. These are adherent in the centre or umbilicated (ichthyosis scutelata, Schönlein), turned up at the margins, and with a glassy translucence. Aside from sharply marking the lines and furrows of the skin, they give the latter a distinct tessellated look—*ichthyosis nacrée* (Alibert), *nitida*.

A still higher degree of the process is represented by *ichthyosis serpentina*, in which the above-named surfaces with those of the abdomen and back are grayish green, dirty, as if they had not been bathed for a long time, covered with thicker epidermic scales, while dry, warty elevations are present over the knees and elbows. On all such points the skin is rough, dry, and does not perspire ; passing the hand over them makes a grating noise ; when scratched with the finger nail a white epidermic dust is detached. But a marked desquamation, such as in psoriasis, cannot be noticed in ichthyosis. The skin of the face and neck likewise appears here and there dirty gray, dry, and scaly ; the hairy scalp is branny (pityriasis), covered with thin, brittle hairs. The nails are often punctured and friable.

The palm of the hand and sole of the foot are, as a rule, spared ; still there are cases in which they are likewise or exclusively covered throughout life with thickened epidermis and horny excrescences—ichthyosis localis. Inasmuch as this has long been known, it is a matter for surprise that some authors have cited such occurrences as peculiar formations and under special names (Unna). In like manner the palm and sole are usually implicated in the highest degree of the disease.

This highest degree of the disease is called *ichthyosis hystrix*, or *hystricismus*. In this form there are present, besides the appearances in ichthyosis simplex, thick, diffuse, plate-shaped elevations, resembling nail heads, on the palms and soles, together with horny warts in great numbers and close proximity, often following a course corresponding to that of the nerves, another characteristic feature. For this reason I do not hesitate to look upon the entire process as papillomatous, the papillomata being scattered over the whole body, all the more because pigmentoses are also associated with this condition. In one patient we have observed the body to be divided from the forehead to the symphysis, from the vertex to the coccyx, by an anterior and posterior median brown pigment line, with similar streaks passing along the cutaneous nerves of the extremities ; all of these were accompanied laterally by papillary warts up to one centimetre in height. In the case figured in Hebra's Atlas the warts ran in the direction of the intercostal nerves, as in zoster. In another case. that of a girl in whom the warts were general and arranged in streaks, and in two additional cases in which the warts passed along the sciatic and crural nerves, local inflammation occurred at times,

together with profuse detachment of the epidermis, so that, not knowing the condition before the inflammation, the impression might be gained that they had just appeared acutely.

In the case of a girl of six at my clinic, there are present over the whole trunk and the extremities, especially the extensor surfaces, also on the ears, the forehead, and the hairy scalp, numerous excrescences one to two centimetres in length. These consist of a mollusciform pedicle, the size of a raven's quill, formed from the cutis with a vascular loop, and a tip resembling the plume of a feather, composed of parallel yellowish-white filaments of epidermis. The child presents the appearance of being covered with feathers, and in certain regions, as over both angles of the scapulæ, the sacrum, and the trochanters, where many such excrescences are present, small wings seem to sprout from the body. The palm and sole, however, are occupied by the usual warty and fissured flat lesions, as in ordinary ichthyosis hystrix. The case might be described as ichthyosis hystrix pterygoidea or plumiformis.

The course of ichthyosis presents very little variation in the symptoms. In ichthyosis hystrix the thick epidermic scales may fall off accidentally or under local exudative processes, as stated above ; it has even been reported that a general shedding of the scales may produce a kind of moulting, but the scales form again In one case Hebra observed such a decrustation after severe variola, which was followed by permanent recovery. The forms of ichthyosis simplex likewise produce the impression of a very slow metabolism, but the morbid picture changes occasionally by the occurrence of eczema on both diseased and otherwise healthy portions of the skin. This is caused by scratching, for ichthyosis simplex is always accompanied by rather troublesome itching.

Many observers have endeavored to solve the riddle of this disease by anatomical and chemical examinations of the ichthyotic skin and the products of its secretion, but thus far without result. Although hypertrophy of the epidermis and papillæ had been early demonstrated (Rokitansky, Bärensprung, G. Simon), still the formation of the thick crusts has at the same time been ascribed to a retarded shedding of the cornified cells. The cause of this was thought to lie in a closer adhesion of the epidermis cells in consequence of an altered glandular secretion (Büchner), or fatty degeneration (Schabel), or peculiar chemical constituents (Schlossberger, Franz Simon, Marchand).

The *anatomical* conditions in ichthyosis hystrix do not differ from those in old warts : enormously elongated papillæ, above which the horny layer is piled up in thick cones. The peculiar arrangement of the latter, like the coats of an onion, the varying color of different layers, the formation of lacunæ within them by retraction—all this is

merely the result of the long retention of the masses of epidermis. Dilated vessels and moderate cell infiltration in the papillæ and the corium, with sclerotic change in the connective tissue, complete the anatomical picture (Fig. 41); while the glands and hair follicles are normal at some points, and at others exhibit an extension of the excessive cornification to the root sheaths of the hairs. Similar findings are reported in ichthyosis nitida and serpentina. On portions of the skin from the leg, in ichthyosis characterized by thin layers of scales, I have been unable to demonstrate hypertrophy either of the

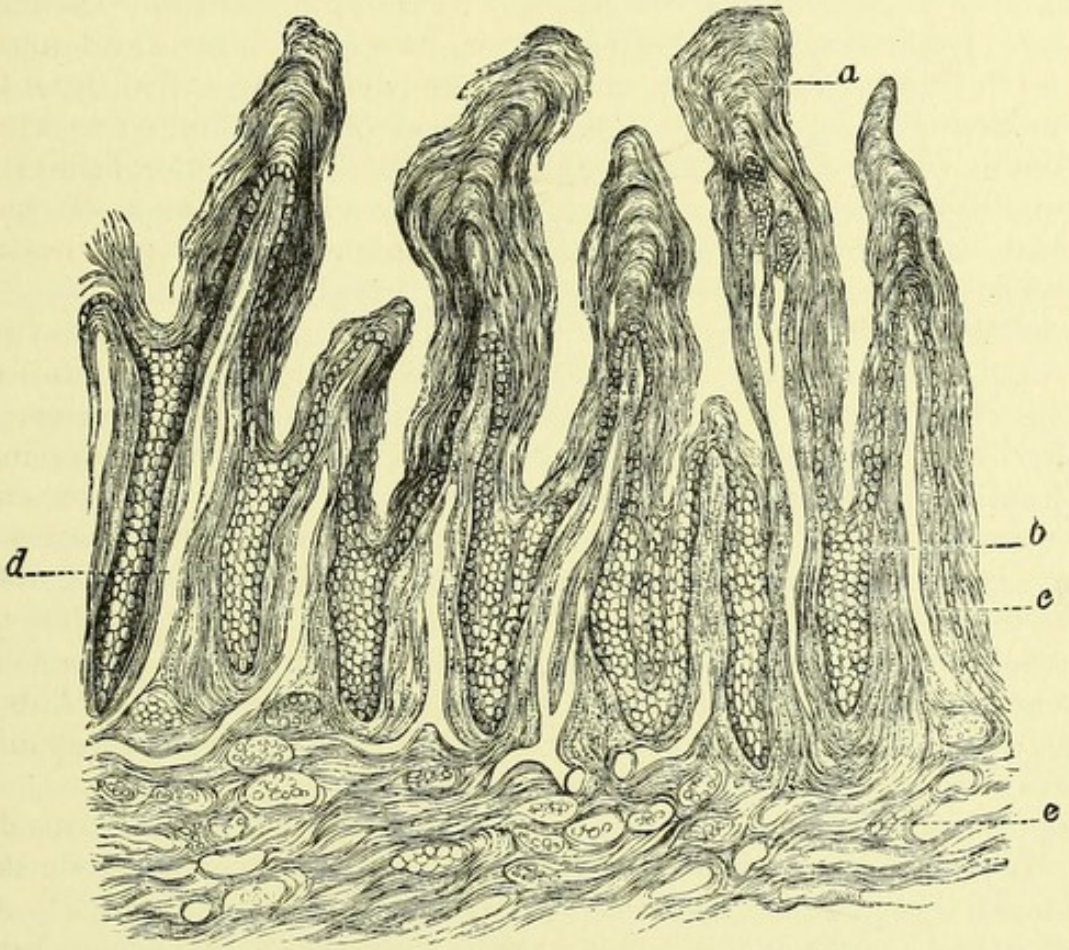

FIG. 41.—ICHTHYOSIS HYSTRIX. VERTICAL SECTION. (LOW POWER.)

a, cone of horny cells; *b*, rete cone; *c*, enlarged papillæ with dilated vessels *d* and infiltrated with cells; *e*, corium with coarse connective tissue and numerous vessels in transverse section.

papillæ or of the epidermis, but I found it present in other locations—for instance, over the knee, where there were thick scales or even warts. At the same time the panniculus adiposus is everywhere poorly developed. What is most striking both in ichthyosis simplex and in ichthyosis hystrix is the sudden transition of the rete cells into the horny layer and an excess of the cement substance between the cells, which would indicate an early cornification of the rete cells. This seems to me to be the cause, on the one hand, of the relative narrowness of the mucous layer as compared with the thicker

horny layer, and, on the other hand, the long retention of the horny cells. This condition is even more marked in ichthyosis hystrix; for while in other papillary keratoses a thick horny layer is associated with a still thicker and actively proliferating rete, as (Fig. 40) in acuminated condylomata, in ichthyosis hystrix (Fig. 41) we see an enormous horny layer over a thin, ill-nourished, slowly vegetating, almost atrophic rete.

The *cause* of ichthyosis, therefore, seems to lie in an hereditary local anomaly of growth of the cutis, especially of the epidermis and its fatty secretion. The disease may be both congenital and hereditary. Still the symptoms of ichthyosis, as a rule, do not develop before the second year of life, and very rarely only do we find them in the new-born. I have seen the condition once, as have Caspary, Tommasoli, G. Behrend, and several others. The affection formerly described as ichthyosis congenita is merely an incrustation formed of seborrhœic masses (cutis testacea) in new-born infants, is a curable and temporary condition, and is more properly termed ichthyosis sebacea (see page 125).

This statement does not exclude the fact that under the head of ichthyosis fœtalis or intrauterina are enumerated the forms combined with congenital malformation of the eyes, ears, and the regions of cutis adjoining them, occurring throughout in non-viable monsters. The first instance and prototype of these was Steinhausen's case (page 125), and analogous cases have been reported by Kyber (keratoma diffusum intrauterinum) and Hans Hebra. Caspary pleads for such a classification in a most thorough paper; for in two observations by Lang and in reports of other authors (Thost, Stühlinger) we find ground for the belief that in this respect there are many transitional forms with reference to the degree, extent, and, regarding the fœtal development, the beginning of the anomaly.

Least of all, as has been stated, should ichthyosis hystrix be classed with true ichthyosis; but even here a strict separation from seborrhœa universalis cannot always be maintained, as is shown by a case of hystricismus cum seborrhœa universali reported by De Amicis.

Heredity in ichthyosis is demonstrable in many cases. Either all the children of an ichthyotic parent suffer from the disease, or only some, of the same or the opposite sex. Thus we knew of an ichthyotic mother, all of whose five sons were affected with the disease, while the three daughters were free from it. Sometimes the disease also passes over one generation to reappear in the succeeding one or in a lateral branch. At times, however, heredity cannot be demonstrated. A certain amount of celebrity was acquired by the Lambert family (father and two sons) affected with ichthyosis hystrix, who for many years during the last century were exhibited as freaks (porcupine men) and were described and figured by Ludwig and

Tilesius. Sex, position, mode of life, lack of proper care during infancy, and other general factors do not seem to furnish any etiological cause of ichthyosis.

Aside from the idiopathic form here described, a *consecutive ichthyosis* has been reported, meaning thereby an hypertrophy of the epidermis and papillæ with pachydermia, occurring in consequence of chronic inflammations and neoplasms, especially on the legs. Esoff classed even the anatomical appearances of a region of skin thus affected without hesitation with ichthyosis. I believe that it is better for us to include these forms in elephantiasis arabum and to confine the term ichthyosis to the congenital and idiopathic, typically localized and persistent affection described above.

The milder grades of ichthyosis simplex can be ameliorated or removed by careful attention to the skin, continued for years. In the more intense form continual care will be called for by complicating eczema and temporary increase of the dryness and desquamation of the skin. Ichthyosis hystrix, of course, is incurable, and the prognosis of ichthyosis in general is not favorable. The fact that transmission to offspring is probable may have to be pointed out, particularly as an objection to marriage.

In the *treatment* of ichthyosis all those measures and procedures are suitable which cause a rapid softening and exfoliation of the epidermic scales, and have been recommended under the head of general treatment and in connection with psoriasis, prurigo, squamous eczema, and tylosis—namely, courses of inunction with soft soap, Wilkinson's ointment, cod-liver oil, and other fats; furthermore, baths, washing with soap, covering with rubber tissue, and especially, in accordance with my most recent experience, the methodical treatment with five-per-cent naphthol ointment, which is rubbed in thinly once or twice daily, while washing with naphthol soap is done every other day. When by this means the ichthyotic skin has become smooth and supple, efforts are made to keep it in this condition by diligent bathing and the inunction of bland fats, such as vaseline, lard, cold cream, glycerin, unguentum glycerini, lanolin, etc. The addition of croton oil (5 ad 100, according to Wilson), citric acid, etc., has no specific effect. In like manner all the internal remedies thus far tried, such as arsenic or tar water, have proved useless.

Lesions of greater size in severe forms of ichthyosis can be specially softened by the employment of applications of green soap, of concentrated potash solution (1 : 2) or acetic acid, the application of mercurial plaster or ten-per-cent pyrogallic acid ointment. They may also be scraped off with the dermal curette, while papillomatous excrescences require the methods discussed under the head of papilloma in general. Of course in ichthyosis hystrix interference would

be resorted to only in excrescences in especially prominent situations, since the removal of all hypertrophic formations is practically impossible.

KERATOSIS FOLLICULARIS AND PSOROSPERMOSIS.

Besides the previously described typical forms of congenital or acquired hyperkeratoses, there are certain other varieties of keratosis to be distinguished from them. There is the hyperplasia of the epidermis and thickening of the layers of horny cells, either as a simple tylosis or in combination with hyperplasia of the papillary body, manifested as a simple thickening of the superficial corium or associated with a warty outgrowth of the papillæ and implication of the epidermal investing cells of the follicles and sebaceous glands, in the shape of verrucoso-corneous formations. They may or may not be associated with hyperchromatosis of a sepia, blackish-brown, grayish-brown, or bronze-brown color. Should they occur isolated they would hardly be noted, but if numerous and extensively developed they are quite apt to excite attention. Under certain conditions they are very disfiguring, annoying in social and business relations, and not quite fully explained pathologically and etiologically.

To the same class belong the tumid thickenings of the integument which occur in the sixth decade of life in many persons whose skin is exposed to no caloric or chemical irritation. They appear on the palm, the fingers, in the temporal region, and elsewhere; spread gradually, become rough and fissured, and undoubtedly are signs of presenile age, since the epidermal hyperplasias and cornifications become at that time more prominent as compared with the re-formations and new formations of tissues of a higher grade (in the connective tissue, in the muscles, in the vascular system). To the same class belongs Besnier's *keratodermia symmetrica erythematosa* of the palms and, according to my observation, of the soles; also cases such as those described by Pollitzer and Janowsky. In these, one a woman aged sixty-three, the other a man aged forty-three, there developed on the nucha, in the axilla, the genital, crural, and anal regions, at the umbilicus, on the dorsum of the hand, and elsewhere, in conjunction with dark pigmentation of the skin, flat and warty formations thickly covered with a peculiar verrucose outgrowth, the borders smooth or serrated. Analogous excrescences appeared on the buccal mucosa, the tongue, and the palate. I have seen similar cases, and one, which was quite analogous as regards the distribution, in a man above fifty, but I fail to understand why the authors named call the condition "*akanthosis nigricans*"; for it is not the prickle-cell layer alone that takes part in the hyperplasia, but the entire anatomical portion of the skin which makes a physio-

logical whole—*i.e.*, the papillary layer, its vessels, the rete, and the pigment-forming cells. Accordingly not only the epidermis but also the superficial cutis is thickened, the furrows of the skin are deepened, the papillæ and their vessels are enlarged into warty-tumid outgrowths, and the pigment is increased above the normal. I should think, therefore, that the term "keratosis" nigricans would be more suitable, for the latter conception presupposes the possible and usual participation of the papillary layer which forms the epidermis.

In recent years a large number of dermatologists have claimed a distinct, special position for a group of dermatoses which likewise appear in the shape of excessive epidermis formation and cornification, and which are known in the literature as *Paget's disease, keratosis follicularis* (White), and *psorospermosis cutanea* or Darier's disease.

Since Paget's original paper (1874) a number of authors (Lawson, H. Morris, G. Thin, R. Monro, McCall Anderson, Sherwell, Duhring and Wile, Jamieson, Radcliff-Crocker, Lewis, Lassar, Busch, Neisser, Vidal, Wickham, and others) have described a disease occurring on and around the nipple in women. To this they applied the above name, Paget's disease, while G. Thin calls it a malignant disease of the nipple.

As affecting the nipple it is described as a disease occurring exclusively in women of advanced age. It begins with redness and swelling of the nipple and its follicles, together with those of the areola. The further course is characterized by alternate weeping, crusting, or dry desquamation and fissuring, and superficial ulceration. In some cases it terminates in the course of years in complete recovery, but usually it causes a red, glossy, rather firm condition of the upper cutis (sclerosis). The nipple at the same time retracts below the level of the affected skin, and finally it leads to cancer of the mammary gland. Accordingly Paget and other English authors, in view of this issue of the disease, discuss the question whether the extirpation of the breast would not be indicated in the early, "eczematous" stage. According to observations by Pick, Neisser, Crocker, Pospelof, and Tarnowsky, the disease occurs in an analogous manner on the penis and scrotum.

The suspicion has occurred to me that in the affection thus described we have to deal, in a certain number, merely with eczema of the nipple and areola, which indeed progresses in the manner described, and occurs also in much more excessive forms with marked sclerotic thickening of the skin, but which can be cured, as I have found in cases that were looked upon as carcinoma ripe for operation; in the remainder, however, with true carcinoma which had

resulted from the originally benign dermatitis and folliculitis or had given rise to the latter.

The investigations of Darier, however, have led to another view of Paget's disease. This author thought he could consider the disease as essentially identical with that described by him as "psorospermose folliculaire végétante," and, on the other hand, James White identified two cases reported by him as "keratosis follicularis" with those of Darier.

Including the two cases of White's "keratosis follicularis" there have been reported thus far in the literature of the dermatosis which Darier termed "psorospermose folliculaire végétante" 16 cases (Darier 2, White 2, C. Boeck 4, Lustgarten 1, Buzzi and Miethke 1, Schwimmer 1, Zeleneff 1, Krösing 1, De Amicis 1, Janowsky 1, Campana 1). Soon after the first reports of this particular form of disease by Darier and Thibault (1889) came the microscopical demonstration of peculiar large, roundish bodies, previously described by Malassez, in and between the enormously proliferated epithelial cells in this process and in Paget's disease. These were interpreted as "psorospermiæ" by Darier, who based upon them his assumption of the identity of the newly observed form of keratosis with Paget's disease. Then followed White's statement of the identity of his keratosis follicularis with Darier's disease. All these statements rested on the facts that both the clinical picture in the three forms named showed many points in common, and that in all the three the psorosperms were present. Regarding the proof of the identity of the two first-named processes, Darier's view has been materially strengthened by Wickham's thorough study of Paget's disease and by Jonathan Hutchinson Jr.'s confirmation of the occurrence of these peculiar cells in the true Paget's disease.

Darier's disease, as we shall call it in honor of the observer who first described it, following herein C. Boeck, Buzzi, Miethke, and others, may be scattered generally over the whole body. When occurring in patches its points of predilection are the hairy scalp, the forehead, the naso-labial fold, the auricles, the axillæ, the inguinal and pudendal regions, the abdomen and navel, over the sternal and clavicular regions, between the shoulders, on the dorsum of the hand in isolated efflorescences and small patches, and elsewhere on the trunk and extremities. It develops on isolated points which may be far apart, either simultaneously or successively, and spreads partly peripherally from these, partly by the appearance of fresh efflorescences at various points. The course is extremely chronic and afebrile.

According to Darier's description, the primary efflorescence consists of a pale-red nodule, from a millet seed to a pinhead in size, covered with a blackish-brown or grayish, hard, dry crust. The

little crust is very firmly adherent, and if detachment is effected it may be perceived that, like a small corn, it is embedded, by means of a conical or cylindrical, dirty-white, friable, fatty plug, in a funnel-shaped depression at the opening of a follicle.

By the formation of marginal and neighboring similar efflorescences patches result which may reach the size of a lentil, a penny, or finally occupy large surfaces of the axilla, the axillary and inguinal folds, etc. They are elevated, dirty gray and brown, rough, warty and tumid, and feel like a grater. Their base is hard, their surface is covered sometimes with dry or soft, fatty, ill-smelling epidermis, or else it is deeply fissured, and, after the epidermis is detached, weeping or crusted. Pressure forces out yellowish-white, pasty, and friable epidermal plugs, sometimes mixed with pus ; corresponding cavities or funnel-shaped depressions being left behind. In the genito-crural fold, the axilla, and on the mons veneris the patches may be elevated even into larger tumors with steep margins, whose surface may be as described, or show here and there numerous roundish and crateriform depressions, or superficial excoriation, weeping, and possibly ulceration. In Boeck's cases could be observed on the flexor side of the fingers, hands, forearm, feet, and toes, hard, warty, plate-like, and rough horny thickenings of the skin, while the nails were degenerated, opaque, thickened. Glandular swelling seems to occur only in connection with local inflammatory processes.

The course of the disease, according to the recorded observations, occupies many years (thirty-three in one of Boeck's cases) ; its cure has thus far never been witnessed either spontaneously or through medication, although pyrogallic acid (Boeck) and carbolated sublimate (Buzzi, Miethke, and Schwimmer) seemed to have a good local effect. But, on the other hand, the process in the many years of its course exerts no injurious influence on the general organism, and the patients die only of a late marasmus or a complication.

When I call to mind the case exhibited by Darier at the Paris Congress in 1889, and take into consideration the excellent descriptions published since, especially those of C. Boeck's four cases, there can be no doubt, from a clinical point of view, that this process represents essentially a keratosis and to some extent resembles lichen ruber acuminatus in those regions where the dry, horny nodules and patches are present.

Under the microscope we can demonstrate in the actively proliferating rete, partly in its lacunæ, partly within the cells near the nucleus which is crowded aside, those large round cells that Darier pronounced to be coccidiæ. These he looked upon as the cause of the great proliferation and as justifying the title of " psorospermosis " for the entire process. Many other investigators share this view, as Besnier, Lustgarten, Mansuroff, and others. Grave doubts have

been expressed by Bowen as to these formations being coccidiæ, in so far as they have been found in White's keratosis follicularis, which has been identified with Darier's disease. Boeck, Buzzi, and Miethke, however, have even directly denied it by explaining them as peculiar degenerative forms of the greatly and atypically proliferating rete. Others have also expressed doubt, and not a single naturalist has confirmed their interpretation as psorospermiæ.

Inasmuch as similar contradictions and doubts have been uttered in regard to the same bodies found in Paget's disease (G. Thin and others), the etiological and clinical identification of the two processes may be called in question. Moreover, the most recent histological examinations by Ribbert, Noeggerath, and other investigators have shown that these much-discussed bodies are the same as those described by Virchow fifty years ago in the atypical epithelial proliferations of carcinoma as of regular occurrence and as nuclear degenerations. They have proved that, according to Kiener, they are found also in inflammatory epithelial hyperplasiæ, and, according to Ehrmann, in the epithelium of pemphigus; that they represent atypical nuclear metamorphoses which, according to Hansemann, are probably due to the defective nutrition of the greatly and rapidly proliferated epithelium. Hence Noeggerath pronounces Wickham's so-called "psorospermiæ" plainly a misconception.

Again, in Paget's disease and the carcinoma following it, and in its metastases, no trace can be found of these unicellular formations (Neisser and others), and, furthermore, neither in this nor in Darier's disease has contagion been observed among married people or other persons. In White's second case of "keratosis follicularis," a girl aged twenty-one, a daughter of the man who formed his first case, was attacked in her fifth year; she had been separated from her father since her first year. Hence we might suppose here rather a hereditary origin of the keratosis, as in Boeck's three cases affecting a father and his two sons, and in Campana's case of "ittiosi cornea e psorospermosi," in which the disease was present a few months after birth as nævus papillaris cornutus, and, finally, because in some of the observed cases an ichthyotic condition of the skin in regions not affected by the disease is mentioned.

The microscopical examinations thus far made in "psorospermosis cutanea" strengthen this view, for they have shown a great and rapid proliferation of the entire rete, not only of the follicles and glands, with corresponding thickening of the horny cells and atypical transitional and degenerative forms of the cells, together with a tendency to a conical penetration of the proliferating cell layers toward the cutis (Boeck).

Certainly the whole question cannot yet be considered as decided, and the present state of our knowledge merely forces us to look upon

Darier's disease as a clinically very important and pathologically most interesting form of keratosis.

For completeness' sake we must add to the group of keratoses hypertrophy of the hair and nails.

HYPERTROPHY OF THE HAIR.

Hypertrichosis, hirsuties (polytrichia, trichauxis), appears as an increased growth of hair which is abnormal with reference to the patient's age and sex, as well as its peculiar localization. Of course this does not mean a new formation of hair, but merely an excessive growth of hair already present and physiologically preformed.

Excessive hirsuties is either congenital or develops in the course of extrauterine life—hirsuties adnata et acquisita. Many children are born with uncommonly long hair on the scalp and trunk (lanugo), which usually falls, but rarely persists. A monstrosity is presented by hirsuties universalis (dasytes), in which face and body are covered with soft blonde lanugo hairs up to several centimetres in length. They follow in their direction and arrangement, diverging outward from the median line of the face, the lines and circles given by Voigt for the position of the hair. Waldeyer thinks that such abundant lanugo has been caused by the replacement of the fallen fœtal lanugo. I maintain that it represents the persisting lanugo of the fœtal condition, developed to excess and abnormally long retained. We do not need to fall back upon old tales and myths of maternal impressions for such cases. In our clinical lecture room hang the life-size portraits of a family affected with hirsuties universalis—father, son, and daughter—from the sixteenth century. These have been described, so Dr. Bartels tells me, by Felix Plater, of Basle, in 1583, and painted by Ulysses Aldrovandi, later by Georg Hoefnagel ; the latter originals are in our Fideicommiss Library. Very similar were the two Russians (father and son) who exhibited themselves here and elsewhere in 1873 and were the subject of special publiçations. Among some races of South Sea islanders this anomaly occurs much more frequently (Miklucho-Maclay). M. Bartels has thoroughly studied the particular relations of hypertrichosis, and so has Michelson, who at the same time lays stress upon the tooth defects associated with it, also observed by others—that is, an arrest of development analogous with hypertrichosis congenita.

Hirsuties acquisita is usually confined to smaller surfaces. To this category belong the growth of hair on pigment moles (nævi pilosi), the rare monstrosity of a full male beard in women, and the frequent anomaly of the sprouting of thick, bristly hairs on the upper lip and chin of female persons, seldom in the young with normal sexual functions, and more frequently in the sterile and those beyond the climacteric. Still, the woman with a beautiful full beard

figured by Duhring, who has borne several children, may be mentioned as an exception.

Finally, we might include here an excessive development of the hair of the scalp and face in general. I have known a medium-sized young lady whose light-blonde, profuse hair reached to the ground; and I have the portrait of a miner whose beard, four feet in length, reached to his feet. The man was in the habit of folding it and putting it inside his vest.

Anatomically the excessively close, thick, and long hair of hypertrichosis does not differ from the normal.

To many forms of hypertrichosis we can assign a plausible *cause* —for instance, to hirsuties universalis, heredity, as shown by the above example; to hirsuties faciei in women, sometimes sexual disturbances, although they were certainly absent in other cases. A locally increased or altered nutrition may explain the excessive sprouting of the hair sometimes observed in regions irritated by cantharides and blue ointment or on paralyzed extremities.

The *treatment* of disfiguring hypertrichosis is demanded, as a rule, only in certain forms. Hirsuties adnata universalis is hardly susceptible of treatment. In most cases, too. the hair soon falls, to give place to lanugo of normal length, and the condition persists as a rare exception only. Aid is sought most frequently for thick, bristly hairs on exposed warts and pigment moles and for the abnormal beard in females.

The former are best removed by the radical extirpation of the nævi. Where the latter are left intact, and merely the disfiguring growth of hair here or on otherwise normal skin of the face, hands, etc., is to be removed, different methods must be employed according to circumstances. The palliative measure of shaving with the razor serves the purpose but incompletely, as the stumps projecting from the follicles would disfigure a female face almost as much as the long hair. A better plan is the destruction of the hair by a caustic paste which is in habitual use in the Orient and among the Jews for the periodical removal of the stubble of the beard. Orpiment (yellow sulphide of arsenic, auripigment) and unslaked lime are mixed with water to a paste and boiled. This is applied by means of a spatula and allowed to remain for about ten minutes until it dries, and then rapidly scraped off with a dull knife. The skin is then washed with lukewarm water, covered with a white cosmetic, and powdered. Sulphide of calcium paste, which is prepared by the introduction of hydrogen sulphide into hydrated lime, acts still more rapidly (Boettger's paste), and is therefore more convenient in practice, especially as it can be procured ready-made from the druggist.

As these pastes cauterize the hair shaft into the follicle, the skin thus treated looks smooth, and the after-growth does not appear

until two or three weeks, when the application is repeated. Where the number of hairs is small epilation is probably best, but of course it must likewise be repeated at intervals. As a radical treatment it has been advised to insert into the several follicles needles heated in the fire or by the galvanic current, or dipped into caustic fluids such as carbolic or chromic acid.

On the other hand, epilation by electrolysis, first performed by American physicians and since perfected, has risen to the importance of a methodical and commendable treatment of hypertrichosis.

Epilation by electrolysis was invented by the ophthalmologist Dr. Michel in St. Louis, and was first employed for trichiasis. To Hardaway belongs the credit of having introduced it into dermatology. Used for years in America, this method has been recently also more generally employed in Europe. C. Heitzmann, George Thomas Jackson, G. H. Jos. Müller, Michelsohn, Behrend, Lustgarten, and others have reported favorable results. The method is essentially this: A needle-shaped negative (zinc pole) electrode is introduced into the hair follicle and the hair papilla is destroyed. The latter is the result of the decomposition products formed, namely, the caustic alkalies—a fact which admits of no doubt when the physical process is considered. As to the details of the operation, the number and nature of the galvanic elements, duration of the action, and conformation of the needle-holder, statements differ so widely that I prefer to sketch briefly the procedure employed at my clinic. The needles we use are polished sewing needles or German silver needles without points, so as to avoid as far as possible injury of the hair follicle. The needle-holder, five inches in length, is very light (seventy-five grains), and carries a conducting cord which is likewise as light as possible. For localities which do not permit manipulation with the long needle holder—as, for instance, in the submental region—Moeller's clamp forceps are used. The intensity of the current is measured and regulated by a galvanometer of tested reliability and by Dr. Gärtner's graphite rheostat; the strength ranges from one-half to one milliampère. The use of the latter apparatus makes us independent of the quality and condition of the battery and the number of the elements, and hence the effect is very uniform. Every single hair, according to its thickness, is exposed to the action of the current for from twenty to thirty seconds, and then extracted with pincers or left to spontaneous exfoliation. Three sittings are given every week, and in each about thirty hairs are destroyed without giving rise to marked local irritation, especially if aided by protective ointments and powders. The epilated spots heal partly without leaving a trace, partly extremely fine, shallow depressions, often only noticeable in oblique light and which hardly affect the cosmetic purpose, indicate the place where the hair

was seated. At times, though, quite ugly and keloid-like cicatrices result.

Before closing this section devoted to the hypertrophy of the hair, it may not be inappropriate to consider the condition known in the literature as "plica polonica." Practically it means a felting of the hairs which usually affects those of the head, more rarely those of the beard or the pudendal region. For a long time, however, even a nosological importance was ascribed to this condition, especially in Poland and Russia where numerous cases occurred; for instance, in the former country, in 1842, there were more than five thousand of them. The disease was "endemic," as it were. It having been noticed that such plicæ formed in very sick patients who were long bedridden, people soon came to look upon them as metastases of internal diseases. Then, as a logical deduction, when the plica which raged within the body in the form of eclampsia, epilepsy, rheumatism, etc., was tardy in coming out, an effort was made to produce it artificially by felting the hairs with pitch or honey and favoring its further development by abstaining from the use of the comb. Even the recession of the plica had to be feared, and therefore it had to be guarded against "taking cold" and still more against healing. The outgrowth and falling of the plica, the hemorrhage and painfulness when it was cut off, were cited as proofs of its organic life; while a fungus ascribed to it by Günsburg, and its classification into male and female, simple and complicated (Alibert), served to support its scientific existence. The literary battles which, even in the sixth decade of this century, were waged by Beschorner, Hamburger, Hebra, and others against the superstition of the plica disease, have now become superfluous. We know that the felting of the hairs occurs only when the comb is neglected, and thus such cases are met with by us and others in persons who do not comb themselves owing to slovenliness or some painful affection of the hairy scalp (eczema idiopathicum et e pediculis, syphilitic ulcers), especially when ulcerous secretions and exudates favor the adhesion of the hairs. The endemics of plica have died out since the respective populations were induced to extirpate their plicæ with scissors and comb, either by enlightenment or the interference of the authorities, and since the younger generation has come to use the comb regularly. Sporadic cases of plica are treated like eczema of the scalp. The adhering crusts having been softened by ordinary or lice-killing oils (petroleum, Peru balsam, naphtholated oil [two per cent]) and detached by washing with soap, the tangled hairs are separated without much trouble with the finger and comb, passing from their points toward the scalp, and that ends the plica.

HYPERTROPHY OF THE NAILS.

An increase above the normal in mass and circumference of the nail is called hypertrophy. These conditions do not always occur simultaneously ; but they are accompanied, as a rule, by changes in structure, color, consistence, and form of the nail.

The hypertrophic nail appears excessively long and projects beyond the tip of the finger several times its normal length ; either it maintains its normal breadth, direction, and quality, or else its protruding portion is thin, glassy, fractured or widened, thickened, caseous opaque, uneven, simply claw-shaped or spirally twisted like a ram's horn (onychogryphosis). At other times the nail is not lengthened, but, while otherwise normal, is widened so that its edges cut into the nail fold and cause painful bleeding, inflammation, suppuration, and profuse granulations (paronychia). Finally, the nail appears shapelessly thickened, tumid, uneven, set with prickly spines, at the same time hard or brittle, its surface marked with longitudinal and transverse furrows and pits, rough (asperitas, scabrities unguium), at its anterior margin thick and compact, or coarsely cellular and detached from the nail bed. This alteration affects either a few finger or toe nails, the latter more frequently, or all of them together.

Anatomically the degenerated and hypertrophic nail alone shows an abnormal arrangement and quality of the horny cells. There is associated with this, in excessive and chronic forms of hypertrophy, an outgrowth of the papillæ of the matrix which project as vascular new formations from the latter as much as several centimetres beyond the middle of the nail bed into the body of the nail, so that when the latter is cut at that point bleeding papillæ are found. In acute and temporary forms of hypertrophy and degeneration of the nail, however, we find merely hyperæmic or inflammatory swelling of the papillæ or no noticeable alteration. Sometimes the nail bed is apparently unaltered, or its ridges are likewise hypertrophied and set with numerous papillæ (Virchow), in which case from the ridges also a hyperplastic epidermal mass is produced which thickens the substance of the nail from below or lifts its body. These changes are in direct proportion to the special cause of the hypertrophy of the nail. Some cases may be due to a congenital tendency. Neglecting the periodical trimming of the nails only occasionally leads to their hypertrophy ; more frequently long-repeated pressure on the toes, especially the outer large and small toes, is at fault—a disturbing cause which at other points of the skin produces an analogous formation, cornification, and papillary hypertrophy. Other causes are all those chronic processes of the skin which at other points induce cell infiltration of the papillary layer and hyperplasia of the epidermis,

like chronic eczema, psoriasis, lichen ruber, elephantiasis arabum, lepra, syphilis, and ichthyosis. In the latter we often meet with gryphotic degeneration of the nails. In syphilis the alteration is frequently confined to a portion of the nail, corresponding to a papule infiltrating merely a part of the matrix papillæ ; the alteration is permanent when a portion of the papillæ has perished by atrophy or ulceration. Eczema, psoriasis, and lichen ruber produce degeneration of all the nails, even when those processes do not affect the fingers directly, that is, in a reflex way. Also some general conditions of the organism seem to give rise to opacity, grooving, and brittleness of the nails. Such is, according to Hutchinson, the syphilitic diathesis—onychia syphilitica—which seems to be nothing else than the onychia (psoriasis unguium, Anderson) occurring in consequence of chlorosis or acute febrile conditions (Vogl), and finds its analogue in the formation of fatty scales in other regions (pityriasis tabescentium, seborrhœa capillitii) associated with general nutritive depression. Such onychiasis can also be observed on the fingers in local asphyxia of the hands and in sclérodactylie.

The *prognosis* of the degeneration of the nails depends upon its special cause and the possibility of eliminating it. The most problematical is the form due to general conditions, while those resulting from local processes and chronic exanthemata are more favorable.

The *treatment* is effective only in certain forms. Gryphotic or simply lengthened nails are cut off with scissors or bone forceps ; any papillary outgrowths which may have been divided are cauterized. In the onychia due to syphilitic local infiltration, covering with mercurial plaster produces a rapid cure. The asperitas unguium associated with eczema, psoriasis, and lichen ruber is favorably influenced by all the agencies which are effective against these diseases—namely, tar, diachylon ointment, applications of potash and sublimate, rubber finger stalls, and salicylic plaster ; for as the papillæ of the matrix regain their health, the nail formation becomes normal. Of course the improvement affects only the newly formed nail growing out of the matrix, not the present degenerated one. But as the growth of the nail is very slow and it takes many months before a new one is substituted for the old, the improvement is not manifested until late, often long after the original disease has been cured. The internal use of arsenic and iron in appropriate cases (chlorosis, psoriasis, lichen) acts in like manner and equally slowly.

For the painful ingrowing of the nail (paronychia), which some endeavor to cure by the forcible avulsion or exsection of the nail, a quite painless procedure is to be advised. A pledget of straightened lint, the length of the nail fold, is inserted, thread by thread, between the fold and the edge of the nail by means of a chisel-shaped sound ; then soap plaster is wound around in circular strips, thus securing

the threads and drawing the fold away from the nail. The dressing is renewed daily, and subsequently becomes very easy as the groove of the fold is widened. The inflamed margin heals readily after any proliferations present have been removed with the scissors, sharp spoon, or caustic (powdered alum, chloride of iron, silver nitrate, etc.).

LECTURE XXXIV.

CONNECTIVE-TISSUE HYPERTROPHIES. DIFFUSE : SCLERODERMA (AINHUM—APPENDIX : SCLEREMA NEONATORUM) AND ELEPHANTIASIS ARABUM—ELEPHANTIASIS TELANGIECTODES ET NEUROTICUM—MYXŒDEMA—CIRCUMSCRIBED : PAPILLOMA (FRAMBŒSIA).

THE diseases appearing as hypertrophy of the connective tissue of the skin manifest themselves either as diffuse and more extensive thickenings of the cutis—scleroderma and elephantiasis arabum—or in the shape of circumscribed and prominent tumors, papilloma (frambœsia).

DIFFUSE CONNECTIVE-TISSUE HYPERTROPHIES.

SCLERODERMA.

Sclerema adultorum is the term by which Thirial in 1845 described the very peculiar disease now to be discussed. Before his time it was certainly observed by many, but has been unmistakably described only by Curcio (1752), Henke (1809), and Alibert (1817). Subsequently this cutaneous affection also received the names scleroderma (Gintrac, 1847), scleroma, chorionitis, sclerostenosis cutanea (Forget), cutis tensa chronica (Fuchs), "keloid of Addison," elephantiasis sclerosa (Rasmussen), cicatrizing sclerema of the skin (Wernicke), sclerosis telæ cellulosæ et adiposæ (Wilson), etc.

While on the whole all these manifold terms were intended to mean essentially the same process which, as Besnier justly pointed out, was first characteristically described by Alibert in 1817, still in recent years the effort has been made by many authors to apply different names to the diffuse and localized forms. This confusion was especially due to the fact that, to many physicians, forms resembling scleroderma, which Er. Wilson and other English authors after him had described as morphœa, appeared to be true scleroderma or at least closely related to it ; or that, with reference to the latter, the theory of a trophoneurotic origin seems more probable (Schwimmer) ; or, finally, that the sclérodactylie of French authors (Ball, Hallopeau, Dufour, Lepine) was erroneously identified with scleroderma.

Thus Besnier proposes for the diffuse and general form of the disease the name of sclérémie (sclereme, Alibert) ; for the partial

sclerosis, dermatoscléroses en plaques ; and for morphœa of English authors, scleroderma. Hardy still distinguishes three forms of the disease : 1, sclérodermie œdemateuse (Besnier's sclérémie) ; 2, sclérodermie en plaques ; 3, sclérodermie des extrémités—this would be sclérodactylie. But it would be advisable to retain the term *scleroderma* (sc. adultorum), as opposed to sclerema neonatorum to be discussed in connection with it ; for I cannot share the opinion that the localized forms of the process are of a different nature from those which are diffuse and extensive (généralisées, Besnier).

Although scleroderma on the whole is rare, still a sufficient number of cases are recorded in the literature, and there is no reason for publishing every case. I myself have seen a relatively large number of scleroderma cases, according to a recent calculation probably between one hundred and fifty and two hundred, chiefly in private and clinical out-patient practice ; for it is in the nature of the disease that the sufferers but rarely come to the hospital or remain there. Nevertheless our knowledge of the disease has scarcely passed beyond a tolerably exact, objective symptomatology.

Scleroderma, sclerema adultorum, is a chronic disease characterized by a diffuse hardness, board-like consistence and stiffness, and relative contraction of some circumscribed or very extensive regions of the skin, occurring spontaneously, without inflammatory symptoms or perceptible involvement of the general organism.

The disease affects irregularly the most various regions of the skin, chiefly of the upper half of the body, more rarely of the lower extremities. It is confined either to smaller regions between which the remaining skin continues perfectly normal, or else it is scattered diffusely over large surfaces, the back, the abdomen, the limbs, or the face. According to these variations in localization, extent, and stage of the local process and the general disease, the concrete case of scleroderma presents itself either in forms following a general scheme or exhibiting more special features.

The most pronounced symptom is furnished by what I have termed the first stage of the affection—namely, the sclerosis of the skin. It appears in the form of irregular patches, the size of a dollar, the palm of the hand, or larger, or in band-like streaks which are tightly stretched, depressed, or projecting, or else as a diffuse and uniform thickening of the entire integument. The sclerosed region is either sharply demarcated from the healthy skin by a colorless or pink to bluish-red areola, or it is merged gradually here and there into the normal surroundings. It may project moderately or may be flat or slightly depressed. Its surface may be smooth or covered with wrinkled epidermis and set with thin scales. It may have a lardaceous lustre, or be dull, tawny-white, waxy, or like alabaster, sometimes pink to brownish red. Occasionally it is sprinkled with

yellowish-brown or dark-brown pigment spots resembling freckles, intermingled with white, non-pigmented, and slightly depressed bands and streaks, or it may be diffusely stained dark brown to bronze brown. Pressure with the finger on the sclerosed skin causes no lasting pitting; it feels like a board, stiff and cool as in a frozen corpse. It cannot be pinched into a fold or displaced on the underlying structures—fasciæ, muscles, or periosteum—at all, or at best but very slightly. It rather seems to be intimately adherent to them and merged into them, although at the same time it is contracted. too narrow for the parts it covers. When it affects the elbow and finger joints the sclerosis fixes them in semi-flexion, while the skin of the extensor sides is passively stretched. When the face is attacked the features appear as if frozen, entirely immovable, incapable of the slightest mimetic expression. Neither pain nor joy can alter the countenance, "petrified" as if it were carved in marble. Owing to the condensation of the immovably stiff skin, the nose is at the same time narrowed, the mouth is contracted and can be only imperfectly opened. Streaks of sclerotic skin sometimes sink down deep below the level of the integument, as if drawn down by a subcutaneous tense band; again project ridge-like with one edge. In this manner the sclerosis at times crosses the mamma, dividing its rounded eminence into two halves or retracting the nipple like a navel.

The temperature of the sclerosed skin is sometimes normal, sometimes slightly elevated, but as a rule a little lower than the healthy skin, as much as three degrees Fahr. Pressure is rather painful, while subjectively pain or burning is rare; usually there is only a sensation of tension and itching or deep seated pains (in the bones). The tactile and other senses are generally normal, rarely somewhat increased, and still more rarely slightly dulled, even to anæsthesia. The resistance to the electric current in the sclerotic regions was found diminished in tests made with patients at my clinic by Erben, but Eulenburg found it increased. Such opposite results may depend on the stage of the cutaneous alteration or possibly on the mode of examination (Lewith). The secretion of sweat in the sclerosed region has only a few times been found slightly altered; the sebaceous secretion was normal. Scleroderma likewise does not at first change in other respects the nutritive and functional activity of the affected skin, so that, for instance, chemical and mechanical irritations may cause inflammation and suppuration, and the eruptions of erysipelas, acne, variola, and zoster have been observed on the patches. Yet in one case at our clinic a subcutaneous injection of one-sixth grain of hydrochlorate of pilocarpine, while causing general perspiration, produced but little sweat on the sclerotic regions and those which had become atrophic.

In some cases the mucous membrane of the tongue, the gums, the

soft palate, and the pharynx (Arning, Sedgwick, Fagge, Hallopeau), and in one case the vagina and intravaginal cervix (Heller), as well as the interior of the larynx, were the seat of hard, band-like, retracted streaks.

The localization of scleroderma is in general most irregular. When diffuse we find the skin of the face and arms, the cervical, acromial, and clavicular regions chiefly affected, more rarely the thighs, legs, nates, and abdomen. The greatest extent of the sclerosis which I have observed was in a young lady, aged twenty, from Budapest, in whom only a few fingers and strips the width of the hand in the epigastrium were free. The poor girl appeared as though enclosed in a coat of mail which compressed her actually from all sides and hindered respiration and movement. In most cases, besides diffuse infiltrations, spots and streaks are likewise present. These are usually located without any anatomical arrangement, across the median line of the body, the longitudinal axis of the body, the main course of the nerves, and the cleavage lines of the skin in various directions.

Not uncommonly, however, we find a marked congruence in localization and extent of the scleroderma with the direction and distribution of the peripheral nerves, and a unilateral localization as in zoster, especially corresponding to one or more branches of the trigeminus, as reported by Hutchinson, Higgens, and Nettelship. I myself have among my cases noted in this respect the following :

One case, along the right frontal nerve, in a boy of sixteen.

One case, right unilateral, cervical, acromial, and upper dorsal regions and arm, in longitudinal and spiral streaks as far as the fingers, corresponding to the lower cervico-brachial plexus and the first and second thoracic nerves, quite analogous to a right cervico-brachial zoster, in a girl of twelve.

Two cases right unilateral, along the branches of the trigeminus, in the form of sclerotic strips and patches, in a girl of fourteen and a boy of twelve.

One case along the right saphenus nerve, in a physician.

The development of scleroderma is sometimes preceded by vague neuralgias, rheumatoid pains of the joints and muscles ; but chiefly without any prodromata, the skin being previously perfectly healthy. It arises acutely and unperceived within a few days. An accidental touch or the sensation of tension first calls the attention of the patient to the alteration. Not rarely, too, the sclerosis is preceded by doughy infiltration or a vivid erythematous redness of injection. The latter I have often observed to remain unchanged for weeks, until it was replaced by pallor and accompanying sclerosis. With the characteristic sclerosis of the region the process has reached its height locally. The sclerotic patch or strip may then remain station-

ary for a variable length of time or extend to neighboring regions. This may occasionally be preceded, especially when the patches are sharply demarcated, by a rose-red areola of injection.

The further course may take one of two directions. In the first the sclerosis disappears completely and the region may regain its former condition, flexibility, and mobility. This may ensue in some regions in a few days, in others after many months. Persons with limited experience are then very apt to ascribe this involution of the sclerosis to the influence of the treatment undertaken, but in the disappearance of such sclerotic patches the general disease is not affected. On the contrary, other regions or some of those recently improved are again attacked by the process. In the second the sclerotic skin, which at first feels brawny, hard, and thick, becomes atrophic, thin, parchment-like, cicatricially white or glossy red, irregularly sprinkled with pigment, extremely shortened, tense, and immovable. The underlying adipose tissue and even the muscles disappear under its pressure, so that the atrophic skin seems to be directly attached to the bone. It is quite apt to undergo ulceration and even gangrene, especially on the flexor sides of the joints, and consecutively there may occur thickening of the articular bones and pseudo-ankylosis (sclérodactylie [?], Ball). This condition, therefore, must not, as formerly, be considered a special form (cicatrizing sclerema of the skin, Wernicke), as opposed to the type above described, which appears as sclerema elevatum ; but merely as the terminal stage, the second stage of scleroderma (stadium atrophicum), a process beginning with elevated thickening (stadium elevatum). A return to the normal is no longer possible from the atrophic stage.

The course and termination of the disease depend upon the course of the local process described above. For several years the scleroderma may persist with changing localization and, as has been observed in a few cases, heal, the skin again becoming normal and no new sclerosis occurring. This may be hoped for particularly in persons with a single patch or a few smaller ones. In most cases, however, the patches, though varying at first, increase in number and extent and terminate in atrophy. In this way the process becomes serious not only for the skin, but also for the entire organism. Although the general condition does not seem to be affected either in the beginning or within the earlier years of the disease, the patients being well nourished and none of their important functions disturbed, still there is a gradual failure of nutrition, a pronounced marasmus, coming on with mental depression, sleeplessness, neuralgic and rheumatic pains, contractures of the joints and their local consequences (in one case of Troggler's, oblique pelvis). The fatal issue, thus far observed in rather more than a dozen cases (Förster, Köhler, Gintrac,

Auspitz, Arning, Rasmussen, Stein, Walter, Rossbach, Heller, Mader-Chiari, Chalvet and Luys, Westphal, Schwimmer, Poisson), ensued from the most manifold complications, seemingly individual and not directly connected with the process in the skin, such as Bright's disease, emphysema, bronchiectasis, pulmonary tuberculosis, pneumonia, heart disease, myocarditis, anæmia. Strassmann has seen a case of thirty-one years' standing, in which the patient in general felt pretty well.

Thus far it has not been possible to ascertain the *anatomical* alteration underlying the scleroderma which presents itself in this peculiar manner, although repeated examinations have been made, partly by prominent histologists, of the skin excised both after death and during life. All investigators agree as to a thickening and condensation of the connective tissue of the cutis, with increase of the elastic fibres at the expense of the subcutaneous cellular layer and the atrophying fat lobes, so that the homogeneous, coarsely fibrous, and narrow-meshed cutis tissue reaches close to the fascia or periosteum, adhering to them without any loose intermediate layer. Besides, there were found increase of pigment in the rete and corium, ectasia of the sweat glands, hypertrophy of the organic muscular fibres (Neumann, Rossbach), which alterations, however, seem to possess more of a consecutive importance. A more essential feature, perhaps, might be the narrowing of the vessels, which seem to be compressed partly by closely adjoining parallel trabeculæ of sclerosed connective-tissue fibres, partly by lymph-cell layers which here and there surround the vessels like a sheath several times as wide as the latter (Rasmussen, Kaposi), or, as in the case of Schwimmer-Babes, by concentric hypertrophy of the media and intima. Still, I agree with Wolters' recent objection to the theory of Chiari, who looks upon the process as resulting from an inflammation, since there are no clinical or histological evidences of it. With respect to the latter, we must state in particular that even in fresh patches of sclerosis there is neither dilatation of the vessels nor œdematous widening of the meshes of the tissue. Atrophy of the follicles and glands occurs only in the atrophic stage. Still less is there any reason for looking upon scleroderma as a congestive dermatosis (Auspitz).

Hence the anatomical examinations thus far have failed to elucidate the *cause* either of the alteration affecting the skin itself or, much less, of the general process in scleroderma. In Heller's case alone, the occurrence of an atrophy of the thoracic duct might furnish ground for the assumption that obstruction to the lymph current caused its stagnation in the cutis with consequent hypertrophy. Although I am inclined now, as I was years ago, to look upon a local stagnation of the lymph in the tissue spaces of the cutis as the basis of the local alteration, I cannot believe that a mechanical obstruction

in a collecting vessel should be held responsible; for scleroderma does not correspond to the collecting field of a lymph vessel, but appears in quite irregular localities, and, further, in marked mechanical obstruction to the lymph current another variety of hypertrophy (elephantiasis arabum) develops, but not the specific scleroderma. We are forced, therefore, to assume as a remote cause of the disease a trophic disturbance influenced by the central nervous system, although it has not as yet been possible to prove this definitely. The isolated finding of a sclerosis of the anterior horns (Chalvet and Luys) and foci of sclerosis in the cerebral convolutions (Westphal), as well as thickening of the sheaths and fatty atrophy of some nerves situated within the scleratrophic skin (Babes), should be interpreted rather as local phenomena of the general process than as their cause. The temporary improvement of the disease after galvanization of the sympathetic would just as little justify positive deductions in this respect. For the present the assumption of a central nervous cause for scleroderma is no more than a hypothesis. It may possibly gain force from the statement made by some authors that the disease appeared a few days after a violent mental emotion.

One is inclined to assume an affection of the peripheral nerves as the first cause of the local nutritive disturbance, in view of the above-mentioned cases of unilateral distribution or localization along the course of the nerves, five of which I have seen, as stated before. These must be trophic disturbances, since there are but few data in favor of an angioneurotic one. Thus far, however, we lack any anatomical demonstration of such a nerve alteration, and for the diffuse forms there is not even any external reason for such a hypothesis. It is probable that the apparent correspondence with the course and distribution of the nerves should be referred rather to the vessels, whose congruence with the course and distribution of the nerves Pfeiffer has pointed out in recent years; and that such a distribution of the scleroderma can support only the assumption of an etiological relation of the latter to the demonstrated vascular disease. In some cases there is noted a preceding and relapsing erysipelas or rheumatism; not a true articular rheumatism, but vague pains. Whether these can always be referred to a true myositis, such as has been seen in a few cases and recently placed in the foreground by Thibierge as belonging to the symptom-complex of scleroderma, it will be difficult to decide. In one case observed by me, general myositis of nearly all the muscles of the extremities and trunk, with painful stiffness and contractures and general emaciation, had existed for a year when a scleroderma extending from the thorax to the nates was discovered. But I found the atrophic stage already pronounced, so that the process in the skin had perhaps set in simultaneously with that of the muscles. The latter may also suffer consecutively through

the pressure by the tensely retracted skin and inactivity. I have never observed a direct extension of the process from the skin to the muscles. In one of my earlier cases a sudden mental shock is positively stated as the starting point of the disease. In the majority, however, there is absolutely no plausible etiological basis. The female sex suffers in three-fourths of all the reported scleroderma cases. We can understand that under such conditions, and in view of the fact that persons with heart lesions, Bright's disease, tuberculosis, and other complications which alter the nutrition are among them, chloranæmia is cited as one of the causes; but this furnishes no explanation, since the nutrition seems to be very good in most cases, at least in the early years. The cases thus far affected mainly persons in middle age; but we, like other authors, have observed some in older individuals, as well as in children of six and two years.

The *diagnosis* of scleroderma is not difficult when the elevated stage is present. Even the inexperienced will at once think of scleroderma when the skin to the touch makes the impression of a frozen cadaver. True keloid never has the stiff and immovable feel, nor is it diffuse in extent; myxœdema is more doughy. In the atrophic stage, however, and where there is but a single patch, the differentiation may be difficult from certain forms of lepra (morphœa atrophica et lardea, Wilson; pigment lepra), xeroderma (Kaposi), and when confined to the face and head from other atrophying forms, such as lupus vulgaris and erythematosus, etc.

The *prognosis* of scleroderma as a local cutaneous disease is not favorable, as most cases last for an indefinite length of time and reach the atrophic stage, whence a return to the normal is no longer possible. This applies even to the smallest patches. But for the general organism and the duration of life such patches of scleroderma are of no importance. It is different when the distribution is more general. In such cases, without an exception, in time marasmus will either directly or through a consequent complication bring the disease to a fatal issue, but as long as the sclerous stage is present there is some hope of recovery.

The *treatment*, though in no respect reliable, may do a good deal in this stage, and in some cases when directed to the stimulation of the general nutrition and the metabolism. For internal use may be recommended roborants, iron, quinine, the bitters, cod-liver oil, arsenic, together with tub, steam, mud, iron, sulphur, iodine, and brine baths; during the summer, milk cures, residence at spas and in the mountains, sea and river baths. Locally, methodical massage may be combined with inunctions of bland fats, ointment of oxide of copper, glycerin, vaseline, and lanolin. Inunctions of mercurial ointment, with potassium iodide internally, have proved ineffectual.

On the other hand, some authors claim to have had favorable results from the use of the constant current applied to the sclerotic regions and the sympathetic.

A disease reported from among the Nagôs negroes, called

AINHUM,

will here find its appropriate place. It affects one or both little toes, sometimes also the fourth toe. Without any known cause and without any prodromal symptoms there appears on the lower surface of the first phalanx of the little toe a shallow groove, which gradually becomes deeper and wider, and at the same time extending upward over the lateral margins, finally encircles the member. In this way, in the course of from one to ten years, there results a stem-like thinning of the first phalanx, while the anterior portion of the toe is thickened to a form resembling a tuberous potato, and eventually the spontaneous loss of the toe occurs. The remaining wound skins over in a normal manner. Minute histological examinations, following Dr. Da Silva-Lima's first report (1867) from Bahia, made by Herrmann Weber, Wucherer, Schüppel, and others, favor the assumption that we have to deal here with a hypertrophic thickening resembling scleroderma with subsequent atrophy of the connective tissue of the cutis, and that the atrophy of the underlying bone is merely due to pressure, while the hypertrophy of the anterior phalanges must be looked upon as a sequel. Da Silva-Lima has also succeeded in arresting the process in a few cases by early incision of the constricting sclerotic cord and thus saving the toe, while in the stage of advanced atrophy all measures proved futile.

Similar conditions of the toes occur also in consequence of mechanical, congenital, and other displacements, and are occasionally considered and described as "ainhum."

Essentially different from scleroderma adultorum, and yet resembling it, is the affection known as

SCLEREMA NEONATORUM

(Chaussier), algidité progressive (Hervieux), induratio telæ cellularis neonatorum, induration of the cellular tissue of the new-born of many authors. It usually attacks children during the first months of life, more rarely those of one to two years. It begins with coldness, œdema, and hardness of the feet and legs. The skin at these points is tense, glossy white, or shows a reddish lustre, sometimes dirty yellowish-brown. It becomes cachectic and hard to the touch, but capable of being depressed with the finger—that is, œdematous. Within some hours or one to two days the alteration extends with

similar symptoms over the abdomen, trunk, upper extremities, and face, where the skin also becomes resistant, cool, and immovable, while the lower extremities first attacked, in which the œdema has disappeared, become thinner and harder, wrinkled, and almost mummified. The temperature of the skin and of the internal organs falls steadily from three to five degrees Fahrenheit daily. The face, whose integument is stiff, fixed in the direction of the predominant muscular action, looks wrinkled like that of an old man. The stiffness of the mouth renders suction and nutrition impossible. Children thus affected lie motionless, as if half-frozen; the only signs of life being feeble movements of the less diseased parts of the body and faint moaning. The patients die in from two to ten days, under a continuous fall of temperature. Here and there the œdematous skin may become inflamed, with rise of temperature, cyanotic, and then gangrenous, while complications occur at the same time in other organs. Rarely the fatal issue is delayed longer. Still more rarely the temperature rises again in the cold parts, œdema and sclerema disappear, when gradual improvement and complete recovery ensue.

The proximate *cause* of the symptoms of sclerema lies in a retardation of the capillary circulation in the peripheral parts of the body. The remote cause is furnished by all conditions which weaken the heart's action or inhibit heat production. Hence the disease occurs in children with heart lesions, or those weakened by pleuropneumonia, chronic catarrh of the respiratory and digestive tract, diarrhœa, follicular ulceration of the intestine, improper care, hereditary syphilis, and congenital debility.

Anatomically we and others (Förster, Virchow, etc.) have found, aside from œdematous suffusion of the cutis tissue and firm, stearin-like quality of the panniculus, no marked change, particularly no noteworthy cell infiltration or connective-tissue hypertrophy, in contradistinction to scleroderma. Löschner reports thickening of the corium and the occurrence in patches of embryonal connective tissue with numerous cells. Congestion in the peripheral lymph channels and lymphangitis (Pastorella) represent rather consecutive conditions. Ludwig Langer also explains the occurrence of sclerema neonatorum by the stearin-like quality of the infantile panniculus adiposus.

The *treatment* must aim to raise the body temperature and vital power by artificial heat, friction of the body, appropriate nutrition, and stimulants, and in the end to stimulate the heart action and capillary circulation by overcoming the complications. Improvement manifests itself by more vigorous heart action and rise of temperature in the skin, which is soon followed by the disappearance of the sclerema. In that event recovery may be hoped for.

Here should follow in natural sequence the discussion of the cutaneous œdema (anasarca) which arises from mechanical (pressure) or functional (caused by neuroses or disease of the vessel walls) retardation or obstruction of the blood current; usually on dependent parts of the body, regionally as in varicosity or pressure on the vessels by a callus of bone, etc., or extensive and general in central obstruction to the circulation as in heart lesions, emphysema, etc. But we believe we may restrict ourselves to the mere mention, since it is the province of general and internal medicine and surgical pathology to treat of this subject more in detail.

As less known and belonging more to the domain of dermatopathology we may refer only to the *acute circumscribed cutaneous œdema* described under this head by Quincke and discussed also by Dinkelacker, Jamieson, and, particularly, minutely by Strübing and Riehl in connection with cases under their observation. This occurs in repeated attacks in the same individual, regularly in combination with gastric disturbances, vomiting, and diarrhœa, and appears in the form of œdematous, tumid, whitish swellings on the skin of the face and other regions, also on the mucous membrane of the mouth, pharynx, and larynx. In the latter case dyspnœa and danger of suffocation are the first results of the swelling. It is not difficult to understand that this is an angioneurotic disturbance of circulation and effusion of serum (Strübing, Riehl) in the sense of an urticaria, perhaps a giant urticaria (Milton).

We can trace also to a stasis of the tissue juices that diffuse connective-tissue hypertrophy of the skin represented by the disease known as

ELEPHANTIASIS ARABUM

s. pachydermia (Fuchs).

The term elephantiasis arabum is applied to an hypertrophy of the cutis and subcutaneous cellular tissue which is confined to some regions of the body, due to local circulatory disturbances; it appears with chronically recurring inflammation of the blood and lymph vessels, erysipelas, and persistent œdema. In its further course the underlying structures also take part in the thickening and increase of volume of the affected regions.

The name elephantiasis was first used by the translators from the Arabic (hence elephantiasis arabum) as synonymous with the Arabic original, *dal-fil* (elephant's foot), signifying a thickened leg. This remark is intended to guard against the erroneous impression apt to be conveyed that the disease is one endemic in Arabia. While elephantiasis arabum in certain forms is indeed far more frequent and

almost endemic in many tropical and subtropical regions, in Egypt, on the coasts of the Mediterranean, Arabia, the west coast of Africa, Brazil, the Antilles, the South Sea islands, and the coasts and islands of the southern seas in general, yet isolated instances are met with in all countries and regions, some forms even pretty frequently in our own country.

The most frequent seat of the affection is one lower extremity (rarely both), and it is generally confined to the leg and foot, more rarely implicating also the thigh as far as the gluteal fold or even one of the gluteal regions. Next in frequency it attacks the skin of the penis and scrotum, the labia majora and minora with the clitoris; while the upper extremities suffer but rarely, the cheeks, ears, back, and other parts of the body only exceptionally.

In the two favorite localities, the lower extremity and the genitals, the symptoms of the disease appear in a typically pronounced manner. Elephantiasis arabum of the leg develops, as a rule, in a chronic manner, with inflammatory symptoms renewed occasionally, paroxysmally, or at irregular intervals, and these at first represent the only morbid phenomena. Without known reason or from some local cause a diffuse erysipelatous redness or a more deep-seated dermatitis occurs on the leg, or strips of reddening and painfulness, lymphangitis, phlebitis, pain, tension, and swelling of the skin associated with fever. Shortly after the inflammatory attack has run its course it recurs spontaneously or from local causes. Every such attack leaves behind a slight œdema of the skin, which progressively becomes more marked and enlarges the circumference of the leg as the attacks succeed each other after months or one to two years. Excepting the greater tension and the white or reddish lustre, the surface of the skin appears unaltered. Pressure with the tip of the finger will readily leave a depression which betrays the œdema of the subcutaneous cellular tissue. At the same time, however, we can demonstrate an increase in volume and hardness, and the attempt to lift a fold will show that the skin has become thicker, firmer, and tenser.

In the further course the inguinal glands form large, hard swellings. In some cases the swelling and hardening of the inguinal glands precede the symptoms on the leg, and for this reason the affection has also been called the glandular disease of Barbados (Hendy and Rollo). In from five to ten years the leg becomes enormously enlarged and disfigured, as the hypertrophy meantime has involved not only the skin and subcutaneous cellular tissue, but also the underlying soft parts and bone.

The leg is enlarged to two or three times its normal volume and represents a clumsy cylinder of a monstrous circumference and appearance, which is merged directly, after obliteration of the fossæ

about the ankle, with the tumid, distended dorsum of the foot, and may thus be well likened to an elephant's foot (elephantopus, Barbados leg, Cochin leg, roosbeen of Surinam). A deep furrow over the ankle joint, in which rancid cutaneous secretions and epidermal detritus accumulate, divides the thickened leg from the foot. The foot at the same time is widened, and so are the toes, whose elephantiasic skin is not rarely continuous, with the possible exception of some shallow grooves which indicate their limits. The common integument of a thus monstrously thickened limb is dry, not perspiring, tensely stretched, with a dull lustre or livid, in places dirty brown (elephantiasis fusca s. nigra) from pigment or adhering seborrhœic masses of epidermis. The epidermis is here and there thin, parchment-like, fissured and squamous, elsewhere tessellated, thick and dirty brown as in ichthyosis serpentina or gathered into thick scales or horny cones as in ichthyosis hystrix. Otherwise the elephantiasic skin is smooth (elephantiasis glabra), at other points uneven (elephantiasis tuberosa), or set with numerous filiform and mulberry-like dry or weeping warts (elephantiasis verrucosa s. papillaris). Besides, we find occasionally excoriations, shallow or deep ulcers with callous borders, necrotic at the base or furnishing a thin secretion, or here and there a weeping and crusted eczema. In some cases of pachydermia in which the process is diffuse or extends in strips of induration from the leg over the inner surface of the thigh to the inguinal glands or the nates, spontaneous bursting of the skin, or a cord-like lymph vessel from which lymph exuded in drops (true lymphorrhœa), has occasionally been observed. I have seen such lymphorrhœa from an accidentally injured lymph vessel at the inner surface of the elephantiasic thigh, and once from vesicular lymph varices of the scrotum.

To the touch an extremity affected with elephantiasis is very hard, the skin cannot be pinched into a fold nor can a muscle be isolated by grasping, so that the impression is conveyed that the skin, fasciæ, and muscles have coalesced into a firm mass. The tibia is likewise thickened at the shaft or the articular ends (pedarthrocace of Malabar), and feels smooth or seems covered with sharp or dull, hard nodules projecting from the interior surface and the anterior edge into the sclerotic mass. Exceptionally, under certain complications, caries or necrosis occurs, and, in some forms of elephantiasis of the upper extremity, pressure atrophy of the bone.

Subjectively, elephantiasis cruris causes trouble only by interference with function, which is due not only to the absolute weight of the limb and the stiffness of the skin, but also to the concomitant degeneration of the muscles. Pain occurs only during inflammatory processes and in consequence of complications.

Most, and particularly the excessive, cases of elephantiasis of the

leg are unilateral ; certain forms also attack both legs. The upper extremity is but rarely attacked by elephantiasis arabum (in consequence of syphilitic or lupous inflammation), and then is very strangely distorted.

Elephantiasis genitalium (*i.e.*, scroti, penis, labiorum pudendorum et clitoridis) occurs only sporadically in our country, and then is generally moderate in degree ; but it appears in numerous and excessive forms in the tropical and subtropical regions mentioned above, as reported by Pruner, Rigler, Reyer, and others. The most enormous development is probably reached by elephantiasis scroti, which transforms the scrotum into a "fleshy," bag-shaped mass reaching to the knee or even to the ground and weighing as much as one hundred and twenty pounds (hernia carnosa, Prosper Alpin, Larrey ; sarcocele of several authors). It springs with a pedicle from the inguinal region and encloses the penis and testicles. A shallow, funnel-shaped groove leading to the penis shows on the tumor where the interior layer of the prepuce is fastened to the frenulum, and permits the passage of the urine from the urethra. Pruner states that he has observed attacks of erysipelas preceding elephantiasis penis, but no such observations are on record with reference to elephantiasis scroti. The process begins with the formation of a firm, doughy nodule at the base of the scrotum ; this, growing and hardening, drags the neighboring skin from all sides—penis, abdomen, and thigh—into the tumor, so that the penis in particular, its integument being drawn downward and forward, disappears completely in the tumor, excepting only the above-mentioned preputio-urethral groove. The surface of the tumor is wrinkled, furrowed, here and there weeping or covered with warts, feels firmly nodular, at some points hard or, on the contrary, soft or gelatinous and elastic. Often vesicles form (lymph scrotum) ; when these burst or are accidentally injured there is an exudation for hours or days of true lymph, *i.e.*, a fluid which coagulates in the air and separates lymph cells—true lymphorrhœa. This could be particularly well seen in a boy of seventeen affected with elephantiasis of the left leg, whom I admitted to my clinic in 1883 and presented before the medical society. On the skin of the scrotum in this patient, which then was not yet elephantiasic, were numerous pin-head- to pea-sized, vesiculous, translucent elevations that proved to be ampullar sacculations of varicose, firm, and cord-like formations palpable in the corium, interpreted as dilated lymph vessels—lymph varices. Some of these ampullæ broke occasionally and allowed lymph to exude for hours and days to the amount of two cubic centimetres every fifteen minutes.

Elephantiasis pudendorum mulierum forms pediculated tumors of the labia majora and minora and the prepuce of the clitoris which

are not quite so colossal, but still sometimes reach down to the knees and are of the same nature.

Sporadically only we meet with elephantiasis of the auricles and the adjoining cheek and scalp, and of the upper eyelids, in the shape of thick, bag-like appendages with broad base or pediculated ; or, as I have observed several times after a chronically relapsing facial erysipelas, in the shape of monstrous enlargement and thickening of the auricles and tumid swelling and hardness of the cheeks and lips.

Still rarer is the elephantiasic thickening of other regions of the skin, where it so deviates from the type described that it would admit of another interpretation. Some of these cases should rather be considered as molluscum fibrosum. Others represent sac-like or tumid thickenings of the skin which have resulted from congenital connective-tissue or vascular moles (nævus lipomatodes, molluscoformis, angiomatodes, papilloma neuroticum) by a continuous growth (elephantiasis congenita, lobar elephantiasis, Esmarch and Kullenkampf). The latter kind of tumors, owing to the large number of plethoric blood vessels, could be well described as elephantiasis telangiectodes (Virchow) or angio-elephantiasis. In other instances, owing to their anatomical relations with the neurilemma of the larger and smaller nerve trunks and branches in the skin, and to the presence of plexiform neuromata, they might be interpreted as fibromata of nerves (Recklinghausen). These sometimes give rise to the most violent neuralgic pains from their first appearance, and have been described by Rokitansky, Virchow, Hecker, Czerny, Köbner, Recklinghausen, and others. One case, which I have observed for many years since I was a student, has been repeatedly subjected to operative interference by Schuh, Salzer, and others. On the left arm of a young man there had formed by gradual growth a sac-like tumor which could be compressed like a sponge with the hand and immediately refilled with blood ; the muscles and bone beneath it were undergoing atrophy. Here, as in Czerny and Köbner's cases, painful neuromata were present from the beginning ("plexiform neuromata," Czerny, Verneuil) and embedded in the spongy tissue of the tumor, for which reason the term elephantiasis neuromatosum (P. Bruns) or neurofibroma (Recklinghausen) would be suitable for it. These, therefore, are pathological formations which in their origin, course, and even anatomical significance differ materially from elephantiasis arabum, but which must be mentioned here because they have generally been reported under a similar name. Esmarch and Kullenkampf, in their comprehensive paper on "The Elephantiasic Formations," have appreciated this genetic difference of the last-mentioned formations from typical elephantiasis arabum.

The *anatomical* examination of elephantiasic hypertrophy furnishes a pretty clear insight into the process underlying it. On incision into an elephantiasic extremity in the advanced stage the flesh creaks, and the whole subcutaneous tissue down to the bone appears as a nearly homogeneous, yellowish-white, fibrous or lardaceous mass, in which the different structures of muscles, nerves, and vessels can be recognized with difficulty. Pressure causes the exudation of a quantity of clear yellowish-white lymph from the cut surface. The cutis appears somewhat condensed, but of nearly normal thickness. The subcutaneous cellular-tissue layer, however, is several times the normal width, and on closer examination appears to be of uneven consistence. Some portions are firm, lustrous white and densely fibrous, almost scirrhous (elephantiasis dura s. scirrhosa), others are soft and gelatinous (elephantiasis mollis s. gelatinosa) and limited by fibrous trabeculæ with a tendinous lustre. Between them are found similarly bordered loculi containing fluid lymph. A like condensation and thickening affect the intermuscular connective tissue, the vascular and nerve sheaths, but the nerves themselves are rarely degenerated. The bones are thickened, sclerosed, and smooth or set with osteophytes; they are occasionally eroded here and there, thinned, necrotic or carious.

On microscopic examination we find that the cutis, excepting the condensation of its fibres and excess of pigment in it and the epidermis, is markedly changed only at the points where warty formations have sprung from it, in an analogous manner to the process in ichthyosis simplex and hystrix or the development of warts. The scirrhous masses of the subcutaneous cellular tissue consist of a thick felt of connective-tissue trabeculæ deficient in plasma and cells. The soft masses consist of a young, juicy connective tissue enclosing numerous round and branched cells (connective-tissue corpuscles). The cutaneous glands are here and there preserved or crowded apart, as are the fat lobes; or they are atrophic and the endothelium of the sweat glands shows hyaline swelling (Gay). The muscles are decolorized and in process of fatty degeneration. The arteries and veins, greatly dilated, are enclosed in a thickened adventitia; the latter vessels are also thrombotic here and there. The lymph vessels, however, are dilated throughout, as are the interstitial lymph spaces as far as the extreme point of the papillæ (Teichmann). They are filled with lymph, and here and there contain sacculated ampullæ lined with endothelium (Czerny, Kaposi).

The findings are analogous in elephantiasis scroti. Some special characteristics are to be noted in elephantiasis telangiectodes, such as ectasia and new formation of blood vessels and blood spaces with thick trabeculæ, as well as, in some cases, neuromata (Czerny).

If we consider the course of development of elephantiasis arabum

in comparison with the results of the anatomical examination, it is not difficult to understand that the stagnant œdema, renewed in consequence of the chronic inflammatory attacks, forms the basis for the connective-tissue hypertrophy and the subsequent increase of volume of the affected part, as well as the other tissue alterations. But it is not every variety of œdema that leads in like manner and with the same celerity to hypertrophy of the connective tissue. Serous œdema and that due to congestion in the smallest veins or larger venous trunks, or that caused by reduced renal excretion, are certainly not apt to produce it. Cohnheim, Ranvier, Lassar, Sotnitschewsky, and others have made instructive experiments in this direction, which, though not clear throughout in their results, mark the difference existing between imbibition by the tissue of inflammatory congestive exudation (derived from the vessels under inflammation) and that forced mechanically from normal vessels. This statement, however, does not apply absolutely, for I have seen cases enough of congestive œdema in local asphyxia of the upper and lower extremities in which connective-tissue pachydermia developed in the course of years without any inflammatory symptoms. In elephantiasis arabum, however, we have to deal exclusively with a so-called lymphatic œdema (Virchow)—*i.e.*, a fluid rich in white blood corpuscles which accumulates in the interstices of the tissues. Such a fluid, according to microscopic demonstration (Young), leads directly to the new formation of connective tissue ; the colorless cells send out prolongations which intertwine largely and may also indirectly stimulate the normal (fixed) connective-tissue elements to hyperplasia through the abundance of nutritive fluid.

I admit, however, that I have seen several cases of colossal unilateral and bilateral elephantiasis of the lower extremities and nates which developed in virginal and young females and in otherwise healthy males in the course of two or three years without any inflammation. Such cases are etiologically quite obscure.

The known *causes* of elephantiasis arabum elucidate these conditions still further, in that the affection occurs, as a rule, at points where the local relations favor a relapsing inflammation and stagnation of an inflammatory œdema. Thus, in the case of the leg, chronic eczema, ulcers of the foot, cicatrices, bone callus, chronic neoplasms such as gummous syphilis and especially lupus, cicatricial contraction of the inguinal glands, probably also contracting exudations and tumors within the pelvis ; for in the latter cases and after the puerperium I have observed intense elephantiasis of the leg develop in a short time.

In the same category belong also the œdematous swelling occurring in the domain of paralyzed or irritated nerves (as by cicatrices), which is associated with a red skin of a satiny lustre ("glossy" skin),

cyanosis and lowered temperature, pain or on the contrary diminished sensibility, and may lead to pachydermia (pachydermia neurotica), though in other cases it ends in tissue atrophy; and the pachydermia occasionally resulting from chronic cyanosis, local asphyxia, which I have repeatedly observed and therefore am forced to maintain in spite of the doubt expressed by Esmarch.

Still, as stated above, it is difficult to ascertain the cause of many cases of elephantiasis of the extremities, and especially of the cases of elephantiasis of the genitals and extremities occurring in the Orient and the tropics, or even sporadically in our climate. In such instances, indeed, we hide our ignorance by assuming a racial or individual disposition, or one due to climatic conditions. Hecker and Czerny's case of elephantiasis telangiectodes occurred in three generations of one family—*i.e.*, by heredity. But this, as mentioned before, was not the ordinary form of elephantiasis arabum. We know little concerning the relation of the lymph varices and the lymphorrhagia to the so-called endemic or tropical elephantiasis arabum of the extremities, and especially of the genitals. But stasis in the lymph current has always been pleaded as the essential cause of elephantiasis. Thus for these forms, too, a chronic relapsing erysipelas with consecutive lymph stasis has been given as the cause (Rigler, Rayer, Pruner, and others), but in many cases this was absent, and particularly is not reported in elephantiasis penis. Lymph varices and vesiculous prominences with lymphorrhagia, as in the case observed by me, have often been seen in elephantiasis scroti orientalis.

It has also been claimed that swelling of the lymphatic glands, especially those of the leg, is the primary feature, and the lymph stasis in the peripheral region, followed by connective-tissue hypertrophy, is a secondary characteristic of the disease. For this reason it has been termed the glandular disease of Barbados (Hendy and Rollo). But in many cases, as in my own, there is no notable swelling of the glands.

In recent years the etiology of elephantiasis arabum orientalis has been given a more positive direction by bringing the filaria sanguinis hominis (Lewis) into a causal connection with elephantiasis arabum. This nematode, whose embryos were first found by Wucherer (in Bahia, 1866) in the urine in chyluria, and later by Lewis (in Calcutta) also in the blood of chyluric patients, has been demonstrated as a sexually mature worm in the tissue juice of the elephantiasic scrotum, in lymph abscesses of the arm, and in lymph varices by the latter author and also by Manson, Bancroft (Australia), Da Silva Araujo (Brazil), Felicio dos Santos, Cobbold, and Mackenzie. On the strength of this fact and deductions from experiments, the merit of which belongs, next to Lewis, especially to Manson, the latter has

advanced the opinion that the filaria sanguinis causes sometimes chyluria, sometimes a chylous diarrhœa or chylous hydrocele, or finally elephantiasis and lymphorrhœa; that all these affections, which may mutually vicariate, form an inseparable symptom-complex of filariosis.

The developmental cycle which occurs has been demonstrated experimentally by Manson in 1875. It had been discovered that the embryos of the filaria circulate in the blood only at night during sleep, when they can be demonstrated under the microscope; also that the mosquitoes are the intermediate hosts in which the embryos develop. In the stomach of mosquitoes which had fed on a filariotic patient the developmental phases of the embryos were followed until they had become almost sexually mature filariæ. The conclusion was drawn that the mosquitoes filled with filariæ after three days fall dying into the water, in which the liberated filariæ become sexually mature; they are ingested with the water in drinking, migrate from the human stomach into the tissues, occlude the lymph vessels, and, according to the region affected, cause chyluria or elephantiasis or lymphorrhœa; they liberate their embryos, which, circulating in the blood, are again ingested by the mosquitoes. The facts thus far presented, however, have been unable to secure a general acceptance of Manson's theory.

In my case mentioned above, which exactly corresponded with the tropical forms, the lymph and blood were carefully examined, but no filaria or other embryos could be discovered; nor could they be found in the case reported by Nieder, from the Freiburg medical clinic, of a girl aged fourteen suffering with lymph varices and lymphorrhœa of the labia.

For the *diagnosis* of elephantiasis arabum it will be sufficient to remember the above restricted definition of the disease—*i.e.*, excluding the forms of elephantiasis telangiectodes and fibroneuromata resulting from congenital anomalies, and taking into consideration the described symptoms. The *prognosis* is relatively favorable only during the first stages of the affection, while œdema alone is present and the patient is able or the treatment competent to obviate the factor causing inflammation. It is only the œdema, not the fully formed hypertrophic connective tissue, which can be made to disappear.

In the *treatment* the general measures must carry out this idea. In elephantiasis of the leg, in the first place any inflammatory symptoms present, and whenever they recur, must be managed according to general rules. Horizontal position of the extremity during the local rise of temperature and painfulness; the application of cold, later warm, so-called "dissipating" fomentations and lukewarm baths, are most appropriate. Complicating ulcers, eczema, and warty

outgrowths are treated by the well-known measures (ointments, lotions, caustics, surgical manipulations) ; adhering scales and crusts are softened and detached, and the patients are instructed to avoid everything that might excite renewed inflammation and increase the œdema. The next object is to endeavor to reduce the volume of the extremity by absorption of the œdematous infiltration. Methodical frictions with mercurial ointment, unguentum juniperi, lanolin, vaseline, with or without the addition of resorptive drugs, skilful massage combined with continual lukewarm fomentations and baths and horizontal position of the leg, often materially lessen in a few days the hardness and circumference of the limb.

An additional effect is produced by a pressure bandage, which, however, can only be applied when no acute inflammatory symptoms are present. A flannel, rubber, or still better a cotton roller, previously dipped in water, is wound firmly and as evenly as possible from the toes upward beyond the elephantiasic portion ; as the circumference of the limb rapidly diminishes under it, it is renewed two or three times in the first few days. After the œdema has been dissipated and absorbed as far as possible, nothing remains but the increased volume due to connective-tissue hypertrophy. Many attempts have been made to limit this by diminishing the afflux of blood through methodical compression of the femoral artery or (after Carnochan, 1851) by ligating this vessel and the iliac artery or resection of the sciatic. Aside from the cases lost by gangrene and pyæmia, no more improvement was secured in the other cases than would have resulted from the horizontal position, necessitated for some weeks after such operations, through the lessened œdema. Such being the case, one would be inclined in pronounced elephantiasis of the leg to recommend rather amputation, which would at once deliver the patient from the useless and cumbersome limb and enable him to employ a suitable prothesis. Unfortunately even this result cannot be relied upon, as most patients thus far have died in consequence of the amputation. This operation is altogether out of the question when the disease implicates the nates. Moncorvo and Silva Araujo claimed brilliant results from the electrolytic action of the constant current. These methods have entirely failed in my hands. On the contrary, owing to the unfavorable circulatory conditions in the affected tissues, I have not rarely observed renewed inflammation, erysipelas, and even gangrene from their use.

Elephantiasis of the genitals and other regions can be cured only by operation, and since the time of Gaëtani-Bey the operative methods for elephantiasis scroti have been so perfected that even the most colossal tumors can be effectually removed. The excision requires care in order to leave flaps of sufficient size for the penis and testicles.

In connection with the affection last discussed I wish to call attention to the peculiar disease termed

MYXŒDEMA

after Ord, of which I saw several pronounced examples in London in the year 1881, but none here in Vienna. Thus far the disease has been described mainly by English and French physicians, first by William Gull, who in 1873 called the affection "cretinoid œdema," owing to the intellectual depression resembling cretinism presented by the patients; then by Ord in 1877, who was the first to furnish the interesting anatomical proof that the apparent œdema of the skin and other tissues consisted in a deposit and proliferation of mucoid material, and therefore proposed the name myxœdema as the most significant; later by Charcot, who introduced the term "cachexie pachydermique" for the malady (1879). Other observers (Savage, Hadden, Ballet, Thaon, Bourneville and d'Ollier, Ingliss Hammond, Greenfield) have materially amplified the symptomatology of the disease.

It has been observed chiefly in women, rarely in men, and manifests itself by œdematoid swelling, thickening, and hardening of different portions of the skin, mainly the face, but also the trunk and extremities, the tongue and soft palate. The eyelids, nose, and lips appear turgid, yet hard, not to be indented as in œdema; the skin is smooth, dry, with a dull lustre, often waxy. The facial expression becomes imbecile, idiotic. A similar swelling and thickening affects the fingers and the entire hands, which are clumsily disfigured and hampered in their movements through the thickening. The same is true of the skin around the joints, that of the trunk in various regions, and all this in the most irregular form and extent. The thickness of the tongue renders speech labored and indistinct.

This condition is associated with dulling of the intellect and the special senses, particularly the tactile, gustatory, and olfactory; while hearing and vision have thus far been found intact. The patients take a long time to perceive; they reply and move very slowly. Retarded digestion, constipation, failure of the general physical and mental powers, of the working and thinking capacity, are gradually superadded. Experience so far has shown that the disease always terminates fatally by complications from internal organs, the heart, kidneys, at times with maniacal attacks or general marasmus.

Ord demonstrated as cause for the tissue infiltration and degeneration a peculiar gelatinous mass which does not escape after incision and presents all the characteristics of mucoid material. This substance, deposited and continually increasing in the tissues,

causing in the beginning swelling and thickening of the skin, leads later on, through pressure, to an atrophy of the parenchyma cells, the connective tissue, the thyroid gland (Ord, Hard) and other glands, the vessels, and, not only by mechanical interference but also by pressure atrophy of the nervous elements, to functional disturbances, reduction of cutaneous sensation, secretion, and nutrition. The same mucoid infiltration affects also the internal organs, the liver, kidneys, muscles, and thus explains their disturbed functions. This is true especially of the brain and spinal cord, so that the early depression of mental activity and muscular function is easily understood.

Since the first observations of Ord many attempts have been made to explain this enigmatical disease, partly on the strength of additional cases, partly from pathologico-anatomical findings and experiments on animals.

In the first place, many authors have been inclined to find in myxœdema an analogue to the so-called cachexia strumipriva, first observed by Reverdin and later by Kocher after the extirpation of goitre, because in the latter case likewise circumscribed œdema and dulness were observed (myxœdème opératoire), and in myxœdema many thought they could detect absence or atrophy of the thyroid gland. This view was taken by the English commission charged with the study of myxœdema (Ord, Semon, Horsley, and others), by H. Hun (basing his opinion on a collection of one hundred and fifty-four cases), by Köhler, Garré, Mosler, and Schnied, and others. But various observers were unable, on the one hand, to demonstrate atrophy of the thyroid gland in myxœdema, and, on the other, a surgeon like Billroth had never noticed the occurrence of myxœdema after the extirpation of goitre.

Horsley, believing in the analogy between myxœdema and cachexia strumipriva, and basing his theory on Von Eiselsberg's experiments with tetany after the extirpation of goitre, has proposed to stitch the thyroid gland from the sheep under the skin of the abdomen in myxœdema. In fact, Bircher claims to have observed improvement of the myxœdema and the concomitant "cretinism" in two cases thus treated, and Wallace Beatty makes the same claim for subcutaneous glycerin injections of thyroid extract (combined with massage); the idea being that the secretion from the sheep's thyroid thus supplied destroys the mucoid material present in the tissues.

All these theoretical and experimental results have been disputed (by Munk, Drobnick, and others), and the question of the nature and cause of myxœdema is still unsettled. Neither has any light been derived from the histological findings in the myxœdematous skin. H. Hun states that he found the connective-tissue spaces in the corium crowded apart by a fluid which is not mucin, together with

a general atheromatous endarteritis. On the other hand, P. Grawitz in Mosler's case and Caspary found no notable changes in the cutaneous vessels and only some œdema of the tissue. Some authors accept the theory of a general nutritive depression.

The known facts, therefore, although very instructive, do not suffice to decide whether the pathological alteration affects the various organs and tissues, cutaneous and nervous centres, in an irregular manner without any internal causal connection, or whether, as Charcot seems to believe, the disturbance of the nerve centres is primary and the accompanying disease of the skin and other organs is to be looked upon as the result, as it were, of a nutritive and functional alteration brought about by the trophic centre.

CIRCUMSCRIBED CONNECTIVE-TISSUE HYPERTROPHIES.

The lesions resulting from circumscribed connective-tissue hypertrophy of the skin appear as red, warty, single or many lobed, cauliflower-like, slightly sensitive excrescences of the skin which project moderately up to a few centimetres; they are here and there dry, but usually furnish a thin, sticky secretion which becomes rapidly offensive. They may occupy limited regions or large surfaces, and in their external appearance and anatomical structure represent enormously enlarged papillæ—*i.e.*, *papillomata*. Their anatomical composition, therefore, corresponds exactly to that of single and aggregated warts (Fig. 40), the papillary outgrowths in ichthyosis hystrix (Fig. 41), and dermatitis papillaris capillitii (Fig. 3[illegible]); in other words, they are simple or dendritic branching connective-tissue cones, the axis of whose trunk and twigs is pierced by a greatly dilated and correspondingly anastomosing capillary vessel, while their surface is covered with a proliferating rete. When the outgrowths proliferate rapidly, their epidermis, being imperfectly cornified, is detached as a dirty detritus or cast off by the formation of vesicles. In that case the rete, partly weeping, partly encrusted, is exposed and the connective-tissue layer is juicy and rich in cells. In the stationary forms a thick horny covering develops as in ichthyosis hystrix, and the connective-tissue framework is coarsely fibrous, close-meshed, and deficient in cells, sometimes even scirrhous.

Aside from the congenital forms, which are to be considered as papillomatous nævi, these anatomically similar forms belong to clinically widely differing processes. One of them has been introduced into pathology by Sauvages (1786) as *frambœsia*, which was stated to represent a disease endemic in Western Africa and the West Indies (called "pian" in the former, "yaws" in the latter country), characterized by the development of mulberry- and raspberry-like weeping excrescences, and said by some to be of syphilitic, by others of idiopathic origin. Alibert substituted for frambœsia the name

mycosis (frambœsioides et syphiloides, also fungoides) and later pian, and looked upon the affection as syphilitic. Subsequent experiences, however, have shown that frambœsia had been misapplied as a collective term for very different syphilitic or various other chronic infiltrating and ulcerative processes of the skin, all of them, indeed, generally associated with papillary formations. A similar confusion existed formerly with reference to diseases said to be endemic in different countries, and known by the terms "sivvens" in Scotland, "radesyge" in Norway, "falcadina," "skurljevo" in Istria, etc., but which experienced physicians at once recognized, not as strange, but as belonging to syphilitic, scrofulous, lupous, and other well-known processes. As the pathological basis for these titles vanished, the names themselves likewise disappeared from our pathology. To give an appropriate example for frambœsia. Alibert describes and figures as a prototype of "pian ruboid" (according to the importance attached by this author in general to frambœsia and syphilis) that interesting affection of the nucha and occiput which my observations and microscopical examinations (see page 382) show to be an idiopathic, non-syphilitic, chronic inflammatory process which leads to the formation of papillomata, and later to scirrhous connective-tissue hypertrophy.

In the majority of the cases of the kind we have to deal with secondary formations, with exuberant outgrowths of the cutaneous papillæ or (on suppurating raw surfaces) of the granulations on a portion of skin which is in a state of chronic inflammation or infiltrated with neoplasms or suppurating. To them belong the profuse proliferations observed in sycosis, eczema, pemphigus, ulcerating syphilide of the hairy scalp, above carious bone, upon and alongside chronic ulcers of the foot—vegetations described by me under the head of dermatitis diabetica and as papilloma diabetica; and, most frequently of all, the tumid, warty excrescences which develop on patches of lupus and often persist after it. It is proper, therefore, to designate such forms according to the fundamental process, perhaps with the addition of an adjective expressing the complication, thus: lupus papillaris s. frambœsioides, syphilis vegetans s. frambœsioides. Sometimes after the fundamental process has run its course the papillary excrescences remain behind as firm connective-tissue tumors, liable to interstitial inflammation and the formation of abscesses. Such formations of course may at times be difficult to diagnosticate, but giving them a new name ("inflammatory papilloma of the skin," Roser, Weil) will not help the matter. In most cases, however, the original affection (caries, lupus, syphilis) can be traced by concomitant circumstances or established beyond a doubt.

Still, numerous modern authors, Milroy, Imray, Bowerbank, Nicholls, T. Fox, Duhring, Hirsch, and Pontoppidan, partly, like the

latter, on the strength of personal observations, maintain the existence of an endemic contagious disease named yaws. This is said to be peculiar to many tropical and subtropical regions in Africa, the East and West Indies, and characterized by the development of mulberry-like nodules and tumors which heal spontaneously after a course of several months or one to two years. Pontoppidan, on the strength of histological examinations, even looks upon them as proliferations springing from an inflammatory base.

A similar description is given of a disease endemic in Peru, known as "veruga," whose chief characteristic is a tendency to hæmorrhages. I can express no personal opinion regarding all these forms.

An analogous disease, framboesiform excrescences developing on an inflammatory base, discussed originally by Bazin, Köbner, Wegscheider, L. Mayer, and more recently by Geber, Vidal, myself, and many others, which is associated with an invariably fatal process, will be considered more fully hereafter under the name of mycosis fungoides.

The consecutive papillary forms, however, possess merely an importance corresponding to their source and extent. Should they fail to undergo involution with the fundamental process (syphilis, lupus, etc) and by the measures effective against the latter—*e.g.*, mercurial plaster or general antisyphilitic treatment—they may be removed by the methods employed for warts (caustics, the curette, or extirpation).

CLASS VII.

ATROPHIES.

DISEASES OF SKIN CHARACTERIZED BY TISSUE ATROPHY.

LECTURE XXXV.

GENERAL REMARKS ON ATROPHY—PIGMENT ATROPHY OF THE EPIDERMIS: CONGENITAL (ALBINISM), ACQUIRED (VITILIGO)—PIGMENT DEFECT OF THE HAIR, CONGENITAL AND ACQUIRED—CANITIES PRÆMATURA, SENILIS.

ATROPHY, simple or degenerative loss of tissue, and the condition which is equivalent to it in its results—namely, absence or defective development of some elements of the cutis—represent pathological states which in part are the direct opposite of the hypertrophies, and in part form peculiar diseases. Like hypertrophy, atrophy also affects either exclusively or largely some elements of the skin, pigment, hairs, nails, or else the connective-tissue framework of the cutis, together with the vessels and glands. Accordingly we divide the diseases of this class into the following groups : pigment atrophy, atrophy of the hairs, atrophy of the nails, and atrophy of the skin proper.

PIGMENT ATROPHY,

achromatia, leucopathia, means the loss of the yellowish-brown, dark-brown, or black color which normally belongs to the horny structures of the skin—*i.e.*, the mucous layer and the hairs—these then appearing white and gray, decolored. Both conditions sometimes coexist, but usually they occur separately.

ABSENCE OF PIGMENT OF THE EPIDERMIS

causes a lustrous or dull white appearance of the skin—leucoderma, achromasia—with rosy translucence from the blood injection. The condition is either congenital (albinism) or acquired (vitiligo).

Leucoderma congenitale affects either the whole of the common integument—albinismus universalis—or merely certain regions—albinismus partialis. The former constitutes the peculiarity of the so-called albinos, in whom the pigment is lacking not only in the

skin but also in the hair, iris, and choroid. For this reason the skin looks very white with a rosy translucence and unusually delicate; the hair yellowish or dull white and silky; but the iris and pupil (owing to the reflection of the light from the fundus of the eye) appear red, and there is extreme photophobia with nystagmus. The cause of this defective development, which persists through life, is obscure. We know that normally pigmented parents may procreate albinos, but no experience is recorded as to whether the anomaly is transmitted from albino parents to their descendants. Among the negro races (in whom, according to Beigel, a half-whitening of the dark color, semi-albinism, occasionally occurs) albinism is more fre-

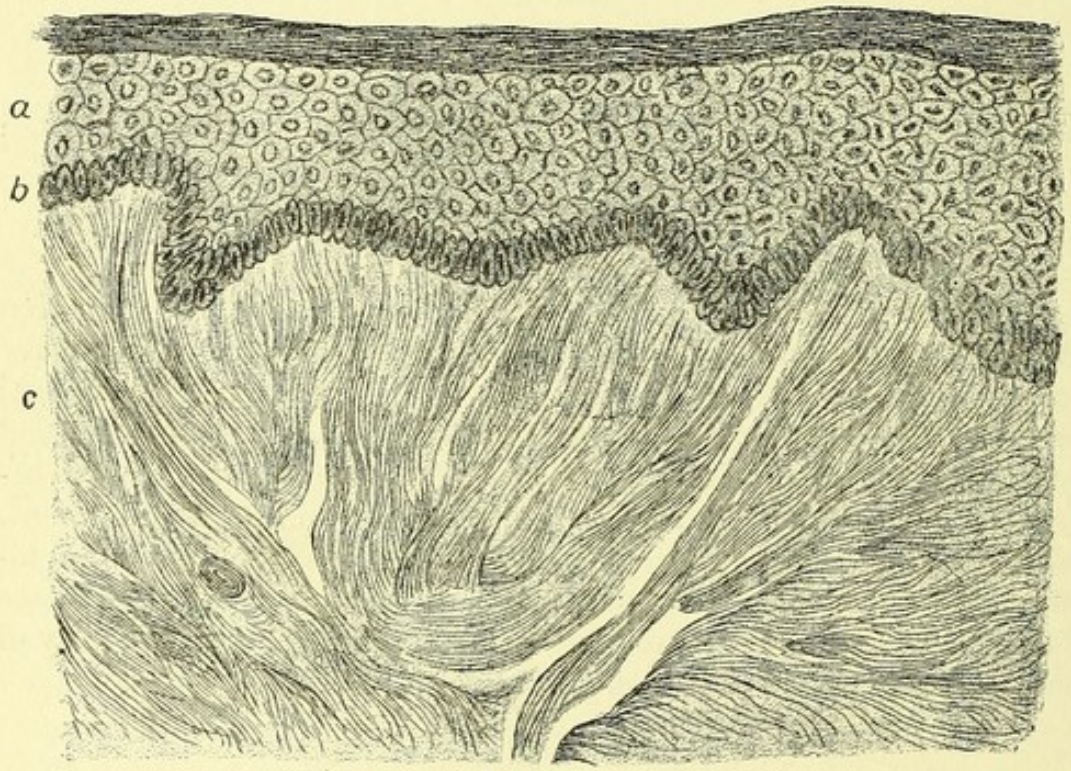

FIG. 42.—SECTION OF THE SKIN OF A NEGRO.

b, brown granular pigment of the deepest rete cells; *a*, diffuse yellowish pigment of the prickle-cell layer; *c*, corium.

quent than in the light-colored races, just as the former are more subject to pigment defects in general, as for instance to

Albinismus partialis, which represents an analogy to the piebald development of animals; some regions of the skin, chiefly of the scalp and genitals, appearing from birth white and non-pigmented, in spots or streaks. Such spotted individuals are known as piebald negroes, nègres mouchetés, nègres pies, piedes nègros Not uncommonly, too, the hairs growing within such spots remain white (poliosis). Though usually irregular, the albinotic spots are sometimes arranged symmetrically or corresponding to the course of the

peripheral nerves in a manner identical with certain pigment and verrucose nævi; occasionally they accompany the latter, so that white and dark streaks run side by side. Altogether pigment atrophy and pigment hypertrophy frequently are, as it were, complementary in their association, pigment atrophy often originating in a hyperpigmentosis—for instance, a pigment mole. If we remember that the skin of the negro *per se* shows an excessive amount of pigment in the rete cells (Fig. 42), we find that their tendency to achromasia has its analogue in the frequent occurrence of like abnormalities (vitiligo) on and about pigment nævi in the Caucasian race.

Partial albinism, like albinismus universalis, is generally stationary, but alters in some cases by the advance of the pigment atrophy, and now and then proves hereditary.

Leucoderma acquisitum arises either idiopathically or consecutively and symptomatically. The idiopathic form, *vitiligo*, achroma vitiligo, likewise is more frequent among negroes, but may be seen often enough in the Caucasian race. Without any known cause, without any perceptible local disturbance of sensation or nutrition, there appear on one or more regions of the body pale (non-pigmented) spots ranging in size from a cent to a quarter, while the immediately adjoining skin is colored dark brown. It seems as if the pigment had been displaced or transferred from these centres toward their periphery. The hairs, too, generally fade within the achromatic spots. In the course of months and years the loss of color advances steadily in like manner, the white spots becoming large, round or oval discs with convex borders, enclosed by the more darkly pigmented neighboring skin with concave margins. In time the contrasting effect is the reverse to the eye. While at first the small white discs in the normal and more darkly pigmented skin are most striking—the face, for instance, appearing piebald, the fingers encircled with white and brown rings—later, when the leucopathic surfaces have become extensive, it is the darkly pigmented interspaces that are more striking, so that the inexperienced believe the white spots to be normal and the dark regions to be affected. According to one of my observations on a man aged fifty-six, the process after many decades may finally extend over nearly the whole body; in the patient referred to all the skin was decolored excepting a few dark streaks of pigment on the most peripheral parts.

Otherwise the leucopathic skin is in no wise altered, its functions and sensations are normal, and only at times it perspires less (after the exhibition of pilocarpine).

The *diagnosis* of vitiligo is easy in view of its striking symptoms. If the disease is occasionally mistaken for leprosy in localities where that affection is present, this is due partly to the fact that in the latter, too, white and dark dyschromasis occur, and partly to the old

theory which identified with lepra the "zaraath" of the Bible, in which the region of the skin and the hair within it had become white. The disease here discussed does not furnish a good prognosis, inasmuch as it cannot be cured or limited; but, on the other hand, aside from the disfigurement it may cause, it has not the least influence on the general health or the other functions of the skin.

The *anatomical* change in vitiligo consists solely in the absence of the pigment granules in the deep rete cells corresponding to the achromatic spots, while on the adjoining, more highly pigmented regions the rete, on the contrary, is more abundantly supplied with pigment. The few migratory cells loaded with pigment which are present in the corium (Fig. 34) contribute little or nothing to the coloring of the skin. Leloir and Chabrier have discovered atrophy of the subdermal nerve fibres corresponding to the non-pigmented spots.

In some cases a general disturbance of innervation—for instance, after exhausting diseases—has been cited as the *cause* of the vitiligo. The large majority of cases, however, occur in healthy persons of middle age, and for most of the cases of vitiligo we are absolutely at a loss to suggest a plausible cause. Frequently local factors may furnish the exciting cause. Among these I would include any kind of displacement of the normal distribution of the pigment and any stimulus to its more rapid metabolism. In this way, pigment atrophy results either directly or indirectly through pigment hypertrophy. Thus, as is well known, vitiligo frequently springs from pigment moles, and stationary or advancing decolorations arise from the pressure of bandages or start from the cicatrices of burns and ulcers. In the latter case the pigment is drawn into the current of absorption peculiar to the process of involution of cicatrices, in the same way as other tissue elements (infiltrating cells, connective-tissue corpuscles). Whether the migratory cells by their carrying of pigment play an intermediate part in this displacement of the pigment and the extent to which they do so, or whether they, perhaps, merely indicate the direction of the centripetal plasma current which leads to resorption, may be gathered from the findings of Riehl in leucoderma syphiliticum (see page 421 and Fig. 34). In the acquired achromasia described above, the absorption of the pigment likewise starts from a richly pigmented cell infiltration. Here, too, there is an implication of the rete pigment in the resorptive current of pathological deposits of tissue. Ehrmann, as stated above, basing his theory on the same anatomical findings, interprets the intimate process of the pigment transportation somewhat differently, as does Halpern.

The concomitant and consecutive forms of vitiligo, representing patchwork of leucopathic and pigmented spots, belong to an analogous causative category; they occur in xeroderma, scleroderma, and lepra. The same may be said of the achromasia remaining after

the absorption of inflammatory and neoplastic infiltrations and their pigment remnants. after furuncles, variola, lupus, syphilitic papules, the so-called cicatrices of pregnancy, etc. Still, it has never been decided why in such cases the pigment atrophy sometimes advances without limit, or even affects other regions of the skin as in idiopathic vitiligo. It is clear, however, that a permanent loss of pigment occurs at the points affected by the last-named processes over the extent of the local lesion ; for with the atrophy of the papillæ and the rete there is a loss of the tissue which produces and preserves the pigment.

The direct *treatment* of the leucopathias of any kind has thus far proved ineffectual. While we are able to cause, as by cantharides, a deeper coloring of the spots of vitiligo, this does not correspond to the normal tint of the skin, and moreover disappears again. On the other hand, we can remove the piebald appearance of the skin, the contrasting effect of the light and dark spots, by treating the pigmented—*i.e.*, the really normal—regions in the same way as we do pigment spots (page 424). Such measures may be desirable in the early stages of vitiligo of the face and hands, and of partial achroma. Internal remedies, such as arsenic and iron, are without the least influence on the process of vitiligo.

ATROPHY OF THE HAIR PIGMENT

manifests itself by the hair turning gray—*canities*, *poliosis*. The hair appears silver gray or white. The condition may be congenital, both general and partial, corresponding to albinism ; but we may also find from birth a partial poliosis—a whisp of white or gray hairs in the midst of dark-colored scalp hair, without associated decoloration of the underlying skin.

Occurring abnormally, as acquired in extrauterine life, there is the premature whitening, *canities præmatura*, which may affect all the hair of the scalp and beard or be a partial poliosis, a consequence of individual or in many cases of congenital disposition, or of intense physical and mental suffering, rarely observed also in the new growth of hair after its loss occurring in typhoid fever, erysipelas, zoster, alopecia areata, etc. Canities præmatura occasionally disappears through a new growth of pigmented hairs. Usually, however, it is permanent, like the physiological whitening due to age—*canities senilis*—in which, as a rule, the gray hairs appear first in the temporal regions, later elsewhere about the scalp and beard, until gradually, *i e.*, in the course of several years, most or all of the hair of the regions named and that of the body have become gray.

All these forms of blanching of the hair have the same *anatomical* basis. The normal pigmentation of the hair is due (Fig. 43) to the

deposit of dark-brown or yellowish-brown pigment granules in and between the cells of the cortex of the hair, and the shade of color (black, brown, blonde, red) depends upon the density and distribution of the pigment. The matrix for the formation of the pigment is constituted by the papilla (Fig. 44, P), as the pigment of the epidermis is formed from the skin papillæ; and the constancy in the color of each hair depends upon the fact that its papilla, or the basal cells above the latter, are constantly reproducing pigment. The young cells of the hair first contain the pigment; they carry it along with them, inasmuch as they are pushed forward with the

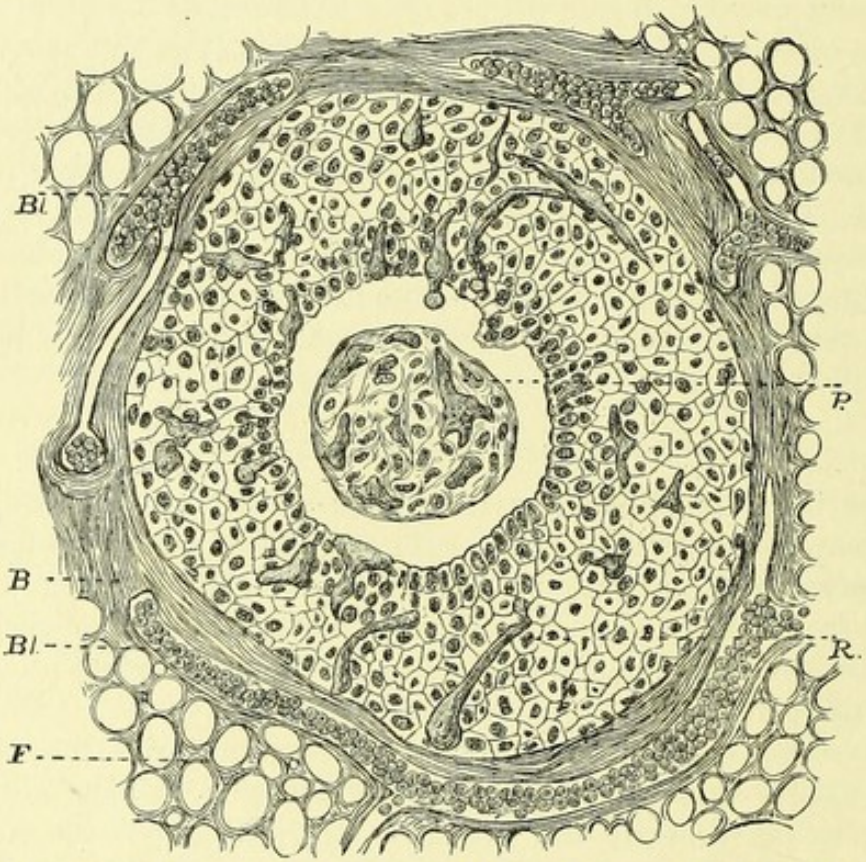

FIG. 43.—TRANSVERSE SECTION OF THE ROOT OF THE HAIR NEAR THE POINT OF THE PAPILLA.

P, papilla; B, hair follicle; R, root part of the hair; F, lobule of fat; Bl, blood vessels surrounding the hair follicle. In the abundantly cellular stroma of the papillæ, between the matrix (or root) cells of the hair, are numerous dark-brown, pigmented, migratory cells, originating from the vessels of the papilla. (After Riehl.)

growth of the hair, are ranged alongside of the cortical substance, and finally become cornified. In the congenital forms of poliosis the pigment-producing function is lacking in the hair papillæ (also in the cutaneous papillæ in albinism) or in the basal rete cells. When the hair turns gray at a later period this property is lost suddenly (in vitiligo) or gradually, either from a general depression of nutrition or innervation (after disease, grief, excessive labor), from local destruction of the papillæ (in cicatrices), or from senile atrophy of the

tissues. From his studies on the comparative anatomy of the frog's skin, Ehrmann has come to the conclusion that the pigment cells of the papillæ and corium do not emigrate as such among the cells of the rete and there deposit their pigment. He believes that similar cells are found among the latter and also within the epidermic layer of the hair, and that these receive pigment from the analogous cells of the papillæ by means of protoplasmic movement, not as the result of the circulation of the tissue juices. Hence the pigmented multipolar cells in the rete and the hair are autochthonous, not migratory structures. As he found pigmented cells in the papillæ of those hairs whose shafts were already devoid of pigment (gray hair), he

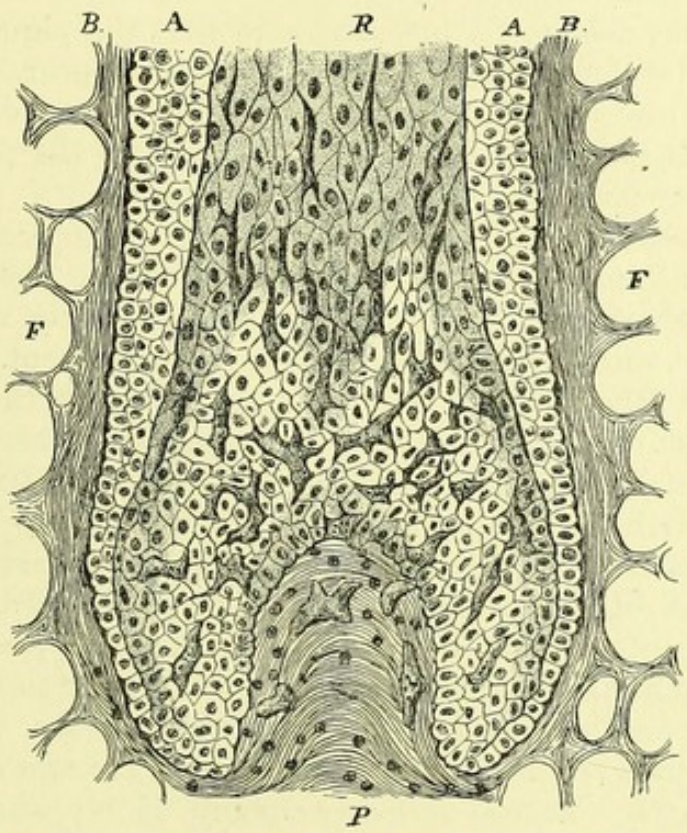

FIG. 44.—LONGITUDINAL SECTION OF THE ROOT AND PAPILLA OF A HAIR.

P, papilla; B, sheath of hair; R, root of hair; F, fat cells; Bl, blood vessels of the sheath. Pigment cells, thickly studded, and consisting of a granular protoplasm, in the papilla and matrix of the hair; sparsely scattered, and poor in protoplasm, in the interstices of the horny, cortical cells of the shaft.

believes that the grayness depends, not upon the cessation of the formation of pigment by the papillæ, but on the cessation of the absorption of pigment by the large protoplasmic cells of the hair. Ehrmann assumes a similar condition for the loss of pigment in vitiligo, but the anatomical demonstration of this statement is wanting.

Whichever interpretation is more correct, the grayness of the hair depends, not upon the blanching of already pigmented hair, but upon the after-growth of hair which is deficient and finally entirely

devoid of pigment. In the grayness of old age we always find hairs whose tip is still dark while the basal portion is already deficient in pigment.

Before the hairs are entirely gray certain follicles produce hairs which show brown and gray rings—a proof that, prior to the complete cessation of the formation of pigment, the papillæ and rete were capable of producing pigment at times. As the hairs grow gray only in the after-growth—*i.e.*, in a period proportional to their physiological growth (a few weeks)—the cases reported even by scientific men (Landois. Brown-Séquard, Raymond, Michelson) of grayness developing suddenly, "over-night," in shipwrecked individuals, those condemned to death, etc., must be based on errors of observation. It is physiologically inconceivable that the pigment granules in the fully developed hair should suddenly disappear. Nor can we believe that, under the influence of fright, etc., gases develop in the hair, and that these bubbles of gas or air conceal the pigment, inasmuch as many normally colored hairs contain air.

Nevertheless Waldeyer maintains that a certain proportion of air in the hair will make the latter appear gray as the result of a complete reflection of light produced thereby. This does not appear to me to be true, and has not been proven by experiment. Such hairs would assume their normal color after expression of the air, but this is not the case. Lesser, who described a case of "ringed hair," and found the thickened, spindle-shaped parts lighter than the narrow ones, also attributes the lighter color to the presence of air in the cortical substance. Reinhard, who found in an insane patient a change in the color of the hair from dark golden red to yellowish blonde, corresponding to the periodical changes of psychical excitement and quiet, also attributes this change of color to the change in the amount of air as the result of nervous phenomena.

Räuber observed a periodically recurring change of hair in an epileptic. After a fit, the normally smooth, glossy, and dark-blonde hair of the scalp became coarse and matted for several days ; it was devoid of gloss and had a fox-red color ; some of the hairs became nodulated and brittle as in trichorrhexis nodosa. Räuber explains the matting and curling of the hair by a spasm of the arrectores muscles, and the change in the nutrition and color of the hairs by a spasm of the papillary vessels.

I have no doubt that physical and psychical changes may produce considerable change in the character of the hair of the scalp and beard, and even grayness, within a short time, but I do not believe that this occurs suddenly and in fully developed hairs.

The *treatment* of gray hairs can only attempt to conceal the loss of pigment by the use of artificial dyes. The most frequently used dye is nitrate of silver, whose solution colors the hair brown to black

(according to its concentration), inasmuch as the oxide of silver is reduced by the action of light. The hair is deprived of fat by washing with soap before the silver is applied. The discoloration of the skin, which is unavoidably stained by the solution, is prevented by immediate washing with chloride of sodium solution and cyanide of potassium. Combinations of silver and lead solutions, or iron salts with reducing agents, are also employed a good deal. One solution is brushed upon the hair, and the second is applied after the former has dried. By a proper combination of the amount and concentration of the fluids the color may be varied from light brown to black or yellowish red. As Dr. J. E. Polak has shown, various shades of black may be obtained by the use of the henna indica which is employed by the Persians. The powder, rubbed with water into a paste, is applied to the hair, and then indigo powder is applied, after steaming for half an hour. Henna powder, when applied as a paste, gives a fine, golden-blonde color. As a matter of course the application of dyes must be renewed as often as the gray hairs reappear. I will here mention a few formulæ: (*a*) To produce a black color: argenti nitr. 1.0, ammon. carb. 1.5, ung. emoll. 30.0; argenti nitr. 1.25, aquæ destil. 60.0, liq. hydrarg. oxynitr., spir. resedæ, āā 5.0; argent. nitr. 5.0, plumb. acet. 1.0, aq. rosar. 100.0, aq. colon. 1.0. For combined application: argent. nitr. fusi 5.0, aq. destil. 50.0 (Sig. No. 1), acid. pyrogal. 3.0, aq. destil. 40.0, spir. vin. rectif. 10.0 (Sig. No. 2); or argent. nitric. fusi 8.0, aq. destil. 70.0 (Sig. No. 1), hepat. sulph. 8.0, aq. destil. 70.0 (Sig. No. 2). (*b*) To produce a brown color: acid. pyrogallic. 1.0, aq. rosar. 40.0, spir. colon. 2.0. Another popular method is the inunction of the hair with "sulphur balsam" (sulphur stirred with the yolk of egg), and then washing with a mixture of vinegar and iron rust. The hairs are made darker by all fatty oils (ol. nucum juglandis, ol. macidis, ol. cassiæ, etc.), and they may be prescribed pure or in the form of pomade. For example, ol. ovorum, medull. ossium bovis āā 20.0, lact. ferri 1.50, ol. cassiæ æth. 1.0 (Pfaff). Dyeing of the hair yellowish blonde to golden red by means of "gold-water" (hydrogen hyperoxide) has recently come into vogue. Unless their unskilful application gives rise to eczema, the use of metallic hair dyes produces no evil effects, and I know many women who for years have resorted to hair dyes containing lead without the slightest injury to health. After a while the use of gold-water causes brittleness of the hairs.

LECTURE XXXVI.

ATROPHY OF THE HAIRS—ALOPECIA ADNATA, ACQUISITA, IDIOPATHICA, AND SYMPTOMATICA—SPECIAL FORMS : ALOPECIA SENILIS, ALOPECIA PRÆMATURA—ALOPECIA AREATA—ALOPECIA NEUROTICA—ALOPECIA PRÆMATURA SYMPTOMATICA—ALOPECIA FURFURACEA—ALTERATION IN THE HAIR—ATROPHIA PILORUM PROPRIA—TRICHORRHEXIS NODOSA—ATROPHY OF THE NAILS.

ATROPHY of the hairs includes every form of morbid modification of their typical growth. It may appear as an alteration of the general hair growth or as a structural change in individual hairs.

Imperfect growth of the hair, whatever its cause or character, is known as

ALOPECIA.

Celsus applied this term to every form of baldness of the scalp and beard. But baldness, calvities, is usually the final result of a combined process, of excessive falling-out (effluvium or defluvium, lapsus pilorum, psilosis) with insufficient after-growth of the hair, so that under such circumstances these pathological phenomena must be included in the conception of alopecia. Some writers apply the term alopecia merely to circumscribed falling-out of the hair of the scalp and beard, and use special terms for other forms of baldness. For example, phalacrosis s. calvities for baldness of the front part of the head ; ophiasis for a hairless streak running transversely across the scalp to both ears ; opistophalacrosis for baldness of the occiput ; hemiphalacrosis for unilateral baldness ; anaphalantiasis for loss of the eyebrows ; alopecia areata for disc-shaped losses of hair.

In view of the most essential symptoms and the causal factors, the following classification may be recommended :

Alopecia adnata.—Congenital deficiency or absence of hair (oligotrichia or atrichia), either partial or general. The condition is rarely permanent ; the hairs usually appear at a later period. It constitutes an inhibition of development and is often associated with delayed teething.

Alopecia acquisita.—Loss of hair during extrauterine life ; occurs as senile or premature alopecia.

Alopecia senilis begins with advancing age. The hairs first disappear from the frontal border of the scalp, so that the forehead

grows higher. When the baldness reaches its highest development in old age, it extends through the middle from the forehead to the vertebræ, and on the sides to about the middle of the temporal bones, while the hair remains at the occiput and lateral part of the forehead. The bald skin appears smooth, tense, shining, often fatty and atrophied. In later years the openings of the follicles are recognized with difficulty, and here and there contain downy hairs. It is much more frequent in males than in females. Alopecia is usually preceded by grayness, but the latter is not the cause of the former condition. The hair of the beard and mons veneris is very little affected by the atrophy of old age.

The diminished growth which is also evident in other tissues in old age is the direct *cause* of alopecia senilis, but it is noteworthy that baldness is more rare in women.

The *anatomical* conditions in the scalp cannot be regarded as the direct cause of the alopecia. The evidences of atrophy are found only after the skin has been deprived of hair for years. On microscopical section the sebaceous glands appear shrunken in places, others are dilated ; the hair bulbs are filled with epithelial scales and often contain a thin hair ; in many follicles the papilla and adipose lobules have disappeared ; the corium is thinned ; the connective-tissue bundles decreased in size, and their fibres exhibit in places vitreous or colloid degeneration and granulo-fatty changes.

Alopecia præmatura may be *idiopathic or symptomatic.*

In *idiopathic premature alopecia* the baldness occurs without demonstrable disease of the hair, the follicle, or the cutis. Such forms lack the independence necessary to the creation of a typical morbid picture, with the exception of a single variety—viz.,

ALOPECIA AREATA.

Sauvages was the first to describe under this term a form of alopecia in which the hair is lost over disc-shaped areas. Willan described it under the term porrigo decalvans, or bald ringworm, and distinguished it from common ringworm. In the former the discs of skin are bald and smooth from simple falling-out of the hair. In the latter the parts are covered with vesicles, pustules, and scales, and the hairs break off short. Both processes have often been confounded, especially since we have learned the mycotic character of Willan's porrigo scutulata, and since Gruby and others have also assumed a fungus in alopecia areata.

The process begins in one or more parts of the scalp or beard, more rarely in the hair of the axilla or mons veneris. In a limited area the hair falls out in an extremely short period of time. The hairs of the adjacent zone are so loose that they come out on the slightest traction, and also fall out spontaneously in a few days.

In the bald spot the scalp is smooth, white, sometimes moderately red, without scales or eruption, of normal temperature and sensation. Tenderness, anæsthesia, diminution of pigment, œdema, striking pallor, have been reported in rare cases. Pain and itching are absent. From the constant growth and union of the bald areas the larger part of the scalp becomes denuded of hair within six to ten months. As a rule the process ceases at the end of several months. The hairs at the borders of the bald spots first become more firmly adherent, then a few thin, at first non-pigmented. later pigmented hairs sprout up within the bald area Finally there is a new growth of hair throughout (sometimes only after the lapse of one to two years).

In a few cases the disease does not remain circumscribed. All the hairs of the body finally fall out, and the skin is as smooth as that of an eel. Even in such cases restitution may occur, but in certain others the hair is never restored.

A differential *diagnosis* must be made between alopecia areata and herpes tonsurans. The bald spot of the former sometimes contains fine, fatty scales, occasionally a pronounced seborrhœic deposit ; a few hairs may be broken off or are found to be brittle. But such exceptional conditions (which depend upon general seborrhœa of the scalp, or upon the brittleness of the hairs due to the fact that the latter have been separated from their nutritive base) will not lead to error. In children we often find one or more disc-shaped spots of seborrhœa congestiva or seborrhœa eczema, in which the hairs will fall out upon the slightest traction. Such forms must not be mistaken for alopecia areata.

The *prognosis* is not unfavorable, because, as a rule, the hair is restored within a few months or one to two years.

The *cause* of this peculiar disease is unknown. It does not appear to be due to general weakness, anæmia, local injuries, etc. It occurs suddenly in young and middle-aged people, and almost all the patients exhibit a fine growth of hair. It is not due to the internal administration of arsenic.

The *anatomical* changes of alopecia areata are also obscure. The presence of microphytes (Gruby's microsporon Audouini, etc.) has been repeatedly maintained by Malassez, Eichhorst, Thin, and others, but has never been demonstrated as an etiological factor, while Giovannini assumes an inflammatory process in the hair papilla (accumulation of leucocytes around the papillary vessels). The hairs which fall out are narrowed in the root portion and are broken off above the bulb ; in the latter locality a dilatation of the hair has been described by Rindfleisch.

We are forced to assume that alopecia areata is due to trophic disturbance (trophoneurosis) of entirely unknown origin. This the-

ory is also supported by the fact that the hairs reappear at the same time over the entire area. Max Joseph and Mibelli found that after division of the second cervical nerves in cats and rabbits, on the peripheral side of the intervertebral ganglion, the hairs fall out in disc-shaped spots on symmetrical parts of the ears. But these spots are only analogous to alopecia areata, and, in addition, the methods adopted in these experiments have also been combated (Samuel, Behrend, etc.).

In two cases of alopecia universalis, L. Froehlich observed amaurosis as the result of choroidoretinitis which developed after the alopecia. The relation between these two conditions is obscure, as I have never observed it in my cases, but the possibility of an identity of the primary process cannot be denied.

As a general thing the diagnosis is not difficult. The smoothness of the bald disc in which no stumps of hairs are visible, and the loosening of the hairs at the periphery which fall out on the slightest traction, are characteristic features. In a few cases, however, and in certain stages of the disease—for example, during the incomplete after-growth of new hair—it may be difficult to distinguish from herpes tonsurans and seborrhœa areata, since there are very often seborrhœal spots in alopecia areata or single hairs broken off and their stumps left. With regard to herpes tonsurans, the demonstration of the fungus is alone decisive.

The affection occurs in young people and adults of both sexes, and must be regarded as non-contagious. It is a noteworthy fact that endemics of alopecia areata have been repeatedly observed in France among certain companies of soldiers or in certain barracks. According to Feulard's recent statistics, these endemics are so numerous that we are forced to assume the transmission of contagion from one individual to another. In certain cases the use of a patient's headgear by a healthy individual was found to be the exciting cause. I am personally unacquainted with this "endemic" occurrence of the disease, and must therefore regard it as non-contagious. Even our French colleagues maintain the validity of the trophoneurotic form, but assume also a contagious variety.

Treatment can neither shorten the course of the disease nor prevent its outbreak in another locality. We may try irritating alcoholic-ethereal fluids to which have been added small amounts of acid. carbol., tinct. aconit., cantharides, capsici, veratri, ol. macidis, corrosive sublimate, pilocarpine; blisters, subcutaneous injections of pilocarpine muriate 0.2 : 20, with general tonics, are also indicated; electricity has been employed. Time often accomplishes more than all else.

Alopecia has also been observed in direct connection with diseases of the nervous system, and is then called idiopathic or *neurotic alo-*

pecia. The hair may fall out in the distribution of a sensory nerve whose function has been impaired as the result of injury, spontaneous disease, or changes in the nerve centres. Ravaton observed a corresponding alopecia in connection with right-sided amaurosis, Romberg in connection with unilateral facial paralysis, Cooper Todd in connection with concussion of the brain, and Deghilage in connection with trigeminal neuralgia. To the same category belong the cases of Schütz, Askanazy, Pontoppidan, but they are not identical with alopecia areata.

Among the neuropathic, idiopathic forms may be included the early baldness which is hereditary in some families, and the premature loss of hair owing to depressing emotions, very intense mental strain, and frequent attacks of migraine. Frédet reported a case of a girl, aged seventeen years, who, after escaping a serious danger, lost all her hair, including that of the body, within a few days (general effluvium capillorum). I have rarely observed pronounced neuroses or nervous and anæmic conditions in connection with alopecia universalis or alopecia areata regionalis.

Alopecia præmatura symptomatica includes those forms of rapid loss of hair and baldness which are due to material disease of the skin, particularly of the hair follicles and the sebaceous glands. The loss of hair appears to be confined to a few follicles or groups of follicles when the latter have been destroyed by suppuration or cicatrization, as in acne, sycosis, variola, ulcerative syphilis, lupus; or when the hair papillæ have become atrophied from dense cellular infiltration, as in the papules of the small papular syphilide, lichen ruber, or lupus erythematosus; finally, in favus and herpes tonsurans, in which the mechanical pressure and the influence of the fungous masses peculiar to these processes, together with the attendant inflammatory phenomena, give rise to loosening and falling-out of the hair and later to atrophy of the hair papillæ and obliteration of the follicles. In the same category should be mentioned ichthyosis, a congenital disease, as well as the previously described forms of alopecia of the eyebrows and scalp in lichen pilaris, with obliteration of the follicles and atrophy which Taenzer described as ulerythema ophryogenes, Besnier's *alopécie cicatricielle innominée,* Unna's ulerythema acneiforme, and other rare affections of a similar nature.

More extensive effluvium capillorum, even extending over the entire scalp, may be due to diffuse, acute inflammatory processes, which give rise to copious exudation in the rete and epithelial layers of the root sheaths. This causes loosening and exfoliation of the latter, and probably an analogous disturbance in the succulent root portions of the hair. This occurs in acute eczema and erysipelas of the scalp, after which all the hair is often lost within a few weeks.

Chronic exudative processes, such as eczema, psoriasis, lichen ruber, and seborrhœa, cause a loss of hair which, on account of the characteristic attendant desquamation of the epidermis, is known as *Alopecia furfuracea s. pityrodes.* Seborrhœa is the most frequent cause. It may develop subacutely and is then less unfavorable. This occurs after variola, typhoid fever, the puerperal state, and exhausting losses of blood. Seborrhœa and effluvium capillorum make their appearance, and, as a rule, at the end of a few months the hairs are restored.

Alopecia furfuracea runs a more unfavorable course when it is due to chronic seborrhœa. For one or two years the symptoms of the latter alone are noticeable, then the effluvium and baldness follow. Many hairs fall out on combing and spontaneously; the growth of hair is less dense; shorter and finer hairs are developed; and finally, as a rule, the fronto-temporal region remains permanently bald. This process is understood if we bear in mind the physiological process of the growth of the hair.

Each hair has a certain typical duration of life, at the termination of which it falls out. This is replaced by a new hair in the old follicle. This process corresponds to the typical shedding of hair which occurs at regular intervals in many animals. On the scalp of man it is occurring constantly, but with considerable variations in degree, dependent in part on the general condition of the organism and in part on local processes. The finer anatomical changes which accompany the desquamation of the mature hair and the production of the young hair have been studied by Heusinger, Kölliker, Langer, Stieda, Unna, Waldeyer, etc. It appears to be certain that each hair, after it has reached typical maturity, does not grow any further, inasmuch as the new formation of epidermis cells over the hair papilla ceases. If the cells which have been produced latest are cornified, they form, between the hair bulb and the hair papilla, a septum which is impervious to the nutritive fluids, and the hair is thus separated from the papilla. This separation involves the shaft of the hair with the inner root sheath, which, in moving upward, is often converted into a single layer of cells covering the papilla (Von Ebner). The basal cells and the external root sheath may also be converted into a layer which lines the bottom of the hair follicle and covers the neck of the papilla. On account of the diminished turgescence of the cellular masses filling the bottom of the hair follicle and the consequent diminution of the internal pressure, the greater pressure of the tissues surrounding the follicle will force the wall of the hair bulb inward and the mass of desquamated cells of the external root sheath will be pushed between the root of the hair and the papilla. Hence the hair in its entirety will be raised and pushed upward. The lower end of the root of the hair, which was formerly

concave to correspond with the enclosed papilla, now forms, together with the compressed cellular masses of the external root sheath, an apparently fibrous wedge with its apex directed downward (Fig. 45, *b*) The hair remains at first a little below the opening of the sebaceous gland or at the level of the insertion of the arrector pili. The bottom of the hair bulb is at the same time narrowed and shortened, because the vitreous membrane is often forced inward in folds (Von Ebner) and is pushed upward, together with the body of the papilla. The external and middle (probably muscular) sheaths of the hair bulb remain at their former depth because they are more firmly adherent to surrounding parts. Between them and the body of the papilla the neck of the latter is stretched, and a formation results to which Wertheim applies the term hair calyx. After a while active cellular infiltration of the papilla begins and a new epithelial wedge is formed above the papilla. This forces the papilla back to its former depth, and in its outer part, which corresponds to the border zone of the papilla, it is differentiated into the two layers (Henle's and Huxley's) of the internal root sheath. After it has extended nearly to the cast-off hair there develops in the middle portion—*i.e.*, from the covering cells of the papilla—a thin, pigmented, and at first non-medullated hair; the external root sheath is reproduced from the cellular layer left over in the fundus. According to this view (Langer, Von Ebner), the new hair develops from the old papilla; while others believe that this undergoes complete atrophy, and that, as in the embryonic development of the hair, a new papilla develops within the proliferating rete (Steinlin, Stieda).

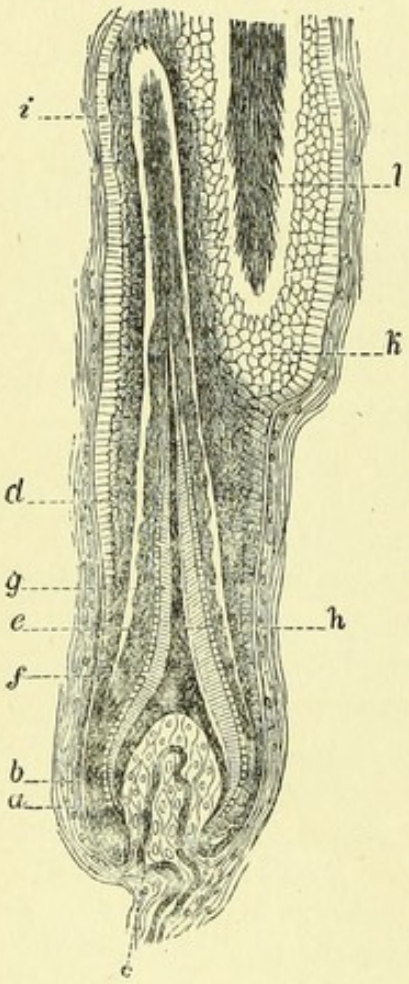

FIG. 45.—SECTION THROUGH A FOLLICLE IN PROCESS OF SHEDDING OF THE HAIR.

a, outer sheath; *b*, basement or vitreous membrane; *c*, papilla with vascular loop; *d*, outer, *e*, inner root sheath (divided into the layers of Henle and Huxley); *f*, cuticle of the latter; *h*, young hair; *i*, beginning of the new hair follicle; *l*, bulb of hair shed, with (*k*) the remains of its root sheath.

Various views are entertained concerning the fate of the cast-off hair which still remains in the follicle. Götte regards this hair, or rather the hair bulb (Fig. 45, *l*), as a supernumerary hair which has developed from proliferation of the rete cells during the interim in which the hair remaining is pushed up by the papilla. Unna calls it

"bed hair," and believes that the cast-off hair continues to grow for a time at the bulbous extremity. This growth is due to proliferation of the surrounding external root sheath ("hair bed"), and continues until it is reached by the new papillary hair ("secondary hair"). The same opinion is entertained by Esoff and Schulin, except that, according to them, a new papilla develops from the hair bed. Waldeyer also believes that new hair follicles may develop.

The longer the typical duration of the life of a hair the thicker and longer does it become. When the growth is luxuriant the papilla extends more deeply, while in fine and short-lived hairs the follicles remain shallow and the papilla is seated more superficially (Von Ebner).

The steadiness of the growth of the hair depends, accordingly, upon the constancy in the relation of the typical duration of life and the typical after-growth of the individual hairs. In chronic seborrhœa this relation is disturbed in an unfavorable manner. The individual hairs are shorter-lived ; they are shorter and finer, and fall out at an earlier period. According to Pincus the daily loss of hair varies from a minimum of thirteen to seventeen hairs to a maximum of sixty-two to two hundred and three hairs.

The reproduction of the hairs also becomes insufficient, qualitatively and quantitatively, as the result of the seborrhœa. The sebaceous glands and hair follicles are surrounded by the same network of vessels and nerves (Arnstein) and suffer from the same nutritive disturbance. Epidermis which is unequal to its physiological purposes is produced in the sebaceous glands, and in a similar manner the root sheaths of the hair which are continuous with the glandular cells are exfoliated, together with those formed by the papilla which are destined for the formation of the hair. The latter takes place imperfectly—*i.e.*, a delicate lanugo hair is formed or a loose column of epidermis which remains in the follicle. Finally, there is atrophy of the papilla and obliteration of the follicle, and permanent baldness develops in places.

In the majority of men premature baldness develops as alopecia furfuracea. In females the seborrhœa is more frequent, but is more subacute. Hence effluvium occurs repeatedly in them, but the hair is restored and baldness is much rarer. In a few cases, however, I have observed steadily advancing alopecia, with increasing thinness and shortness of the after-growth, in women suffering from anæmic seborrhœa or general anæmia. Michelson has also observed the alopecia upon other parts of the body (alopecia pityrodes universalis). The cocci and bacteria found in the scales of the skin have also been regarded as possessing an etiological importance (C. Boeck) in alopecia furfuracea.

Anatomically the cast-off hairs show nothing abnormal. They

appear broken off at the root, are often fibrillated and narrowed. In time the bald skin changes, as in alopecia senilis. The *prognosis* is better in the acute and subacute forms of alopecia furfuracea and during the first few years, unfavorable at a later period.

The *causes* of the seborrhœa which gives rise to such alopecia have been mentioned in part. The remote causes are, in general, anæmia (spontaneous or following acute and chronic diseases), chlorosis in women, chronic gastricism and anæmia in men, the cachexia of cancer and phthisis. The syphilitic diathesis, in its later stages, may also give rise to seborrhœa and alopecia. The alopecia associated with seborrhœa sometimes attacks the eyebrows or beard (it may even attack them exclusively), and in exceptional cases involves the entire scalp in a relatively short time (malignant form of alopecia pityrodes, Michelson).

The *treatment* of alopecia furfuracea must be directed against the seborrhœa. After the scaly masses have been softened with oil and removed by washing with soap, the scalp is brushed once or twice a day with alcohol to which acid. carbolic. or acid. salicyl. (1 : 200), veratrin (0.50 : 300), resorcin, tinct. benzoes (1 : 200), bals. peruvian., æth. sulph., æth. petrolei are added. The scalp is also washed once or twice a week with spir. sapon. kalin. or sapo-naphtholo-sulphur., and cold douches are frequently used. Applications of tinct. rusci or sulphur-alcohol paste, naphthol oil or alcohol, are advisable when the skin is congested. On account of the dryness of the skin resulting from such treatment, we must make inunctions of pomades to which are added tannin, quinine, cantharides tincture, capsicum, veratrin, ethereal oils, white precipitate. The so-called tanno-quinine pomade and ung. gemmarum populi are often used. We may prescribe simpler formulæ—for example, præcipit. alb. 0.50, ung. emoll. 50.0, tinct. benzoes 1.0, ol. rosar. gtt. 5 ; or Dupuytren's pomade (medull. ossium 75.0, extr. chinæ frigid. 10.0, tinct. canthar., succi citri āā 5.0, ol. de cedro, ol. bergam. āā gtt. 10). Schmitz recommends subcutaneous injections of pilocarpine in order to stimulate the growth of the hair.

Cutting the hair short does not have the desired effect and should not be recommended to female patients. In addition to local treatment, the remote cause—seborrhœa, chlorosis, anæmia, chronic gastricism—must be treated by iron, bitters, arsenic, milk and whey cures, baths, country air. Recovery can only be expected after appropriate treatment for several months.

ATROPHIA PILORUM PROPRIA is the term applied to a destructive change in the shaft of the hair itself. This takes place secondarily to disease of the follicle, and more directly by the action of the fungi which separate its elements in favus and herpes tonsurans. The hairs lose their gloss, become brittle, and break off above their point

of exit. This category also includes the dryness and dulness of the hair in phthisical and febrile patients.

Trichoptilosis is an idiopathic form of atrophy of the hair. The long hairs (which have not been cut, and hence usually in women) are often found split into two or more fibres from the tip. It may be the result of local dryness, because otherwise the growth of the hair is normal. Duhring has observed a special form of longitudinal splitting in which the separation occurs upward from the bulb. A more frequent form is that known as *trichorrhexis nodosa*, in which the hairs are dilated and fissured. I have found this very often in the beard and moustache, rarely on the scalp. Over a circumscribed area or more diffusely we find one or more spherical or spindle-shaped swellings upon the hairs ; they look like nits or give the appearance of a rosary to the shaft of the hair. In addition we find stumps of hair which terminate in a dull, glistening, spherical enlargement, and if many of these are present the hairs look as if they had been burned. If traction is made upon the hairs they break at once in the middle of a nodular enlargement, the lower half remaining upon the stump. The microscope shows that the cortex of each nodule is distended and fissured, while the internodular parts are normal, except that the medullary space is enlarged in places. Schwimmer states that the roots of the hair are smaller and that the nutrition is thus impaired. Michelson also regards the condition as the result of desiccation owing to impaired nutrition and supply of nutritive juices. Although I concur in his opinion, this does not explain the fact that the hairs become enlarged and break off at certain places. The cocci which are found occasionally in the interstices of the fibrillated part of the hair are mere accidental deposits.

Behrend believes that the "ringed hairs" (Karsch) constitute a special form of disease. In this condition the hairs exhibit, at regular intervals of about one millimetre, alternate spindle-shaped thickenings and narrow interspaces, but without any tendency to splitting or breaking. Behrend also found this condition upon the interfollicular part of the hair shaft. He believes, with Virchow, that this anomaly is due to periodical aplasia of the hair (aplasia pilorum intermittens of Virchow, aplasia moniliformis pilorum of various authors), corresponding to the narrow part of the hair, while the spindle shaped part belongs to the period of normal growth.

Trichorrhexis disfigures the individual very decidedly and is extremely obstinate. Local applications have been of little benefit, and shaving has been effective in only a few cases.

None of these conditions have anything in common with that in which yellowish-brown to orange-red, very hard, finely granular deposits are found upon the hairs, especially in the axillæ. These

dissolve with difficulty in acids and alkalis, and, apart from the presence of cocci, consist chiefly of the products of desiccation of the cutaneous gland secretions, especially of the axillary glands.

ATROPHY OF THE NAILS.

Onychatrophia is often congenital. It occurs as an absence or imperfect development of the nails upon certain rudimentary fingers and toes, and is generally associated with absence of the hairs. In the acquired condition the appearances are the same as those of hypertrophy (deformity, discoloration, fissuring, brittleness), and it also occurs under the same local or general influences (see page 451).

Imperfect formation of the nails may result from all processes which are associated with disturbances in the development of the epidermis, such as chronic eczema, psoriasis, ichthyosis, lichen ruber, syphilis, etc. As the result of a psoriasis of the fingers which had lasted for years, I found that all the nails formed soft, membrane-like plates, without the slightest tendency to cornification (hapalonychia, from ἁπαλός, soft). This condition lasted ten years, and not alone interfered with the use of the hands, but was also very painful, because there were constant suppurations beneath the nail. Finally, however, the development of the nails became normal. In local asphyxia of the hands I have seen thin, vitreous, and brittle nails.

LECTURE XXXVII.

ATROPHIA CUTIS PROPRIA, IDIOPATHICA (XERODERMA, STRIÆ ATROPHICÆ, ATROPHIA SENILIS) ET SYMPTOMATICA (SCARS OF PREGNANCY)— QUANTITATIVE AND DEGENERATIVE ATROPHY— LUPUS ERYTHEMATOSUS.

ATROPHIA CUTIS PROPRIA.

ATROPHY of the skin proper occurs as a diminution in the mass of the general integument or as an abnormality in its chemico-biological qualities. It is easily understood that quantitative and qualitative atrophy are often found in combination. The atrophy is either spontaneous or secondary; it may be diffused over large areas of skin or confined to small points, stripes, or patches.

The *idiopathic diffuse* forms include xeroderma and senile atrophy. *Xeroderma* or parchment skin occurs in two forms.

One type, which I call *xeroderma pigmentosum*, I have seen in fourteen cases (eleven females, three males) between the ages of three and twenty-two years (only once in a woman of sixty years). Geber, Taylor, Heitzmann and Duhring, Rüder, Neisser, Vidal, Crocker, Pick, J. C. White and Janowsky, Elsinger, Besnier, Brayton, and others have also reported cases, almost exclusively in young girls (seven to eighteen years). Until 1886 forty-three cases had been observed, and fifteen to twenty have been reported since that time. According to Brayton thirteen have been observed in the United States.

The face, ears, neck, shoulders, the chest to the level of the third rib, the arms and backs of the hands, sometimes the legs and dorsal surfaces of the feet, are dotted with larger and smaller yellowish-brown patches like freckles. Between these are found pock-like, whitish, glistening, shallow depressions, or the integument has a normal color. The variegated appearance of the skin is intensified by numerous punctate or linear telangiectases. The epidermis is thin, in places smooth, in other parts it is detached in thin lamellæ or finely furrowed, fissured, wrinkled, dry like parchment. The cutis itself feels thin, but after the disease has lasted for some time it is with difficulty raised into folds; it is more firmly adherent to the underlying tissues and is poor in fat. The general integument of the rest of the body is normal in every respect. So far as we can learn, the

disease always begins in earliest childhood and progresses steadily. Small telangiectases and pigment patches first develop; then the vessels are almost entirely obliterated, and corresponding achromatic, whitish, glistening, atrophic depressions are left over. Later there is diffuse shrinking of the skin, above which the epidermis becomes wrinkled and is detached in lamellæ.

The increasing shrinking of the skin is followed by eczema, shallow rhagades and ulcers, narrowing of the mouth and nares, and ectropion of the lower lids (this gives rise occasionally to xerosis of the cornea). In the majority of cases (five times among my ten cases) carcinoma, sarcoma, or angioma has developed in a few months upon scattered parts of the face, lips, nose, lids, cheeks, concha of the ear. In two cases internal metastases developed with a fatal termination.

The majority of writers attach the chief importance in this disease to the new formation of vessels and telangiectases or to the pigmentation. Geber regards it as a peculiar form of nævus pigmentosus. Taylor calls it angioma pigmentosum et atrophicum. Pick calls it melanosis lenticularis progressiva. Neisser, who attaches importance to the atrophy, speaks of liodermia cum melanosi et telangiectasia; Crocker, of atrophoderma pigmentosum.

I see no reason for abandoning the term xeroderma pigmentosum which I selected originally. It is justified historically and expresses briefly that the process is a cutaneous atrophy, attended by, or giving rise to, the formation of pigment and disease of the vessels.

Beginning, as it does, in earliest childhood, and characterized by pigment patches and telangiectases, the disease reminds us of melanosis adnata, of nævus and lentigo. But it is distinguished from the stationary nævi by its constant and rapid progress and by the continued atrophic change in the tissues.

Anatomical investigations have confirmed our original views. The process appears to begin with proliferation of the connective tissue of the papillæ and the vascular endothelium, followed by retraction of the former and obliteration or, in some parts, dilatation or new formation of vessels. Secondarily there is an irregular accumulation of pigment with a prolongation of rete pegs into the derma, ectasia of the glands and degeneration of their epithelium. It is evidently this change in the developmental relations of the epithelioidal tissues which furnishes the starting point for the development of cancer and sarcoma—certainly a striking circumstance in such young individuals.

The *cause* of xeroderma pigmentosum is unknown. It must be due to a congenital anomaly of development and nutrition of the papillary stratum, of its vascular and pigmented portions. The congenital predisposition is also shown by its frequent occurrence in children of the same family. Among my ten cases it occurred once

in two and once in three brothers and sisters, and among the entire forty-three cases observed it occurred six times in two, four times in three, and once in seven brothers and sisters. The influence of light (Unna) or the sun (Pick) has been regarded as an exciting cause. This is not borne out by the facts.

The *diagnosis* is not difficult. It closely resembles scleroderma in the atrophic stage, but the latter disease always begins with sclerosis of the tissues. There is a still greater resemblance to a certain form of macular leprosy, but the latter is attended by anæsthesia and mutilations. It is distinguished from multiple pigmentoses, lentigines, and ephelides by its steady progress and the atrophy.

The *prognosis* is unfavorable, especially on account of the tendency to the development of cancer and sarcoma. No case has ever recovered. The *treatment* must be confined to relieving the subjective symptoms (tension, dryness, pain of the rhagades, excoriations, and ulcers) and to the removal of the malignant complications, such as carcinoma, sarcoma, and angioma. In one case I removed at various times fifty or more large and small cancerous growths, and at the end of two to three years an equal number had again developed. According to the post-mortem findings, there are also metastases of the internal organs in such cases.

The second type of xeroderma is a *stationary condition of atrophy of the skin.* From the middle of the thigh to the sole of the foot (more rarely from the arm to the palm of the hand) the integument is unusually white (poor in pigment), in places tense and raised into folds with difficulty. The epidermis is extremely thin, dull glistening, wrinkled, and raised in thin, shining leaflets. There is great tenderness of the tips of the fingers, palms of the hands, and soles of the feet, on account of the insufficient epidermic protection; this interferes greatly with walking and the use of the hands. The condition remains stationary from earliest childhood. In this way it is easily distinguished from scleroderma atrophicum; the thinning of the skin distinguishes it from ichthyosis. The object of treatment is to mitigate the dryness and tension of the epidermis by bland ointments and plasters, and to protect the soles of the feet from pressure in walking.

In *senile atrophy* the skin is sallow or dark brown, dry, wrinkled, scaly (pityriasis tabescentium), often covered upon the trunk, neck, and arms with scattered, flat, wart-like, dirty yellowish-brown deposits from the size of a lentil to that of a five-cent piece. These are easily crumbled with the finger nail and can be detached. Their base is formed of smooth skin or of a readily bleeding, granular papillary group. They may consist of the expansion of an epidermic cone protruding from the dilated opening of a sebaceous gland, and are composed of an agglomeration of epidermic cells con-

taining fat granules. The skin is usually loose and can be raised in large folds on account of the diminution of adipose tissue.

This condition is due to senile retrogressive changes in most of the cutaneous structures, which are essentially the same as those in other organs. They may be distinguished as dryness, induration (Paget), or simple atrophy (Virchow). Its characteristics are poverty in tissue juices and denseness of the tissues, together with scanty development of new tissue elements, shrinking and atrophy of the parts. The epidermis is thinned and runs over the flattened papillæ without interpapillary projections. The narrow corium contains small, shrivelled connective-tissue corpuscles and pigment granules; the narrow tissue spaces contain a scanty fluid poor in cells, the vessels are partly obliterated (Kölliker) or abnormally dilated (Neumann) and filled with clumps of pigment. In many hair follicles the papilla is shrunken, the hair absent or lanugo-like, the cells of the external root sheath cornified. Many sebaceous glands are dilated, especially in certain acini, and filled with a crumbling mass of epidermis. The adipose cells are flabby or absent in places, where the rhombic network of the connective tissue alone can be found.

The second form of senile atrophy is mainly degenerative in character. The elements of the cutis undergo an organic change, so that their vegetative and functional properties are impaired. The connective-tissue fibres are granular or their contours become effaced; they are converted into a more homogeneous, tough, or brittle mass (vitreous swelling, amyloid, colloid, hyaline, waxy, fatty degeneration).

In one case of yellow, transparent, molluscum-like papules of the face (which resembled E. Wagner's colloid milium), Besnier demonstrated colloid degeneration of the dermal connective tissue.

Circumscribed idiopathic atrophy of the skin occurs in the form of patches from the size of a finger nail to that of a dollar, or in whitish, scar-like streaks, several centimetres long and two to five millimetres wide, which are slightly depressed below the level of the surrounding normal skin. These develop imperceptibly, in adult males and females, upon the buttocks, trochanters, anterior rim of the pelvis, thighs, also not infrequently upon the trunk, neck, and arm. The atrophic patches are usually isolated, the stripes occur in numbers arranged in parallel sinuous lines, at various angles to the long axis of the body; they are often arranged symmetrically upon the two halves of the body, the upper and lower limbs. On microscopical examination it is found that the bundles of fibres are separated in places and that the loops of connective tissue which enter into the structure of the papillæ are elongated so that the latter appear to be almost entirely obliterated. The vessels, glands, and adipose lobules are scanty within the atrophic regions.

These atrophic streaks and patches have been rightly attributed by B. S. Schultze to the stretching of the skin during rapid growth of the pelvis and limbs. He found the affection in thirty-six per cent of females (non-pregnant) and six per cent of males. In Cantani's case (a young man of twenty years) the atrophic stripes seem to have been caused by sudden stretching of the skin due to rapid increase of adipose.

Not infrequently the developing streaks and patches present hæmorrhagic phenomena, so that there is no doubt that they are preceded by sudden or gradual distension of the meshes of the cutis with coincident rupture of the corresponding vessels, as in the cicatrices of pregnancy. A similar effect may also be produced by forced movements of extension, and hence the number of atrophic patches may increase in time.

The vascular ruptures and hæmorrhages associated with the stretching of the skin are attended, in rare cases, with painful and inflammatory swelling. This causes wheals or swollen patches with the well-known changes in color. These do not disappear until several weeks have elapsed, and the corresponding macula or stria atrophica is then visible. Podratzky observed a case of this kind in a soldier, and a case described by Pelizzari as "erythema urticans atrophicans" belongs to the same category. Some individuals appear to have a special predisposition in this respect, so that these ruptures develop on forced extension of the skin in standing, jumping, etc.

Traumata acting from without may also cause distension of the deeper layers of the corium without lesion of the upper layers, and may result in maculæ and striæ atrophicæ. Wilson describes several cases of "linear atrophy" after injury (corresponding to the frontal and naso-alar nerves).

In old, marasmic people, more rarely during middle age, atrophy of the subcutaneous tissue, perhaps also a coincident degenerative change of the cutis similar to that of senility, may produce a *diffuse, progressive atrophy* of the skin. Cases have been described by Buchwald ("idiopathic atrophy"), Touton ("acquired idiopathic atrophy"), and Pospelow ("atrophia idiopathica progressiva"). In large patches upon the lower limbs and nates, more rarely on the backs of the hands, the arms and trunk, the skin appears thin, flabby, devoid of adipose, dry, finely scaly, of a glistening brownish or whitish color, like crumpled cigarette paper (Pospelow). In dots, streaks, and patches it appears speckled, in various shades of blue to brownish red, as if ecchymotic from contusion. Such spots disappear, leaving brown pigmentation. At a later period the integument of the legs becomes thinner and more tense. The process gradually advances. In a few cases I have seen the

same process in women, starting from the backs of the hands and extending to the arms, in one case limited to one elbow and the adjacent extensor surfaces of the arm and forearm.

The *secondary atrophies* of the skin are the result of a traumatic or pathological process, and occur as simple or degenerative atrophy. The former include compression atrophy, resulting from a tumor which pushes the skin before it. If the pressure continues, the part which is subjected to the greatest pressure suffers complete loss of its tissues or undergoes inflammation and gangrene. If the stretching is temporary, as in ascites and anasarca, atrophic patches and stripes develop, similar to the idiopathic striæ atrophicæ. In primiparæ the stretching of the abdominal integument by the uterus first produces bluish-red, hæmorrhagic spots; after these grow pale, whitish cicatricial patches and striations are left over (the cicatrices of pregnancy). The corium also undergoes partial atrophy as the result of external pressure (under corns or favus crusts).

Finally, this class includes the scar-like, punctate, and macular depressions of the skin which are left over at the site of absorbed inflammatory and neoplastic infiltrations of the corium after syphilitic papules, lupus, leprosy, lichen ruber. Such atrophic depressions disappear almost entirely in time, on account of the elasticity of the interstitial healthy parts of the skin.

Degenerative symptomatic atrophy involves the same tissue changes as senile atrophy. It usually occurs as the result of chronic processes of inflammation and new formation in the general integument. Hence it is diffuse and appears after chronic eczema, pemphigus, chronic dermatitis, pityriasis rubra, local asphyxia and its secondary œdema, the sclerosis of chancre. In all these processes the tissue elements of the skin undergo fatty, waxy, hyaline, vitreous degenerations, as the result of pressure by the infiltrating masses or obliteration of the vessels.

It is questionable whether the peculiar condition described by Kopp, Du Mesnil, and Otto Seifert as *cutis laxa* is a degenerative one. I have observed two cases. These persons could raise the skin in long folds, which, when released, recoiled like rubber. The cutis felt everywhere soft and jelly-like, and a firm fatty layer was nowhere perceptible. The adhesions to the bony prominences were either absent or very lax. In one of these cases Du Mesnil and Otto Seifert found absence of the fibrous bundles of connective tissue, so that the elastic fibres were not hindered by the resistance otherwise offered by the former; at the same time the fibrous bundles were converted into myxomatous tissue. This would indicate a degenerative condition. Perhaps, however, we have to deal with an (inherited) inhibition of development—*i.e.*, a persistence of the cutis

tissue in an embryonic condition. This view is favored by the occurrence of the disease in father and son (Kopp's cases).

LUPUS ERYTHEMATOSUS.

These forms of degenerative atrophy are allied to a peculiar type of degeneration and atrophy of the skin which develops from an inflammatory process of the skin and is known as lupus erythematosus.

Although the term lupus is connected with an entirely different process which is manifested by local destructive action, lupus erythematosus does not bear the slightest relation to lupus vulgaris. It is not surprising that the older surgeons confused the two diseases, when many dermatologists of experience, even in latter years, describe transition of lupus erythematosus into lupus vulgaris, as well as mixed forms of the two processes.

The main clinical and anatomical characteristic of lupus erythematosus is inflammatory. It consists essentially of an inflammation of the skin, a dermatitis, which may begin in various layers and regions of the cutis and subcutaneous tissues, but is confined chiefly to the follicles and sweat glands. The nature of the disease is determined, however, not by the inflammation, but by the constant termination in a peculiar atrophy of the skin. Hence I believe the disease should be considered here, in conjunction with the degenerative forms of cutaneous atrophy.

We have referred to this essential feature on former occasions, but without entering into details. In regard to the inflammatory forms of seborrhœa of the face (page 124), we stated that Hebra had described such forms as seborrhœa congestiva and that the latter may form the preliminary stage of lupus erythematosus. The former may last for months and years without changing into lupus erythematosus. Inflammatory seborrhœa is only converted into lupus erythematosus by its termination in cicatricial retraction.

The beginning of the disease is characterized by the appearance of one or more red, slightly raised patches, which grow pale under the pressure of the finger and vary from the size of a pin's head to that of a lentil; each patch is depressed in the centre or has a cicatricial gloss, or is provided with a thin, firmly adherent scale. The central scale with the red, elevated border furnishes a characteristic appearance and forms a sort of primary efflorescence of lupus erythematosus. From this a twofold form of the disease may develop:

1. *Lupus erythematosus discoides.* This develops into the characteristic discoid form in the course of several months or one to two years. The red, elevated border spreads peripherally, while at the centre the skin appears depressed, cicatricial, shining, or covered with firmly adherent dry scales. The border is usually sprinkled

with numerous black comedo specks or large, gaping openings of the sebaceous glands.

In this way discs develop from the size of a lentil to that of a dollar or even the palm of the hand. Lupus erythematosus discoides is found mainly upon the cheeks and the bridge of the nose, in which event it resembles a butterfly (Hebra); also upon the tip and alæ of the nose, the eyelids, ears, lips and their vermilion border, scalp (attended here by loss of hair), the flexor surfaces of the fingers and toes, and all parts of the face. The discs are arranged irregularly, discrete, or combined into arcs of circles and serpiginous lines.

Each disc enlarges to a certain extent, persists unchanged for many months or years, and disappears after fading and flattening of the borders. As a matter of course, the cicatrix remains. In the meantime a new disc appears in an adjacent region, and in this way the process may last fifteen to twenty years. With the exception of rare complications with submaxillary adenitis, swelling of the parotid, and more rarely with erysipelas, the patients enjoy good health, and the only bad effects of the disease are the deformity and the permanent alopecia of the beard and scalp.

2. *Lupus erythematosus disseminatus s. aggregatus* also develops from the primary efflorescence described above. These appear, at the start, in large numbers upon the face, cheeks, and other localities, and the disease spreads by an increase in the number of the efflorescences, not of their size. Some patches resolve quite rapidly, while others persist for months. They sometimes grow to the size of a pea or larger, have a brownish-red color, are traversed by dilated vessels, are elastic, and have a flattened globular shape. Otherwise the central part undergoes cicatricial retraction or exhibits a depression corresponding to the mouths of the follicles. These present a great resemblance to syphilitic papules, to the nodules of lupus and leprosy, or, when situated on the ears and flexor surfaces of the fingers, resemble perniones.

The eruption is found not alone on the face, scalp, lips, ears, and auditory canal, but is also thickly scattered over the trunk, upper extremities, fingers, hands, toes. In rare cases it extends over almost the entire integument. The process gradually attains such an extension, but occasionally there is an acute febrile eruption, attended with nocturnal pains in the bones, pains and exudation in the joints, and headache. In a series of cases we have seen severe erysipelatoid swelling of the face, which did not extend beyond this region ("erysipelas perstans faciei"). This was attended by a typhoid condition, a temperature exceeding 40°, coma, stupor, a dry leathery tongue; half the cases terminated fatally.

In several cases we have seen the development of many hundred hæmorrhagic or clear, watery vesicles, as in herpes iris. These

soon dried into crusts, and, after the latter fell off, characteristic efflorescences of lupus erythematosus, with a depressed centre, were left behind. In the last few years other dermatologists have also seen such severe cases of acute lupus erythematosus, with or without erysipelas perstans faciei (Besnier, Tenneson, Hardaway, etc.).

Such acute eruptions are peculiar to the disseminated form of lupus erythematosus. They rarely complicate an already existing discoid form, and then the latter also occurs in the shape of disseminated patches. Hence both forms are often combined, either from the start or during the further course of the disease.

In a number of cases I have seen the mucous membrane of the hard palate and cheeks attacked by an analogous change : patches from the size of a dot to that of a lentil, shallow, reddish, or gray excoriations, and bluish-white cicatricial patches.

Lupus erythematosus always runs an extremely protracted course, the individual patches lasting months and years, and the entire disease ten to twenty years. It always terminates locally in cicatricial changes in the skin, so that the face looks as if covered with pock-marks, and the hair may be lost in many parts of the scalp. Many patches, however, disappear without leaving a trace.

The *prognosis* is favorable, in so far as the disease does not directly endanger life, and in the majority of cases the constitution in general is not affected. This is particularly true of the discoid form. In the disseminated form, particularly when complicated with acute and general outbreaks, attendant cerebral symptoms, and erysipelas perstans faciei, the prognosis is less favorable. Among eleven cases in which the last-mentioned complications were present, six proved fatal, and in disseminated lupus in general there were nine fatal cases. Pleuropneumonia was the immediate cause of death in six cases, pulmonary tuberculosis in three cases, atrophy of the cerebral cortex and œdema of the meninges in one case, marasmus and anæmia in one case. In one fatal case of lupus erythematosus acutus, Jarisch found inflammatory changes in the central lateral parts of the anterior horns of the spinal cord.

The cutaneous affection *per se* is also less amenable to treatment in the disseminated form, because so many of the morbid foci are present at the same time, and they may appear unexpectedly here and there in large numbers. On the other hand, many of the acute patches disappear in a few days without leaving a trace.

The discoid form is the more favorable in regard to the general course and the more restricted localization, but the local changes (cicatrization) in the skin, as a rule, are more severe. The persistence of many telangiectases in and around the cicatrices is also a result of both forms of lupus erythematosus.

Anatomical investigations have shown that lupus erythematosus

is due to an inflammation of the cutis leading to degeneration and atrophy. It has been found, however, that not alone the sebaceous glands and their vicinity, but also the sweat glands and all other structures and layers of the skin, may be the starting point and site of the disease. Sometimes the process begins in the upper layers and in the distribution of the blood vessels surrounding the sebaceous glands and their excretory duct (red, elevated patches), at other times in the network supplying the sweat glands (as in lupus erythematosus of the palm) and fat lobules (firm, œdematous nodules). From them the disease gradually spreads to all the layers and tissues of the skin.

In recent foci the cutaneous follicles and glands are surrounded by accumulations of cells, with the other histological signs of inflammation (dilatation of the vessels, proliferation in the walls of the vessels, œdema, cellular infiltration of the connective tissue, proliferation of the connective-tissue corpuscles). These changes are found in the depth of the corium (nodules) or in the superficial layers (red patches). Their effects are visible in proliferation of the glandular cells (seborrhœa), firmness and nodular swelling of the skin, desquamation of the epidermis. In acute exacerbations of the inflammation there is an exudation of serum and bloody fluid among the layers of the epidermis (formation of vesicles) and extravasation into the corium and papillary body (hæmorrhage). In many places resolution occurs after this stage. The inflammatory phenomena disappear and the infiltration is absorbed. As a rule, however, the inflammation passes into degenerative changes of the tissues. We then find, in addition to a slight tendency to the formation of granulation tissue (Geber), granulo-fatty cloudiness of the rete, of the round cells, and of the infiltrated connective tissue ; this is followed by absorption and retraction. The same changes in the glandular elements and their surrounding connective tissue lead to obliteration of the hair follicles, sebaceous and sweat glands, and the fat cells ; some of the blood vessels are contracted, others become dilated.

The *diagnosis* may be attended with difficulty. The disc-shaped form may be mistaken for herpes tonsurans or an orbicular syphilide. It is distinguished from herpes tonsurans by the cicatricial retraction in the centre of the disc ; from the syphilide by the fact that the rim shows evidences of inflammation (redness which fades under the pressure of the finger, and œdematous infiltration), while in syphilis there is a hard, shining infiltration of the border. The first outbreaks of lupus erythematosus disseminatus may resemble eczema impetiginosum, squamosum, herpes tonsurans maculosus, syphilis papulosa, pernio (when located on the fingers). bromide acne, and herpes iris, and great care is often necessary in order to recognize the differential characteristics.

The *etiology* of lupus erythematosus is only partially understood. There is no doubt that seborrhœa congestiva, whether it develops spontaneously or after erysipelas or variola, may lead to lupus erythematosus—*i.e.*, it forms an early stage of the latter affection. In other regards the assumed etiological factors are general in character. The majority of cases occur in middle life, but I have seen one case in a child of three years and another in a very old person. Two-thirds of the patients are females.

The acute and fatal course of certain cases and their peculiar symptomatology remain entirely unexplained, inasmuch as the results of autopsies shed no light on the question. Chlorosis, anæmia, dysmenorrhœa, seborrhœa capillitii, sometimes sterility, chronic catarrh of the apices of the lungs and beginning pulmonary tuberculosis, swelling of the submaxillary glands—*i.e.*, the symptoms of cacotrophy—are often observed in such female patients. Male patients, on the other hand, usually appear to enjoy good general health.

The *prognosis* in regard to the duration of the disease and the results of treatment must be made cautiously. It may happen, for example, that a very extensive lupus erythematosus disseminatus which has lasted a year is completely cured within a few weeks, while a single disc constantly enlarges under treatment, and, as new lesions develop, the entire process may continue for years. The patients should also be informed that, although many patches disappear without leaving a trace, the majority of them leave superficial, delicate scars and telangiectases.

As to the means and methods of *treatment*, we must first employ mild remedies which act superficially, and only after they have proven useless do we resort to caustics. Milder means are again employed as soon as any improvement is obtained.

The best remedy is washing with spiritus saponatus kalinus, which is sometimes curative when used alone, and is also employed intercurrently with other forms of treatment. More intense effects are produced by soft soap spread on flannel and applied to firmly infiltrated lupus discs, or by washing with naphthol-sulphur soap, or cauterization with a concentrated solution of potash 1 : 2 aq. destil., or ammonia, acetic acid, or hydrochloric acid.

If one of these remedies, when rubbed into the border of the lupus with a hard brush, succeeds in making the surface uniformly raw (so that blood and serum exude), the surface usually grows flat and pale at the end of a few days. Recovery may then be completed by simple washing with soap.

Methodical applications of sulphur pastes, tar, iodine, sulphur, mixtures of sulphur, tar, and spirit. sapon. kalinus, tincture iodine and iodine-glycerin, according to the formulæ recommended in the

treatment of acne (see page 376), give rise to a reactive inflammation and swelling. This runs its course in a few days and often improves the condition to such an extent that simple soap washings may do the rest.

I have not seen much good effect from cauterization with carbolic and salicylic acids or white precipitate ointment, while deeper cauterizations with hydrochloric and nitric acids, zinc chloride, and chromic acid are not to be recommended. We have seen excellent results from the application of emplast. hydrargyri, beneath which lupus erythematosus discoides as well as extensive lupus disseminatus may disappear completely within a few days or weeks. Painful lupus spots on the fingers (lupus pernio, Besnier) improve with particular rapidity under this plan. Chrysarobin and pyrogallic acid ointments are also very effective, while covering with rubber acts merely as a softening remedy.

Scraping with a sharp spoon, and scarification, either alone or followed by cauterization (chloride of zinc or thermocautery) are often successful, and are especially indicated in cases of deep infiltration with vascular dilatation. In acute outbreaks with signs of inflammatory, painful swelling and deep formation of nodules, I have often secured rapid resolution of the majority of patches by the application of ice bags, compresses of acetate of lead, or liquor Burowii. Cold douches and baths may be recommended for the same purpose.

In view of the large number of remedies employed, it is to be kept in mind that all may prove successful or leave us in the lurch, and that a remedy which proved useless in a case several months ago may be effective in the same case upon making another trial.

Among internal remedies, starch iodide (McCall Anderson), iodoform (Besnier), and arsenic (Hutchinson) have been recommended as curative, but this has not been confirmed. Among the remedies which aid local treatment indirectly are those which improve the general condition in cases of chlorosis, anæmia, pulmonary catarrh, and tuberculosis (iron, arsenic, cod-liver oil, mountain air, milk cure, cold-water cure, etc.).

We are entirely unable to prevent a relapse, but fortunately many cases, when cured, remain permanently well.

CLASSES VIII. AND IX.

NEOPLASMATA.

LECTURE XXXVIII.

NEW GROWTHS—GENERAL REMARKS—CLASSIFICATION—BENIGN NEOPLASMS—CONNECTIVE TISSUE NEOPLASMS—KELOID—SCARS—MOLLUSCUM FIBROSUM—XANTHOMA—FIBROMA—LIPOMA—NEUROMA—DERMATOMYOMA—OSTEOMA.

THE conviction has gained ground that a neoplasm must be regarded merely as a clinical conception, as a term applied to a pathological formation which appears, on account of its general qualities and its relations to surrounding tissues and organs, as something foreign, whether its elements are similar to the mother soil (homœoplasia) or foreign (heteroplasia). In regard to their clinical course neoplasms are still divided into benign and malignant. The first class includes those which exercise little or no local destructive action, although they may last for years, and do not have an injurious effect on the general organism. Malignant tumors have a local destructive action and a deleterious effect on the general constitution.

A classification may also be made according to their histological characteristics—for example, connective-tissue neoplasms, vessel neoplasms, granulation tumors or granulomata, and epithelioid tumors.

The granulomata are also distinguished etiologically from the other forms, because they are the result of local or general infection by specific pathogenic micro-organisms. Hence they are also called chronic infectious diseases (Neisser), infection tumors (Klebs), infection granulomata (Ziegler).

Finally, it is to be noted that the term tumors, which is applied by pathological anatomy to the majority of formations here referred to, is not always suitable, either from a clinical or anatomical standpoint. Some constitute tumors in the main—for example, lipomata. Others form tumors only in certain stages of development, such as xanthoma, syphilides, mycosis fungoides. In other stages they

exhibit changes which approximate an inflammatory process, both clinically and histologically. In this event the term inflammatory new formations and tumors, employed by Virchow, still appears suitable.

It is well to bear in mind that the neoplasms of the skin are tissue formations which develop in the place and at the expense of the normal cutis, and have, in a sense, a destructive character, inasmuch as they destroy, to some extent, the normal cutis within their territory. At the same time the morbid forms belonging to this class constitute distinct pathological individualities so far as regards their cause, course, clinical and anatomical importance, and appearance.

These conditions will be shown in the following scheme, and the reader should not lose sight of the fact that the sarcomata, although closely related to the connective tissue new growths, on account of their clinical malignity are placed next to the carcinomata.

I. Connective-tissue New Growths.	Keloid.	Infection Neoplasms.	
	Cicatrix.		
	Molluscum fibrosum.		
	Xanthoma,		
	Lipoma.		
	Neuroma.		
	Myoma.		
	Osteoma.		
II. Vessel New Growths.	Angioma.		
	Lymphangioma.		
III. Granuloses.	Rhinoscleroma.	Malignant Neoplasms.	
	Lupus (vulgaris).		
	Tuberculosis cutis.		
	Scrofuloderma.		
	Lepra.		
	Syphiloderma.		
	Mycosis fungoides.		
	Lymphodermia perniciosa (mihi).	Benign Neoplasms.	
	Lymphoma.		
	Sarcoma.		
IV. Epithelial New Growths.	Carcinoma.		

GROUP I.

CONNECTIVE-TISSUE NEW FORMATIONS.

KELOID

is a scar-like, flat, stripe-shaped or nodular tumor which develops spontaneously, without inflammatory symptoms, in the substance of the cutis, and can only be moved with the latter. It continues without further change, or, in rare cases, disappears spontaneously.

It may appear as a flat, elevated, sharply defined, firmly elastic ridge, which looks as if pushed into the skin and projects two to three millimetres above surrounding surface ; it resembles an hypertrophic cicatrix very closely. In other cases it looks like a thick plate and more rarely has the shape of a round nodule. The central portion sometimes projects more prominently, while the edges slope sharply and send stellate prolongations in many directions. The keloid is white or a glistening red, the surface smooth, covered with thin, wrinkled epidermis, bald or covered with a few hairs, firmly elastic, and painful on pressure, sometimes spontaneously painful.

It may be single or very many may be present (as in De Amicis' case). They are found mainly on the sternum, arranged usually in two or more parallel rows, on the mammæ, the ears, face, shoulders and back, genitals, etc.

As to the development of keloid we have little positive knowledge. After it has matured it may continue to grow to a certain extent. Then it remains unchanged for life, or in rare cases disappears completely. Ulceration does not occur, only the most superficial excoriation.

It occurs in people of both sexes and at all times of life. In certain individuals very slight local irritations produce keloid, so that we may assume a special predisposition in some families and also in certain of the Ethiopian races (Von Tschudi). Thus, it develops around the perforation made in the lobe of the ear for ear rings, around leech bites and acne pustules, and I have examined a keloid, as large as a fist, in a negro whose body was said to have been covered with tumors of various sizes.

Many surgeons entertain the idea that keloid is not a true tumor, but is essentially a hypertrophic cicatrix. Others recognize, in addition to true spontaneous keloid, a false keloid which consists of cicatrices due to losses of substance (burns, syphilis, etc.), and has become nodular and tumor-like (cicatricial keloid, Dieberg ; syphilitic keloid, Wilks) ; finally, one form is known as Addison's keloid, which is identical with scleroderma (page 454).

Anatomical investigations show that there are three forms of tumor in this class : (1) true keloid (2) the hypertrophic cicatrix, and (3) the cicatricial keloid.

Fine sections of keloid show a whitish, dense, fibrous mass, with the fibres running parallel to the long axis of the tumor and the surface of the skin. It is inserted in the corium in such a way that normal layers of the latter, particularly the papillæ and rete pegs, remain intact above and below. In places the horizontal bundles are traversed by vertical ones. There are a few nuclei and nucleated spindle cells within the keloid body and around the vessels, which are compressed, as if by a sheath, by the dense bundles of fibres.

The cells are abundant in the younger parts of the keloid, around the vessels of the prolongations, so that it appears as if the fibres of the keloid were derived from spindle cells sheathing the vessels. The presence of the papillæ and rete cones also shows that the keloid develops in a previously intact corium and does not replace a loss of substance.

In an hypertrophic cicatrix, on the other hand, not a single papilla is found, because the development of the cicatrix presupposes the loss of the uppermost corium layers by suppuration, excision, etc. The hypertrophic cicatrix never extends beyond the original area of the loss of substance. Moreover, the connective-tissue fibres of the cicatrix are much looser and more irregular than in keloid.

In cicatricial keloid (Fig. 46) the papillæ are absent in the centre;

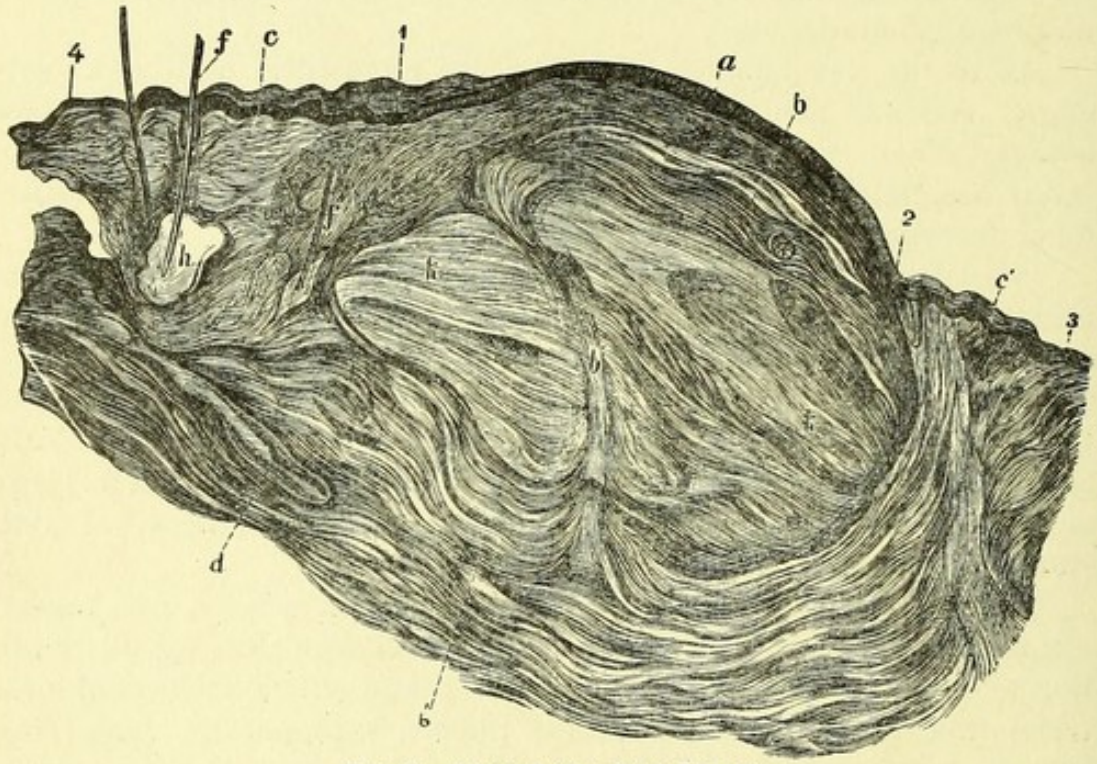

FIG. 46.—SECTION OF A SCAR KELOID.

1-2, cicatrix; the epidermis (*a*) stretched without rete pegs over the scar tissue (*b*), in which no papillæ appear, and whose superficial bundles interlace with the deeper (*b'*) by means of a process (*b''*). Enclosed between *b* and *b'*, the keloid (*k*), which extends to 2-3 and 1-4 in the uninvolved surrounding skin where the papillæ and rete pegs (*c c'*) are seen; *f*, hair shafts; *h*, sebaceous gland; *d*, a follicle enclosed in a sheath of scar tissue.

the irregular connective-tissue bundles of the cicatrix lie beneath a thin layer of epidermis. This is surrounded by the keloid, which is recognizable by the delicately arranged, dense bundles of fibres and the papillæ upon the surface. Here there is evidently a combination of cicatrix and keloid.

Babes did not find any papillæ in Schwimmer's case of multiple keloid. This was either a case of cicatricial keloid, or, if it was true keloid, it is possible that compression atrophy of the papillæ develops in time as the result of the growth of the tumor.

Keloid can be distinguished from hypertrophic cicatrix only by a microscopical examination, and hence the practical *diagnosis* is remarkably difficult. The more the normal structure of the skin, the papillæ, and the follicles is recognizable on the surface, the more certainly is the tumor a keloid and not an hypertrophic scar. Moreover, the diagnosis of keloid is favored by the location on certain parts of the body—for example, the sternum.

The *prognosis* of keloid is not favorable. Spontaneous involution is extremely rare, and we possess no curative measures. When removed by cauterization or excision it always returns.

Emplast. hydrargyri, applications of iodine and iodized glycerin, may be tried in order to favor absorption. Hardaway claims to have seen good effects from electrolysis. The pains, which are sometimes intolerable, must also be treated. We may employ emplastrum de Vigo upon which powdered opium has been strewn, cold, chloroform, subcutaneous injections of morphine, cocaine subcutaneously or in ointments. Quinine and arsenic may be administered when the pains exhibit periodicity.

CICATRIX.

A cicatrix of the skin is a new formation which develops at the site of a loss of substance and permanently replaces it. It has a whitish or reddish glistening, smooth, occasionally finely wrinkled or scaly, dry surface, and a firm consistence. The furrowing, pores and hairs, pigment, sebaceous and sweat glands which are peculiar to the normal skin are lacking in a cicatrix. It lies at the level of the surrounding skin—normal or flat cicatrix ; or it is somewhat depressed—atrophic cicatrix ; or it projects—hypertrophic cicatrix. In the latter event it appears as a linear, cylindrical, or nodular ridge or as net shaped, star-shaped, interlacing ridges. Finally, the cicatrix may be movable with the skin or adherent to the fascia and bones.

The *development* of the cicatrix presupposes the destruction of a connective-tissue part of the skin, the corium, or at least the papillary layer. In this regard we may divide skin diseases into two groups, those without and those with the formation of cicatrices. The first category includes all processes which consist of inflammation terminating in resolution, or which lead, at the most, to desquamation of the epidermis—for example, eczema, erysipelas, superficial dermatitis, pemphigus, herpes zoster, mild variola. A cicatrix develops exceptionally in these conditions, but only as the result of local accidents which cause destruction of the connective tissue. The secondary category includes those tissue destructions which are due to mechanical (scratching, contusion) and chemico-dynamic (burns, congelation, cauterization) influences, and the diseases which

entail extensive necrobiosis (gangrene) or suppurative destruction—for example, lupus, gummatous syphilis, scrofula, suppurative dermatitis.

The development of a cicatrix presents two phases : (1) the formation of granulations, and (2) the formation of epidermis. Both may occur in a normal or abnormal manner.

As soon as the demarcation of the necrotic from the healthy tissue has taken place, the latter exhibits inflammatory infiltration and suppuration (granulation) ; and as soon as the necrotic tissues have been detached, a granulating and suppurating wound is exposed. The granulations are bright red, at first coarsely granular and firm, later finely granular, velvety, and soft to the touch.

The granulations contain an upper, non-vascular, pyogenic layer consisting of pus, and a lower, plasmatic layer which is rich in vessels (Thiersch). The pus is derived partly from the granulation tissue (wandering corpuscles) and its blood vessels (white blood globules), partly from the proliferation of the uppermost tissue elements. The (lower) principal part of the granulations is young connective tissue which has been produced from the elements of the old adjacent tissues. The latter have become productive as the result of hyperæmia and increased supply of plasma. This so-called granulation tissue is typical of many varieties of new formation. It consists of a finely granular or fibrillated intercellular substance (in which numerous oval and spindle-shaped cells with large nuclei are embedded), and new-formed vessels which give a warty, papillary form to the tissue by the production of superficial vascular loops. The new vessels are formed, in part, directly from the old vessels, whose walls hollow out or proliferate in the shape of solid, later hollow, plugs (Meyer, Weber) ; in part they develop independently from cells which are arranged in rows and coalesce with one another (Rokitansky) ; or simple intercellular spaces enter into communication with the preformed blood vessels (Weber, Lehmann) ; or, finally, there is an endogenous development of blood corpuscles in cells and cavities—*i.e.*, a repetition, in a measure, of the embryonal process (Rokitansky, Weber, Billroth, Stricker, Klein).

Beginning in the deepest layers, the granulation tissue is converted gradually into connective tissue. On account of the diminished hyperæmia the immigration and proliferation of the cells become less active. Some of the latter increase in stability. They are converted into connective-tissue fibrillæ by juxtaposition and sending out processes, and the intercellular substance undergoes a corresponding chemical and morphological change. With increasing organization the fibres approach one another, the intercellular and interfibrillar spaces become narrower, the fibres shrink and retract the adjacent integument, so that the area of the wound diminishes.

When the granulations reach the level of the adjacent skin the formation of a new epidermis covering begins. With the diminution of hyperæmia the growth of the granulations ceases, at first at the edge of the wound, and a thin little skin pushes forward from the latter. As the vessels shine through, this has a bluish-red color. Advancing steadily toward the centre, it finally covers the wound, and cicatrization is complete.

Clinical observation appears to demonstrate, almost without question, that the new epidermis is produced by the old epidermis cells of the edges. Even when isolated, epidermic islets appear in the centre of the field of granulations. They may also be derived from old epidermis—viz., from the epidermic lining of some remnants of the sebaceous and sweat glands.

The assumption that epidermis is derived only from epidermis also corresponds to the results of special investigations. After producing losses of substance in the corneal epithelium, Heiberg found that the epithelial cells at the edges sent out processes which were detached as new cells. In the physiological regeneration of the epidermis Lott demonstrated the constriction of buds from the basal, so-called foot cells of the rete, and that in pathological epithelial proliferation the principal part is played by processes of fission in the pre-existing cells. The karyokinesis observed in this process has already been discussed (page 429). In addition wandering cells may also be converted, in part, into epidermis (Biesiadecki, Pagenstecher).

Cicatrization may be abnormal because the granulations develop slowly or in poor quality, so that they become smooth, very slightly papillated, dry, dropsical or hæmorrhagic. They may proliferate above the normal level of the skin (proud flesh). Such granulations are either excessively tender or excessively insensible. The formation of epidermis may also be delayed abnormally.

The causes of such obstacles to cicatrization are either constitutional (anæmia, scurvy, dropsy) or local (mechanical injuries and circulatory disturbances, as in varicose veins of the legs). They may also be due to excessive size of the wound, so that the peripheral cicatrix retracts and compresses the vessels supplying the central portions.

The fully developed cicatrix consists of an irregular connective-tissue network rich in vessels and nerves. Hair follicles, sebaceous and sweat glands, and papillæ are absent, together with the corresponding rete pegs. The polyhedral rete cells, consisting of several layers and a thin layer of horny cells, pass in a single plane across the surface of the cicatrix. Young cicatrices are pigmented, succulent, and rich in cells. They contain many blood vessels and hence have a bright-red color. Cold and mechanical stasis are apt to produce hæmorrhage and destruction of the epidermis. With advancing age

they become shorter, dry, hard, and white. On transverse section they exhibit many obliterated vessels filled with granules, sclerotic connective tissue with a few small corpuscles and narrow meshes.

The shape and size of a cicatrix depend upon the original loss of substance, and this is the result of a number of factors. For example, the scars of an ulcerated, serpiginous syphilide will be white and firm (older) in the centre, while the periphery will be younger, red, pigmented. But this is true only in a general way, and it is erroneous to assume the existence of characteristic cicatrices. The cicatrices due to zoster may be exactly like those of syphilis, and the variegated scars after a burn or a gangrenous bubo are exactly like those after a sulphuric-acid burn. Only a probable diagnosis of the cause of the cicatrix may be made, particularly because its special configuration may be due to other than the immediate causal factor. For example, all scars whose development is often interrupted become uneven and nodular. In some individuals the developed cicatrix undergoes subsequent hypertrophy. Finally, treatment during cicatrization exerts great influence on the quality of the final product.

In addition to the deformity, cicatrices produce traction of the adjacent integument. According to their location, they produce fixation and deformity of the joints, ectropium of the eyelids, stenosis of the openings into the visceral cavities, etc. They are often annoying on account of their spontaneous painfulness and itching, and their protracted vulnerability and tendency to inflammation.

The object of *treatment* is the production of a thin, flat, and movable cicatrix, and it must begin with the development of granulations. When the process runs a normal course any of the ordinary surgical methods is indicated. When it runs an abnormal course we may be called upon to employ the measures described in detail in the chapter on burns (page 275), such as frequent cauterization with the solid stick, ointments of acetate of copper, nitrate of silver, compress and bandage, etc. When the surface of the wound is very large and there is delay in the formation of integument over the middle portions, we may resort to Reverdin's method of transplantation. Little pieces are cut with scissors from the healthy skin of the same or of another individual, divided into parts five to ten millimetres in size, placed upon the granulating wound at a moderate distance from one another, and then kept in position by means of adhesive plaster. This is removed at the end of five to six days. Some pieces then appear shrivelled and fall off; others are firmly adherent. Even the latter lose their old epidermis in part, but at the end of ten to twelve days they produce bluish islets of epidermis, which soon coalesce with adjacent ones. According to Thiersch, it is better to transplant large superficial pieces of skin which have

been removed from healthy parts of the skin by means of a large, broad, and flat blade.

The deformities, fixation of joints, etc., which are due to cicatricial retraction, are treated by simple excision, plastic operations, or forced stretching of the nerves. Hypertrophic cicatrices may be lessened in size by incisions made parallel to the surface; the bleeding surface is then cauterized with the solid stick, and careful attention then paid to the development of the new epidermis. Softening and gradual flattening of callous, rigid cicatrices may be effected by protracted warm baths, by pressure with adhesive plaster or emplastrum hydrargyri, and, finally, by the artificial production of inflammation in the cicatricial tissue. This causes renewed vegetation of the fixed tissue elements, and leads to absorption along newly opened blood vessels and lymphatics. This may be done by repeated cauterization with concentrated solutions of nitrate of silver, methodical applications of iodized glycerin, or electrolysis.

Neuralgic affections of cicatrices are treated by emollient or narcotic applications, like the analogous conditions of keloid, or they require excision of the sensory nerves.

LECTURE XXXIX.

MOLLUSCUM FIBROSUM.

THIS is also known as molluscum simplex s. pendulum (Willan), molluscum non-contagiosum (Bateman), fibroma molluscum (Virchow). It consists of broad or pedunculated tumors. covered with normal skin, usually distinctly defined and of a uniform doughy or firmer consistence. They vary in size from a thickening or projection as large as a pea or bean, barely perceptible by palpation beneath the skin, to tumors as large as a nut, a fist, or a child's head.

The integument over the smaller tumors is pale, over the larger ones bluish red, traversed by enlarged vessels, and, as a rule, tense and free from follicles. In many cases, however, the sebaceous glands are filled with plugs of sebum and dilated, or even in a condition like atheromatous degeneration. No opening leads into the interior of the tumors. They usually have a uniform doughy or hard consistence, but they may be lobulated and vary from that uniformity of structure.

As a rule they are present in large numbers, sometimes even several hundred, in various stages of development. They are situated on the face, scalp, trunk, eyelids, genitalia, nates, etc.

They not only produce deformity by their number and size, but may prove a mechanical obstruction to the function of joints, to vision (the upper lid may hang down in the form of a thick lobe and cover the eye). When the tumors are very large the tension of the skin occasionally produces inflammation and gangrene.

Molluscum fibrosum consists of gelatinoid connective tissue which becomes fibrous in the course of time. According to Rokitansky it starts from the deeper meshes of the corium; according to Fagge and Howse, from the connective-tissue wall of the hair follicle; according to Virchow (with whose view I concur), from the connective-tissue layer around the fat lobules; according to Recklinghausen and Garré, from the nerve sheaths, but this undoubtedly obtains only in a special form of fibromata (neurofibromata, page 468). The new formation then pushes the skin before it and grows into nodular, lobulated, pendulous tumors. At the apex of the tumor it is intimately connected with the skin, the fibres of the former passing into

those of the corium. Otherwise the connection is loose, so that the tumor is easily enucleated.

In the larger and older nodules the inner part consists, *anatomically*, of young gelatinoid, the peripheral part of fibrous connective tissue, so that the histological character coincides with that of elephantiasis arabum. The pedicle contains one or more large vessels, as does the firm nodular extremity beneath the skin—*i.e.*, the part from which the tumor has started. The glands and follicles of the integument covering the tumor are partly normal, partly distorted, shrunken, and degenerated, according to the degree of tension. Numerous comedones and sebaceous-gland tumors (molluscum contagiosum of Bateman) are often found upon and between the tumors of molluscum fibrosum, in addition to numerous larger and smaller pigment patches (lentigo, ephelides), and even extensive pigment nævi.

Some tumors are absorbed spontaneously, and an apparently empty bag-shaped appendage then remains. In one case we found it hanging like a mane from the left forehead and temple over the eye and neck down to the shoulders. But these appendages always contain a part of the molluscum, as is evident from the impossibility of separating the folds.

The majority of tumors continue unchanged at a certain stage. The process always begins in earliest childhood. In the absence of other *causes* the disease was attributed to an hereditary predisposition, which was shown, in Virchow's case, by the occurrence of the molluscum in three generations. Hebra agreed in this opinion and also emphasized the fact that all the patients exhibit mental and physical degeneration.

Nævi mollusciformes et pigmentosi, which are either single or confined to one region of the body, may also grow into lobulated and apron-like tumors. Anatomically they are closely allied to molluscum fibrosum. But the characteristics previously mentioned and the general diffusion, in addition to the absence of other symptoms peculiar to the nævi, enable us to make a clinical *diagnosis*.

The *prognosis* is unfavorable because we are unable to produce absorption of the tumors. Although the general condition is not materially affected, it is to be remembered that marasmus or tuberculosis finally develops in certain cases.

Treatment consists simply in the removal of some of the more annoying tumors by means of excision, the galvano-cautery, or elastic ligature.

XANTHOMA.

Syn. xanthelasma (Wilson), vitiligoidea (Addison and Gull). This term is applied to circumscribed flat patches of a straw or citron color, which look like a simple discoloration, or to firm papules and

nodules of the same tint. They are most frequent on the eyelids, and are found more rarely on the rest of the face and on the body.

According to the original paper by Addison and Gull, there are two forms of the disease, viz., *xanthoma planum* and *xanthoma tuberosum*.

Xanthoma planum forms straw-colored or citron-yellow patches, which may attain the size of a finger nail or may be even larger. They are either uniform or composed of little patches, flat or slightly prominent at the edges. The integument is perfectly smooth and soft; a slight burning or pain is rarely felt. When grasped between the fingers the fold of skin feels perfectly normal. The patches are found mainly on the eyelids, usually quite symmetrical and nearer to the inner angle of the eye, more rarely on adjacent parts of the cheeks, and still more rarely upon the nose, ears, and lateral parts of the cheek and neck. They may also be observed upon the mucous membrane of the mouth, gums, cheek, and palate.

Xanthoma tuberosum (papular and nodular xanthoma, *xanthome en tumeur* of Besnier, xanthoma tuberculosum and tuberosum of Chambard) occurs as white or yellowish-white papules, looking like milium or wheat grains, either isolated or aggregated in stripes and patches. Occasionally they form roundish or elongated nodules, from the size of a pea to that of a nut, which may project four millimetres above the level of the skin. Their surface is covered with smooth epidermis of a yellowish-white color or red at the base. The smaller ones have a slightly greater consistence than the normal skin, while the larger ones are very firm, like fibromata, and are quite painful on pressure. These are less frequent upon the eyelids, somewhat more common upon the cheeks, but are most numerous upon the joints (fingers, toes, elbows, and knees), palms of the hands, and soles of the feet. They even occur upon the scalp, penis, trunk, the limbs (over the tendons), and also upon the previously mentioned parts of the mucous membrane, the trachea, larger bronchi, labia, and vagina. In Lehzen and Knaus' case Leube made a diagnosis of xanthoma of the endocardium, and this was found at the autopsy, in addition to a xanthoma-like proliferation in the aortic walls as far as the subclavian, in the coronary arteries, and in the capsule of the spleen.

Xanthoma planum et tuberosum may occur in combination upon the same individual. So far as known the cutaneous lesions undergo no change. At the most a few nodules may flatten or even disappear. However, we have observed an acute development of numerous xanthoma nodules which disappeared spontaneously at the end of several months or years. This occurred in cases uncomplicated with diabetes, the latter complication having been frequently observed in the last few years.

Anatomically both forms of xanthoma show a deposit in the upper corium, so that when the nodules are not very large the upper part of the papillary body is still intact. Histologically they consist of a new formation of connective tissue with a deposit of fat and fatty degeneration.

There is no doubt that xanthoma is sometimes mistaken for plaque-like milium granules, which may be closely aggregated upon the eyelids and surrounding parts, and may resemble a xanthoma. This occurred in one of my patients and in a case reported by Touton. When the nodules were nicked, little epithelioidal balls could be squeezed out ; this is not possible in xanthoma. When the latter is incised the cut surface has a more or less uniform yellow color, but nothing can be expressed with the exception of blood and serum. These conditions furnish the *diagnostic* difference between milium and xanthoma.

Recent investigations have supplied interesting details concerning the histological conditions. De Vincentiis and Touton have demonstrated large, swollen cells, corresponding to connective-tissue corpuscles, which appeared to be filled with fat granules (xanthoma cells). Both writers regard them as essential parts of xanthoma. De Vincentiis believes they are proliferated endothelial cells of the connective tissue undergoing fatty metamorphosis (endothelioma adiposum), while Touton regards them as proliferated embryonal plasma cells (in Waldeyer's sense) which subsequently undergo proliferation and fatty metamorphosis. The latter view is supported by Köbner's case, in which " xanthoma mollusciforme s. pendulum " developed from nævi pigmentosi et mollusciformes—*i.e.*, from the remains of embryonal tissues. I still consider the new formation of connective tissue as the main element in this pathological process, and that the deposit of fat in the cells and between the fibrillæ, with maintenance of the vitality of the elements, as a further characteristic of the process which impresses the stamp of xanthoma on the new formation. The yellow color depends simply upon the deposit of fat. This deposit of fat may predominate in certain parts ; in others there may also be a deposit of round and spindle cells, and in the larger and older nodules the connective tissue may become firm.

Nothing definite is known concerning the causation of this remarkable condition. It has often been attributed to hepatic affections, because jaundice has been observed in more than half the cases. The relation between the two conditions cannot be explained, but their frequent coincidence cannot be regarded as an accidental occurrence. It seems probable to me, especially in view of Murchison's statements, that the same process of nodular development may also attack the liver and thus give rise to jaundice. We are not

justified in denying, as Carry does, the existence of jaundice, and in attributing the general yellow color of the skin to a simple discoloration of the lower rete (xanthodermia).

Eichhoff observed xanthodermia in a child of two months whose great-grandfather had suffered from the same disease. Eichhoff, Church, and Carini speak of an hereditary predisposition. Chambard believes that the disease is due to a special diathesis (xanthomatosis)—*i.e.*, a tendency to the formation of these tumors with a tendency to the deposit of fat. The exciting cause may be hepatic disease in which, according to Putain and Quinquaud, considerable fat circulates in the blood in an unoxidized condition. It may also be due to the presence of sugar in the blood. Xanthoma has been observed in diabetics in a number of cases. This view appears to me to be worthy of further study. Another question which is undecided is that of the identity of so called "lichen diabeticus" or *xanthoma diabeticum* (Hutchinson) or glycosurique (Besnier) with ordinary xanthoma or xanthoma ictericum. All observers report that the xanthoma of diabetes develops acutely and almost always disappears spontaneously in a few months. Moreover, the nodules are softer and less yellow than in true xanthoma. Isolated xanthoma planum of the eyelids is a purely local formation.

The *diagnosis* of xanthoma is easily made. In regard to *prognosis*, it is to be remembered that the disease continues unchanged for years, but that spontaneous resolution has been observed in several cases. The hepatic affections and jaundice which are associated with xanthoma are sometimes temporary, in other cases they are persistent. The prognosis of xanthoma diabeticum is favorable.

Xanthoma can only be cured by excision or scraping with the sharp spoon, and even then relapses may occur. Max Schütz states that he has seen xanthoma planum of the eyelids grow pale under the application of collodion (ten per cent). Besnier claims that he has obtained rapid resolution of the nodules by means of the internal administration of phosphorus, followed by turpentine.

We may here mention *fibromata*, *lipomata*, and *neuromata*, which form isolated or multiple tumors of the skin and subcutaneous cellular tissue. The *neuromata* are composed essentially of connective tissue (neurofibroma of Recklinghausen) and are connected with the nerves in various ways (adherent to the sheaths, separating the nerve fibres, or connected in an unknown manner as in Duhring's cutaneous nodules). In some cases a true new formation of nerves and plexiform neuromata have been demonstrated, mainly in combination with elephantiasis or nævi (page 468). They are characterized clinically by extreme tenderness on pressure and by spontaneous paroxysmal neuralgias.

The very rare *dermatomyomata* are closely allied to the forms just discussed. They occur on parts of the skin which contain well-developed muscles, such as the scrotum and the vicinity of the nipples. They grow from pre-existing mucular elements and form large tumors. In a woman of sixty years Besnier observed them upon the trunk and upper limbs, in the shape of numerous smooth, slightly elevated, red, firm patches and nodules from the size of a lentil to that of a pea. They developed within a few months without subjective symptoms. The anatomical examination by Balzer showed that they consisted mainly of a network of smooth muscle fibres, with vessels and nerves. Besnier believes that they started from the connective tissue (liomyomata). A similar structure of the nodules was found in one of my cases, which was examined by Lukasiewicz. In this case the nodules were so sensitive that the slightest touch, even the friction of the underclothing, produced the most violent pain and reflex twitchings. They were situated, to the number of fifty, upon the legs, particularly the anterior surface, and a few were situated on the thighs. Similar cases were reported by Arnozan and Vaillard, Hess and Jadassohn. In all these cases the nodules had developed gradually, and some had subsequently undergone spontaneous absorption. They are derived from the organic muscle fibres of the skin, the arrectores pilorum, and the arterioles. Chambard and Gouilloud have described a tumor under the term "myome xanthomateux."

Salzer, Jr., has described an *osteoma cutis* from Billroth's clinic. A bony plate was found embedded in the scalp and perforated by the underlying follicles and glands.

LECTURE XL.

ANGIOMATA.

NEOPLASMS OF THE BLOOD VESSELS AND LYMPHATICS.

THESE include pathological new formations of the skin which consist entirely or in great part of dilated and newly formed vessels, either blood or lymphatic.

New formations of blood vessels (angiomata proper) show their anatomical structure by their clinical appearance. The color and configuration conform to those of vessels injected with blood, and they disappear temporarily under the pressure of the finger. According to their varying characteristics, they may be distinguished as: (1) telangiectasis, (2) nævus vascularis, (3) angio-elephantiasis, (4) tumor cavernosus.

Telangiectases are dilatations and new formations of capillaries and the finest cutaneous vessels, which have developed during extra-uterine life. They appear as light-red to dark-violet patches or papules, which grow pale under the pressure of the finger, and attain the size of a poppy seed or a lentil. They may form a diffuse redness or marbling of the uppermost layers of the skin, traversed by little branches of vessels.

The absence of increased heat, pain, and swelling, and the persistence of redness and vascular ramifications, prevent confusion with hyperæmic and inflammatory redness. Telangiectases develop idiopathically, rarely in childhood, as a rule during middle life. The most frequent sites are the delicate integument of the eyelids, alæ nasi, cheeks, ears, and neck, rarely the backs of the hands and other parts of the body. Turgescent vascular nodules may extend to the vermilion border of the lips and the buccal mucous membrane, and these bleed profusely when injured.

Secondary telangiectases develop in and around cicatrices which have obliterated some of the capillaries, inasmuch as the remainder dilate. They often occupy permanently those parts which have been free from the morbid process proper—for example, in lupus erythematosus, lupus, etc. They also develop over tumors as the result of pressure and traction, and in certain forms of atrophy of the skin. The telangiectases of acne rosacea in the face, and the peripheral

cyanoses due to central obstructions to circulation (endothoracic tumors, pleurisy, valvular lesions), are symptomatic forms.

The course of telangiectases changes very little. A few vessels disappear, others make their appearance.

Nævus vascularis is an abnormal vascularization of the general integument, which is either congenital or acquired during the first months of life. It appears as a diffuse patch, like ink poured into the skin, of a light-red, bluish-red, or leaden-gray color, with or without slight turgescence of the skin (nævus flammæus, tâche de feu, nævus simplex, angioma simplex) ; or in the shape of flat, elevated, tumor-like, turgescent, sometimes even pulsating tumors of smooth or nodular surface (angioma prominens, nævus tuberosus, angioma cavernosum, fungus hæmatodes, venous telangiectasis, erectile vascular tumor, aneurisma spongiosum, etc.). They all grow pale under compression. At the surface they are apparently sharply defined, but deeper the nævi spread into the surrounding parts by fine ramifications. Their most frequent site is the head ; more rarely they are found on the trunk and limbs, and also on the genitalia. They may be found singly or in large numbers, and are often combined locally with pigmented or warty patches (angioma pigmentosum et verrucosum). Those located in the face become turgescent in stasis of the blood (coughing, crying), and grow pale under the opposite conditions (syncope).

The course of nævi varies greatly. The majority grow to a certain size during the first months or years of life and then persist during life, or they do not change in either direction until an advanced age Others disappear spontaneously during the first years of life by gradual obliteration of the vessels, leaving whitish, shining, cicacial or pigmented patches. This is particularly true of the diffuse nævus flammæus and the flat angioma simplex. Larger, turgescent nævi (fungus hæmatodes, venous telangiectasis) usually grow rapidly in circumference and depth, or after they have remained stationary for several years. These penetrate to the adjacent mucous membrane of the cheek, tongue, conjunctiva, etc., and into the underlying tissues, the fat lobules, muscles, nerve sheaths, and bones. The latter are eroded and pushed aside. During this excessive growth they change their anatomical and clinical characteristics. They develop into extensive tumors, which may, for example, occupy an entire upper limb, the thigh, the back. They form firm, granular and nodular, or soft, spongy, compressible tumors, which enlarge spontaneously in a dependent position and collapse on elevation ; they usually exhibit a lobulated structure. Their pressure and growth cause degeneration of the muscles and nerves, and atrophy of the bones. Painful neuromata form an essential part or even the starting point in some cases. They develop not infrequently from

the subcutaneous tissue, starting from the adipose layer or nerve sheaths, and then spread gradually into the cutis (*angio-elephantiasis*, angioma elephantiaticum s. lipomatodes s. neuroticum).

The *anatomical* relations of angiomata may be very simple or very complicated. I will confine myself to a few remarks, referring the reader to the well-known text books on pathological anatomy. The flat and simple angiomata are situated in the papillary and upper corium layer, and the proportion of arterial and venous vessels varies, perhaps, according to the light or dark shade of the redness. In the larger and deeper angioma these vascular differences do not obtain. In the simple as well as the complicated angiomata we find fully developed old and new formed vessels, in addition to manifold sinuosity and communication with one another. Even in the simplest there is a new formation of connective tissue in and around the adventitia. This is the essential basis of the histological complications. The proliferation and outgrowth of the vessels appear to be least in nævus flammæus. A further development is represented by that form in which primary and secondary offshoots and numerous communicating and convoluted vessels are found. According to Billroth, the occurrence of small lobules in such angiomata is due to the fact that the vascular tracts of the sweat glands, hair bulbs, sebaceous glands, and fat lobules are affected separately. In all, the convolutions of vessels constitute the principal element. In the lobulated angiomata the enormously dilated and communicating vessels (whose convolutions look, on transverse section, like a sieve-shaped perforation of the tissues) are supplemented by excessive proliferation of young, gelatinoid connective tissue, and exceptionally of neuromata and fat lobules. According to these different conditions, the tumor is called angioma elephantiaticum or elephantiasis angiomatosa, angioma lipomatodes or neuroticum.

The *tumor cavernosus* proper is distinguished from the other angiomata by the firm connective tissue which surrounds it on all sides. As in the corpus cavernosum, this sends primary and secondary septa into the interior and divides it into numerous larger and smaller cavities. These spaces contain blood and communicate partly with large vessels of the tumor, partly with vessels of the surrounding parts. Some writers believe that the tumor cavernosus grows from the walls of old cutis vessels ; others regard it as independent of the vessels, which only enter into communication with it at a later period. Rindfleisch attributes it to a new formation of connective tissue which develops along the vessels, retracts and secondarily dilates the vessels.

We are entirely in the dark concerning the *causes* of nævi vasculares, apart from the few which may be attributed to fœtal conditions.

Nævi constitute a serious affection on account of the deformity to which they give rise and the danger of their unlimited growth. Turgescent nævi may also become annoying or dangerous by the facility with which they may be injured, the most extensive cavernous angiomata by the lancinating, neuralgic pains and the complication with inflammation and gangrene. The latter complications may also produce suppuration or shrinking and obliteration of the nævus.

The *prognosis* concerning the importance and the course of nævi must be given very cautiously. Flat tumors give usually a more favorable forecast than prominent ones. But there are no signs which will enable us to determine whether the nævus will remain stationary or disappear spontaneously, or whether it will take on an excessive development.

Treatment depends upon the degree and dimensions of the angioma. Telangiectases are destroyed in the manner described in the chapter on acne rosacea (page 379). Flat or small warty and pigmented nævi may be removed with the sharp spoon. Other methods for destruction of turgescent angiomata are intended to produce gradual or rapid coagulation of the contents of the vessels and secondary obliteration of the vessels themselves. This is done by local compression and the application of cold, ligature of some of the larger afferent vessels, injection of ferrum sesquichloride, manganese chloride, cantharidin, etc. (this gives rise occasionally to gangrene and even fatal pyæmia), and by electrolysis. In order to produce shrinkage from inflammation, or destruction from suppuration or cauterization, in small fungous nævi, we may recommend the inoculation of vaccine lymph, cauterization with potash and other caustics (among which fuming nitric acid is the best), galvano-cautery, the Paquelin cautery, the application of tartar emetic ointment (tart. stibiat. 0.75, empl. adhæsivi 5.0), sublimated collodion, etc. Finally, pedunculated tumors may be removed by ligature and excision, and in large tumors of the limbs amputation of the latter may be necessary.

LYMPHANGIOMA CUTIS.

This neoplasm is of rare occurrence and consists, in the main, of a connective-tissue network lined with endothelium and containing lymph. The anatomical relations to the lymphatics and blood vessels, and the clinical appearances, are so manifold that almost every case possesses special peculiarities. Even a single tumor exhibits marked differences in different parts.

These tumors fall chiefly in the domain of surgery and pathological anatomy.

We will refer merely to the already mentioned vesicular lymph varices (lymphangiectases, simple lymphangioma of Wegner), which

vary from the size of a pin's head to that of a pea. They occur in elephantiasis acquisita, and occasionally burst and produce lymphorrhœa. There are also small ectasiæ of the lymphatics, which develop upon congenital mollusciform (connective-tissue) growths of the cutis and subcutis, and in time develop into pendulous, spongy tumors (elephantiasis lymphangiectodes). Finally, lymphangioma may be connected with subcutaneous, cavernous, congenital, or acquired tumors of the lymphatics, such as macrochylia, or with cystoid cavities of the lymphatic vessels and glands, due to stasis. In these cases the cutaneous lymph varices are merely outlets of the cystoid cavities.

LYMPHANGIOMA TUBEROSUM MULTIPLEX

is a term applied by me to a tumor formed by the lymphatic vessels, which belongs exclusively to the cutis. It appeared in the shape of many hundred rounded or elongated, brownish-red, moderately firm papules in the cutis. Their size equalled that of a lentil or less, and the surface was smooth. They were situated on the trunk and neck of a woman of thirty-two years, had lasted from childhood, and had recently increased in number.

The appearance was similar to that of papular syphilis, but resolution, scaling, or depression could not be detected.

A papule which was excised and examined by Biesiadecki and myself showed numerous round and roundish openings and elongated, sharply defined fissures (Fig. 47, I.), which, under higher powers, proved to be the finest lymphatics, enormously dilated, with thickened walls, and lined with endothelium (hence the term lymphangioma multiplex tuberosum). The connective tissue of the corium was dense and sclerotic, especially in the immediate vicinity of the spaces referred to, and with very narrow and scanty lymph fissures. The part of the papule which was examined by Biesiadecki contained normal sebaceous glands, hair follicles, and sweat glands. In the cavernous lymph tumors described by various writers the neoplastic and dilated lymphatics had proliferated from the subcutaneous tissue into the cutis, but in our case they occupied the cutis alone.

Pospelow, Van Harlinger, Lesser, and Henken have subsequently published cases which they regard as identical with our lymphangioma tuberosum multiplex.

Hoggan, Besnier, Jacquet, Török, and Fordyce have recently denied the lymphangiomatous character of the papules described by me. Besnier, in particular, who has seen several analogous cases, regards them all as sweat-gland tumors (idradenoma) or as "cystadenoms épithéliaux bénins," produced by proliferation of embryonal epithelial remains which had been left over, and by the subsequent formation of coils and cysts.

It cannot be denied that these cases resemble mine very closely, but whether they are identical I am unable to decide. The epithelial cones and coils which were visible in the preparations of Darier, Jacquet, and Fordyce could not be found in my case. The connective tissue of the corium was sclerotic, the sweat glands, sebaceous glands, and hair follicles were normal. The latter circumstance is opposed to the view that my case is identical with those of Besnier and others. Biesiadecki called attention to the similarity of the

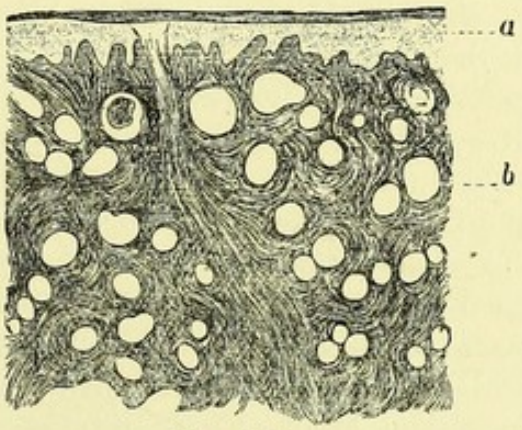

FIG. 47, I.

FIG. 47, I.—SECTION THROUGH A NODULE OF LYMPHANGIOMA TUBEROSUM
a, epidermis and papillary layer ; *b*, cutis (sieve-like, loculated appearance).

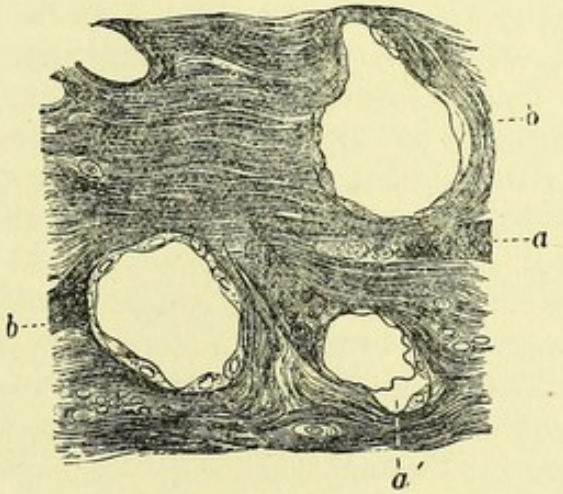

FIG. 47, II.

FIG. 47, II.—A PORTION OF THE SAME SECTION (OF THE CORIUM) GREATLY MAGNIFIED.
a, longitudinal ; *a'*, oblique: *b*. cross-sections of lymph vessels, with walls lined by nucleated endothelium.

structure to that of adenoma of the sweat glands. and nevertheless he regarded it as lymphangioma.

In recent years a large number of writers have described pale, brownish-red, sometimes vesicle-like papules, with a smooth surface and firm, elastic consistence, which were either congenital or developed in early childhood. They varied in size from a pinhead to

that of a pea, were located on the integument of the face, neck, side of the chest, thighs, nates, labia majora, either singly or in dense little groups (resembling warty nævi). On account of their cystoid or cavernous structure and their lymphoid contents they have been called lymphangiomata with more or less positiveness. The published cases have been recently reviewed by A. Schmidt, Noyes, and Török. The "lymphatic warts of children" described by Colcott Fox occur upon the fingers, but only a few of the vesicle-like dilatations contain lymph, while the majority contain blood and are covered with callous, warty epidermis. These are included by Noyes and Török among the angiomata. The large part of the other cases, however, are classed as true lymphangiomata (lymphangioma capillare varicosum cutis). These structures occupy only the uppermost layers of the corium, unlike the lymphangioma tuberosum and the cavernous lymphangioma, which usually infiltrate the deeper cutis and leave the papillary layer free above them. Another interpretation is made by Besnier, who regards cases which others have called lymphangioma capillare partly as hæmatangioma, partly as cavernous lymphangioma. On the whole it appears to us that combinations of angiomata and lymphangiomata, angiectases and lymphangiectases, occur in many places. In view of the intimate relations between the blood vessels and lymphatics, this is not surprising. According as one or the other predominates, and also, perhaps, according as one or another papule is the object of examination, the tumor may be classified as lymphangioma, hæmangioma, or a mixed form.

The essential feature is that these "lymphangiomata" are congenital structures, with or without coincident papillary and epidermal hyperplasia; that they are due to proliferation and new formation of the walls of the vessels, perhaps of connective-tissue endothelium or embryonal-cell agglomerations.

The *prognosis* is favorable. The tumors persist for an indefinite period without increasing in size or undergoing any serious histological changes. Some papules may undergo spontaneous absorption.

The *treatment* consists simply of surgical removal (by excision, cauterization with the Paquelin, electropuncture, or chemicals).

LECTURE XLI.

RHINOSCLEROMA.

THIS new formation, first described in 1870 by Hebra and myself, possesses great practical importance on account of its destructive tendency.

It always attacks the nose and its immediate vicinity, including the adjacent mucous membrane. It occurs in the shape of flat or slightly raised, sharply defined, isolated or coalescing, very hard and elastic plates, ridges, or nodules which are painful on pressure. They are situated on the skin or mucous membrane, the nasal septum, alæ nasi, and adjacent part of the upper lip. From the free border of some of these plates the finger can be pushed beneath them and they may be lifted from their base. They infiltrate the cutis completely, and hence are only movable with the latter.

Their surface has a normal color or is light to dark brownish red, traversed by a few vessels, glossy, free from hair and follicles, like a keloid or an hypertrophic cicatrix, with a smooth or finely wrinkled epidermis. The surrounding integument does not exhibit the slightest abnormal appearance.

It begins either upon one ala nasi or the nasal septum. Without any inflammatory symptoms, the septum cutaneum, one or both alæ nasi grow thick and hard. After the lapse of months the wings of the nose appear to be pushed outward, so that their contour is broadened as in a pug nose. The cutaneous parts are as rigid and immovable as if cast in plaster, and they cannot be brought in apposition by pressure. With the increased thickening the tissues also grow inward, so that the nares are narrowed and finally occluded. As a rule the hard infiltration spreads, with a sharp border, to the upper lip or around the mouth (Billroth's case), causing stenosis of the latter; later the gums are affected. Still more frequently it extends backward along the nasal cavity to the posterior nares and to the velum. In one case we saw thickening over the left temporal bone and the cheek thrown into hard ridges, so that the bridge of the nose was relatively depressed.

Pick observed, in addition to rhinoscleroma of the nose, an analogous hardness of the integument of both auditory canals. I have

observed a similar condition at the lower circumference of one auditory meatus.

Although the process lasts many years, there is never ulceration or other retrograde metamorphosis ; at the most there is superficial excoriation in places, and very rarely a diminution of hardness. If a piece is excised (the ease with which the knife enters the rigid mass is astonishing) the remaining portion does not undergo suppuration. The raw surface is soon covered with a thin crust and with epidermis. The excised portion is soon regenerated, and this usually happens even when the entire formation is removed. As a rule the mucous membrane of the buccal cavity, gums, and hard palate is not involved until a late period. The gums are thrown into ridges, the teeth are loosened and fall out, and the alveoli undergo atrophy. The velum, circumference of the posterior nares, and the palatal arch are affected at an early period, sometimes even primarily.

The palatal arch forms a cicatricial, glistening, at first normally colored, later whitish, almost rigid band. In the course of time the uvula gradually shrivels and may disappear entirely, and the arch of the palate presents the most peculiar shapes and adhesions to the posterior wall of the pharynx. Superficial erosions, from the size of a lentil to that of a penny, develop in the velum and uvula. They resemble syphilitic ulcers, but are not painful, exhibit no zone of infiltration and inflammation, and are never converted into deep ulcers. Perforation of the palate often occurs in some inexplicable manner.

We have also observed an extension of the process to the epiglottis and larynx, with fixation and upward curvature of the rigid epiglottis, stenosis glottidis, associated in one case with aphonia, and often with symptoms of suffocation and epileptoid attacks. In a few cases the laryngeal mucous membrane appears to have been the primary or sole site of the rhinoscleroma, and, according to Chiari, the process sometimes extends to the trachea.

From recent investigations it seems probable that some of the laryngeal stenoses, which have been described under various names, belong to rhinoscleroma.

Apart from the deformity of the face, the subjective symptoms include pain on pressure, the extreme interference with respiration due to occlusion of the nares, the functional disturbances due to stenosis of the mouth and the laryngeal introitus, and the final danger of death from suffocation. In some of my cases this was relieved by tracheotomy ; in one case it occurred suddenly. The general health is unaffected. Dacryocystitis sometimes occurs as the result of occlusion of the lachrymo-nasal duct.

Rhinoscleroma of the nasal tissues is often mistaken for syphilitic gumma. The liability to error is increased by the changes in the mucous membrane of the pharynx and velum. If we consider the

remarkable hardness of the tissues, which Hebra compares to that of ivory, the entire absence of softening and ulceration, the typical localization and course, the indifference to injury, excision, and antisyphilitic medication, the difference between this condition and syphilis becomes patent. It is easier to mistake some forms of rhinoscleroma for keloid, rhinophyma, or nodular (infiltrated) epithelioma. This is only possible, as a matter of course, when the rhinoscleroma is slightly developed.

Anatomically, I demonstrated a small-cell, dense infiltration of the corium and papillæ as the essential element of rhinoscleroma (Fig. 48), and believed that it was most closely related to small-cell sarcoma. Geber and Mikulicz interpreted the same findings as a chronic inflammation. In advanced cases they found that some of the round cells were converted into spindle cells and connective tissue, while other round cells were absorbed. Billroth, who found newly formed true bone in a shrunken part of the tissue, entertains the same opinion. In a piece excised from the region of the upper lip we found a transition of the scleroma tissue into true cartilage (Fig. 48). Finally, all these transitions into connective tissue, cartilage, and bone were found by O. Chiari in a rhinoscleroma of the tracheal mucous membrane. He believes, however, that the cartilage and bone had developed from perichondritic and periosteal ecchondroses and exostoses; the latter were branching, so that, in the majority of sections, the connection with the perichondrium and periosteum was not visible. This does not obtain in regard to my findings (Fig. 49) of cartilage in the midst of scleroma of the upper lip. In view of all these circumstances, I prefer to regard rhinoscleroma as a neoplasm, and believe that it is allied to the sarcomata.

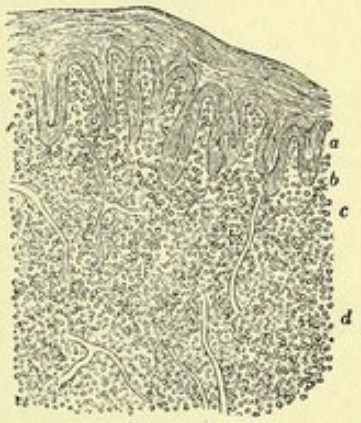

FIG. 48.—SECTION OF RHINOSCLEROMA IN THE EARLY STAGE (FROM THE LEFT ALA NASI).

a, epidermis; *b*, rete; *c*, papillæ; *d*, corium, both the last infiltrated with round cells and traversed by dilated vessels.

The disease was formerly regarded as syphilitic in character, but this opinion is not founded on clinical facts.

The suspicion of an infectious origin has been rendered very probable by the discovery of the constant occurrence of specific bacilli in rhinoscleroma (Frisch, Pelizzari, etc.), and these findings have been confirmed by Lustgarten, myself, and others.

With certain staining methods Cornil and Alvaraez found the bacilli surrounded by ovoid capsules of a "colloid" substance, by the confluence of which the round, coccus-like, or oval and rod-shaped structures appeared to be arranged in twos and fours. They saw

them only in the lymphatic spaces, never within the cells. Frisch stated that they are situated in the large, peculiarly swollen rhinoscleroma cells, which were first described by Mikulicz ; and Paltauf and Eiselsberg also found them in protoplasmic masses which, in

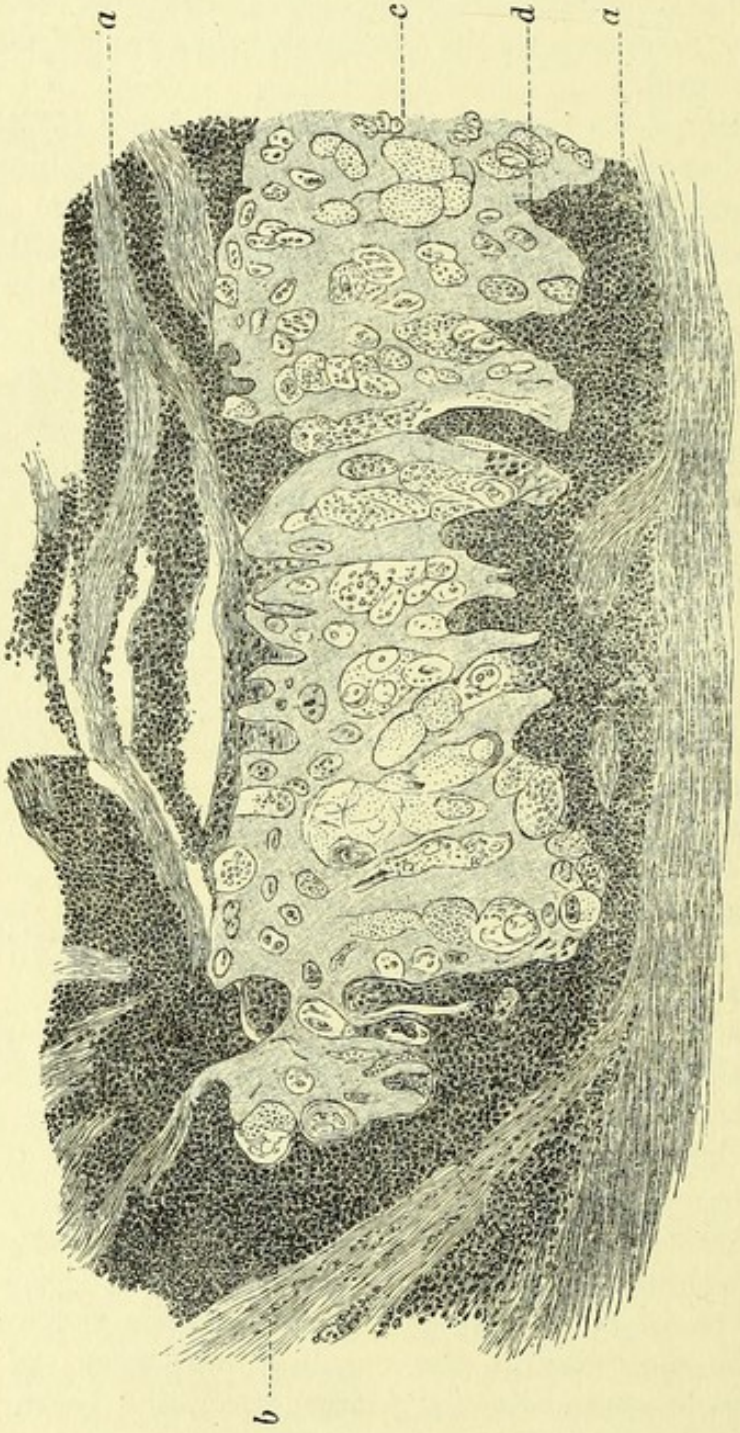

Fig. 49.—Rhinoscleroma. Nodule from the Upper Lip.

a, small-cell infiltration (as in Fig. 48) converted, at *b*, into connective tissue In the centre (*c*), cartilage (matrix and proliferating corpuscles), into which (*d*) the cell infiltration projects.

their opinion, correspond to those cells or to degenerated nuclei which occupy their place. These authors described them as bacilli two to three millimetres long, or as ovoid, almost round, encapsulated cocci united chiefly into diplococci.

Frisch and Barduzzi have made pure cultures. These may be obtained in twelve to twenty four hours at a temperature of 36°–38° C. According to the most recent investigations, however, the rhinoscleroma bacilli cannot be distinguished morphologically or by cultures from Friedländer's pneumococci or from the micro-organisms found by Klamann and Thost in ozæna and simple catarrhal affections of the nose. Pawlowsky claims to have proved the identity of the tissue changes produced in animals, in his experiments, by the scleroma bacillus with those found in scleroma. From their experiments on animals Paltauf and Eiselsberg think that the pneumococci are more virulent than the cocci of rhinoscleroma.

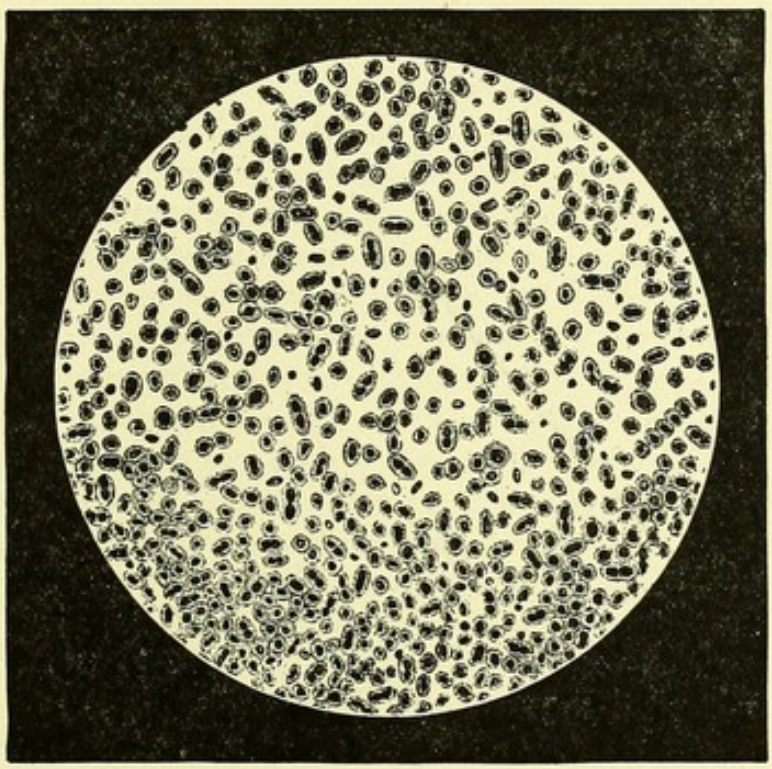

FIG. 50.—RHINOSCLEROMA BACILLI (ENCAPSULATED).

Culture from new tissue, at 36°–38° C., on agar-agar, after twenty-four hours. Stained with methyl violet aqueous solution.

If we take into consideration the localization of rhinoscleroma, its constant development in the nares and upon the nasal mucous membrane, the frequent primary affection of the nasal, pharyngeal, and laryngeal mucous membrane, and, finally, the fact that inflammatory retractile processes take place in these parts as the result of chronic catarrhs, we may assume some relation, which is not yet well defined, between the bacilli of the catarrhs and those of rhinoscleroma, and between the latter bacilli and the rhinoscleroma itself. All this, however, is purely hypothetical. The experiments made by myself, and by Paltauf and Eiselsberg, in which living

rhinoscleroma tissue was inoculated into a dog's snout, proved entirely negative.

I have seen nearly fifty cases of the disease, which occurred in equal number in both sexes, between the ages of fifteen and forty years. The patients belonged to all classes and presented no other specific dyscrasia. The general condition remained entirely unaffected.

The *prognosis* of this neoplasm is unfavorable, because it grows unchecked, relapses even after repeated extirpation, and endangers life by the above-mentioned functional disturbances and attacks of suffocation. In a few cases I observed no relapse for a number of years after extirpation, but I am not convinced of their permanent recovery.

A curative method of *treatment* has not yet been found. The only thing to be done is extirpation of the neoplasm, either in part or in its entirety. When the nares are narrowed we may first attempt to secure dilatation by the introduction of catgut, laminaria, drainage tubes, or compressed sponge. In more severe cases we may excise portions. or cauterize with caustic potash, pyrogallic ointment (ten per cent), Paquelin or galvano-cautery, or may perform total extirpation with subsequent plastic operation. This must be repeated from time to time, as the neoplasm soon returns. Parenchymatous injections of Fowler's solution, osmic acid, salicylic acid, have not proved effective in our hands, and occasionally have produced partial gangrene. The results of such treatment seem to be somewhat more favorable against nodules of the mucous membrane, of the palate and the larynx.

LECTURE XLII.

LUPUS.

Syn. Lupus vulgaris, dartre rougeante, esthiomène. This is a chronic disease of the skin and adjacent mucous membrane, characterized by red or reddish-brown papules which are embedded deeply in the corium. When the papules undergo involution they cause desquamation, ulceration, and cicatricial atrophy of the skin.

The term lupus was first applied to "eating" ulcers. Later it was employed exclusively for ulcers of the legs. It is only since the end of the last century that the word was applied by Willan-Bateman to certain nodules of the face which may also lead to ulceration.

The *development* of lupus is always attended by the formation of bright-red or brownish-red papules, as large as a millet seed or pinhead, and deeply embedded in the corium. They grow pale under the pressure of the finger, but do not disappear. Such papules appear during the entire existence of the lupus, so that they may be called its primary efflorescence.

The different papules pass through a tolerably regular course, whose symptoms constitute those various forms which have been called lupus tumidus, exfoliativus, exulcerans, hypertrophicus, papillaris, sclerosus. These are only various stages of the same process. The individual papules grow very slowly, so that, in the course of weeks and months, they finally project a little above the level of the skin, become accessible to the touch, and are found to have a moderately firm or tough consistence. The union of a number of adjacent papules leads to the formation of nodules which may attain the size of a pea or larger (*lupus tumidus*).

After the papules and nodules have lasted several weeks, at the height of their development retrogression begins. Sometimes the papules sink in because their elements are absorbed (fatty degeneration), and the formerly tense, shining epidermis becomes wrinkled and desquamates (*lupus exfoliativus*).

After absorption is complete a shallow depression is left, over which the skin looks like a cicatrix. Again, the exfoliation is attended by superficial, suppurative destruction and ulceration (*lupus exulcerans*).

The lupus ulcers are round or roundish, with flat, red, flabby borders and a red, granulating, easily bleeding base. They pain very little or not at all. They secrete a moderate amount of pus, which sometimes dries into extensive crusts. A part of the lupus nodule disappears by purulent destruction, another part by absorption, and finally new skin is produced after partial restoration of the loss of substance by means of granulations.

The formation of granulations is often interrupted by intercurrent hæmorrhages, renewed destruction of tissues as the result of secondary inflammation, and an after-growth of lupus papules in the vicinity. The granulations then develop into large, papillary structures, and even grow, in part, into permanent, warty, horny excrescences (*lupus papillaris, verrucosus*).

In other cases the frequently recurring inflammations of the tissues surrounding an ulcerating focus of lupus may lead to local elephantiasic and sclerotic hypertrophy of the connective tissue (*lupus sclerosus* of the French writers).

The arrangement of the lupus also gives rise to differences. When the papules are arranged irregularly we speak of *lupus disseminatus s. discretus.* In *lupus serpiginosus* the new papules develop, at the periphery of the old focus, in curved lines which unite with adjacent ones into larger arcs of a circle.

When it extends deeply the lupous infiltration may reach the subcutaneous connective tissue or the cartilage of the ala nasi and the concha of the ear. It has also been said that lupus may proliferate through the fasciæ to the muscles, periosteum, and bones. I believe, however, that this is due to complicating inflammation and the formation of peculiar inflammatory products analogous to those of scrofula or tuberculosis.

Weber, Hebra, Esmarch, and others have made the interesting observation that a very severe form of carcinoma may develop upon lupus, secondary to the atypical outgrowth of the rete cones which occur in some forms of lupus.

Upon the mucous membrane of the nose, gums, palate, velum, tongue, and larynx the fresh lupus papules are rarely recognizable as brownish-red, easily bleeding, firm prominences, from the size of a pinhead to that of a millet seed, and covered in places with silver-gray, exfoliating epithelium. Later they coalesce into larger plaques with a rough surface, dull-gray epithelial covering, with deep, painful fissures or with a red, finely granular, raw surface. Finally, these also terminate in cicatricial retraction.

The symptoms of the development and course of the disease are modified in various ways by its localization. Lupus of the nose is the most frequent variety ; the papules develop primarily upon the integument of the alæ nasi, whence they gradually extend to the root

of the nose. In the course of years the wings of the nose gradually shrivel from the edges, so that the cutaneous part of the organ appears cicatricial and contracted ; or a part, and finally the entire cutaneous part, of the nose, together with the cartilage, is completely destroyed in the process of ulceration. During the latter process the nose occasionally appears to be enlarged, as the result of the mass of deposited crusts of papillary granulations. It is only when these are removed that a large part of the alæ nasi is found to have been lost.

Upon the nasal mucous membrane lupus generally develops by extension from the skin, but sometimes primarily. For years the ulceration and crustation may simulate the appearance of chronic eczema or polypi until they are made clear by the retraction and destruction, the perforation of the septum, or the extension to the general integument. I have never seen destruction of the bony parts of the nose or of the vomer as the result of lupus.

Lupus is found very often upon the rest of the face, on the cheek and jaw, and thence extending to the neck, where it usually assumes the shape of lupus serpiginosus ; or on the lobe of the ear, which may become enormously swollen by lupus tumidus et papillaris, or, on the other hand, may finally shrivel up. Lupus may also appear on the integument of the external auditory meatus, the lips, and the eyelids.

In these localities it is apt to be complicated with chronic enlargement and suppuration of the submaxillary and parotid glands, and to simulate ordinary scrofula. In rare cases lupus occurs primarily on the conjunctiva and extends to the bulb and cornea ; in this locality it is usually an extension from the cheek. The conjunctiva appears covered with dark reddish-brown, dry, coarse granules, as in trachoma ; in some places smooth, shining, shrunken, the cornea presenting a granular, pannus-like deposit which interferes with sight to a marked degree. Complication or increase of the inflammation sometimes results in perforation of the cornea, with termination in staphyloma and analogous conditions, or, as we have seen in one case, in panophthalmitis and loss of the eye.

Lupus is rarely primary on the forehead and scalp ; it usually occurs there as an extension from adjacent parts. Upon the mucous membrane of the bucco-pharyngeal cavity and larynx lupus occurs quite often (rarely on the tongue) as an extension of the disease of the lips, but often in separate foci. In rare cases it occurs primarily in these localities. Sponginess and hæmorrhage from the gums and the mucous membrane of the hard palate, falling out of the teeth, gray opacity of the epithelium of the tongue, nodular, moderately firm thickenings mingled with cicatricial, depressed retractions of the tongue, ulceration and retraction of the velum, are among the symp-

toms of lupus in this region. Upon the epiglottis, true vocal cords, and the rest of the larynx, especially its posterior wall, lupus gives rise at first to hoarseness, later to retraction, ulceration, chronic inflammation and formation of papillary excrescences, complicating laryngeal perichondritis and chondritis, and stenosis. These changes very rarely lead directly to death, as in Breda's case. Chiari and Riehl found, among seventy cases of lupus, six in which it was localized in the larynx.

Lupus sometimes occurs very extensively upon the trunk. Upon the nates it is apt to develop in a papillary, warty, and elephantiasic form. I have seen the disease situated exclusively upon the penis and scrotum.

The upper and lower limbs are a frequent site (especially in the serpiginous form), upon the flexor and extensor surfaces, as well as the palms of the hands and the soles of the feet.

After it has lasted for a number of years, lupus of the limbs gives rise to very complicated tissue changes and deformities, in addition to the fixation of the joints (pseudoankylosis) due to cicatricial contraction of the skin.

As a result of the frequently repeated inflammatory phenomena, dermatitis, lymphangitis, erysipelas, phlebitis, which accompany the formation of the lupus nodules, gumma-like nodes (*gommes scrophuleuses*, Besnier) as large as a hazelnut, which soon soften and are converted into flabby ulcers, develop along the thickened lymphatics, or we find periostitis, caries and necrosis of certain phalanges, metacarpal and metatarsal bones. As a further result of these conditions, deformity and retraction of some fingers, and the deformity of the hands, legs, and feet known as elephantiasis arabum consecutiva, occur (page 470). The affected hand appears thickened in the cutis as well as the bones, broad, misshapen, with the fingers separated like claws.

The changes are most marked in the lower limbs. The leg is thickened; the skin, with the subcutaneous tissue, soft parts, and bones, converted into a rigid mass and cannot be raised into folds. The surface is unevenly nodular, here and there tense and shining; in other places covered with thick, dirty callosities of epidermis; in others, with warty excrescences and spinous outgrowths. The foot is shapeless, broad, often fixed in a varus position, the toes widened and their boundaries effaced with the exception of furrow-like indications. Lupus nodules may continue to grow for years in the changed skin, so that they are still distinctly recognizable (Fig. 53), or the production of lupus ceases and the elephantiasic degeneration, as such, continues. In such cases the cause of the elephantiasis can only be diagnosed after a ripe experience, unless well-defined lupus is found in other parts of the body.

Lupus may be present in all of the regions mentioned, in the same individual. In one of our patients, a married woman aged forty years, disseminated and serpiginous lupus was present on the face, the trunk (from the neck to the nates), the legs, and forearms.

Lupus begins in early childhood at the age of three to six years. In the most favorable event it appears upon a circumscribed part of the body, exhibits exacerbations during a period of four to ten years, and then disappears permanently, leaving cicatricial atrophy. A new lupus focus may appear at the end of many years or a relapse occur in the old place. In this way it may appear as if the lupus had developed primarily at the age of forty years, although the eruption is merely a relapse.

In more frequent and less favorable cases, an eruption of lupus which appears in early childhood continues to spread for fifteen to twenty years and persists until an advanced age is reached. The most unfavorable cases are those in which the lupus appears, from the start or within a few years, upon several parts of the body—for example, in the face and on the limbs or upon several parts of the trunk. Such a case will never recover, on account of the almost insuperable obstacle to the vigorous treatment of lupus in so many places at the same time.

The *prognosis* is more favorable the more isolated and the smaller the dimensions of the lupus foci, while one which is multiple from the start, especially of the serpiginous variety, runs a less favorable course, inasmuch as relapses always appear at the edges of the old focus and in this way the latter enlarges very rapidly. The prognosis is also unfavorable in so far as relapses are to be dreaded even when temporary complete recovery has been effected. But if the relapses are persistently treated final success may be looked for. Even when the lupus is quite extensive it has no demonstrable injurious influence upon the general condition. Patients suffering from almost general lupus may present a healthy appearance and all the functions may act normally. Women affected in this way may give birth to healthy children.

In this country (Austria) lupus constitutes sixty-six per cent of all skin diseases, and is somewhat more frequent in women than in men; it occurs in the limbs in twenty per cent of all the cases. It almost always begins in early childhood, rarely before the third year, latest at the period of puberty, and in rare cases at a later period. Relapses may occur until the age of seventy years. Its frequency and severity are identical among city and country people, among the poor and the well-to-do. Season, occupation, and diet have no influence on its development.

Among the special or direct *causes*, lupus has been attributed to syphilis, scrofula, and tuberculosis. After passing through various

changes of theory, lupus, scrofulosis, and tuberculosis are to-day regarded as identical and due to the same virus, the tubercle bacillus. They are all included under the term cutaneous tuberculosis (Leloir, "La Scrophulo-Tuberculose de la Peau," 1892).

Certain forms of lupus, especially the serpiginous, and particularly those localized on the limbs and the nose, have been attributed to hereditary syphilis. It was supposed to be a sort of modification of the parental syphilis, according to Ricord's notion that tertiary syphilis is not transmitted as such to the offspring, but is manifested by scrofula of the various tissues and the constitution. Such a connection has never been proven, and experience shows that syphilis in the parents has nothing to do with lupus. The similarity between lupus and ulcerative syphilides may lead to mistakes, but these are simply diagnostic errors. Lupus, as such, is not hereditary, nor is it infectious in the clinical sense. It is therefore surprising to me that Leloir has seen, among three hundred and twelve cases, eleven cases in which one of the parents had also suffered from lupus, and fifteen cases in which the disease appeared among brothers and sisters. It is still maintained occasionally that certain forms of lupus have a genetic relation to hereditary syphilis. But Hebra, I, and others have found lupus and recent syphilis on the same individual—*i.e.*, a patient suffering for years from lupus acquired syphilis (papules and roseola). This would be inexplicable if lupus were syphilitic in character.

There is more reason for believing that lupus is allied to scrofula and tuberculosis. Until very recently the term scrofula has not been associated with any sharply defined clinical notion. The term scrofulous diathesis was applied to a condition in which slight irritation of a part of the body is followed by an inflammation which outlasts its cause, often terminates in suppuration or caseation, and more rarely retains the form of a hyperplastic process. If we regard the existence of such inflammations and cheesy infiltrations in the glands, subcutaneous cellular tissue, and joints, with the well-known ulcers and the perilymphangitic gumma nodes, with the secondary waxy degeneration of the liver, spleen, and kidneys, and the various diseases of the eye (keratitis, conjunctivitis pustulosa) and skin (lichen scrofulosorum, acne cachecticorum)—if we regard these various conditions as an expression of scrofula, then it is true that they are found in a considerable proportion of lupus patients.

This coincidence of symptoms, however, is not sufficient reason for regarding the lupus as due to scrofula. Careful observation teaches that lupus is the primary process and that the scrofulous inflammations are secondary. On the other hand, the scrofula is entirely absent in many cases of lupus, or its manifestations appear at a very late period.

Clinical observation furnishes even fewer data for assuming a relation between lupus and tuberculosis, in so far as the lungs and internal organs are concerned. A coincidence of both diseases is not frequent, and much rarer than pulmonary tuberculosis in lupus erythematosus, lepra, and other chronic inflammatory and suppurative processes.

Still, on account of the coincidence of certain anatomical and bacteriological characteristics, it is now held that lupus, tuberculosis, and scrofulosis are essentially one and the same morbid process, viz., tuberculosis; that lupus and scrofulosis are merely different forms of tuberculosis, and that the former is to be regarded as local or cutaneous tuberculosis.

This doctrine was first founded, on an anatomical basis, upon the discovery in lupus tissue of giant cells and epithelioid cells. It was soon found, however, that giant cells are not characteristic of tubercle, that they are present in the most varied processes and can even be produced experimentally. The inoculations of lupus into the anterior chamber of the eye in rabbits, performed by Waldenburg, Cohnheim, and others, sometimes gave rise to tuberculosis of the iris and ciliary body, but failed to decide the question because they were performed in the prebacillary period.

Demme was the first to report the finding of the tubercle bacillus in six cases of lupus. Pfeiffer found them in a case of lupus conjunctivæ, Krause and Schuchard in two cases of lupus. Doutrelepont reported on eighteen cases. While other observers had found only a few bacilli in twenty to eighty sections, Doutrelepont found them in all sections, sometimes even in groups of ten to twelve. Koch reported their very scanty occurrence in four cases. He found them in giant cells, but only one rod in a cell. The first successful inoculations into the anterior chamber of the eye in rabbits were made by Schüller and Hueter. Positive results were also obtained by Cornil, Leloir, and Martin. In addition to tuberculosis of the iris and cornea in rabbits, they produced general tuberculosis by inoculating the peritoneum in guinea-pigs.

The presence of bacilli in lupus, although in small numbers, was also noted in our clinic. In a non-ulcerated lupus node from the cheek of one of our patients, Fürth found, in one of the cells of the rete, a group of eight bacilli.

For the majority of our colleagues these facts were sufficient to prove the identity of lupus with tuberculosis and scrofulosis. The adherents of this doctrine also utilized the statistics showing the coincidence of scrofulous and tubercular affections with lupus in a considerable proportion of cases. Many of the lupus patients finally die of tubercular infection of the internal organs (pulmonary tuberculosis or tubercular meningitis). It is also claimed that the transi-

tion of scrofula into true lupus, and of the latter into cutaneous tuberculosis, has been seen.

Nevertheless the attempted demonstration of the identity of scrofula, tuberculosis, and lupus has not yet proven such identity. Cases of "inoculation tuberculosis" are reported in constantly increasing numbers, but it seems to be regarded as immaterial that years may have elapsed between the assumed "inoculation" and the occurrence of the "tuberculosis of the skin."

No experimental proof has been offered, however, that characteristic lupus vulgaris can be produced by inoculation of tubercle bacilli. The inoculation of lupus tissue and of the bacilli derived from it has given rise to inflammatory products but not to lupus; nor has any clinical proof been found, unless we accept extremely careless statements.

This is less true concerning a few published cases, such as that of Besnier (lupus in a vaccination scar), Sachs (lupus after piercing the ear), Jadassohn (lupus around tattooing), Wolters (lupus nodules developed within a few weeks in the scar of a scalp wound, in a physician who was busily engaged in examining tuberculous sputa during the healing of the wound). These and other similar, although less carefully observed, cases might demonstrate the occurrence of clinical lupus from inoculation of tubercle bacilli. Although the introduction of tubercle bacilli into the wounded parts can only be regarded as probable, nevertheless these cases are worthy of note. Relying upon them, some writers (Jadassohn, Leloir) have felt justified in regarding every case of lupus as due to the inoculation of tubercle virus, and in denying the so-called spontaneous or hæmatogenous development of the disease.

In my opinion all the data which have been forthcoming are insufficient to prove positively the tubercular character of the three processes in question. The facts are in part capable of another interpretation, and in part they do not prove such identity. I am not shaken in this belief by the experience that the injection of Koch's lymph into lupus tissue produces an inflammatory reaction which is as prompt and severe as that produced by injection into tubercular pulmonary tissue. As we now know, there is an entire series of substances which exert a similar action upon lupus and other vascular tissues.

At all events, all clinicians feel the difficulty of obliterating the clear clinical symptomatology and boundaries of lupus, which runs such a sharply defined course. Hence they plead for the maintenance of lupus vulgaris as a special type of skin disease, though it be only regarded as a special form of cutaneous tuberculosis.

The clinical character of lupus remains typical, even if it lasts fifty years or more in the same individual. It is complicated, in the

manner previously described, by verrucosity, elephantiasis arabum, carcinoma, and inflammatory and suppurative processes of the subcutaneous tissues, grouped under the collective term scrofulosis. Yet the lupus never changes its true clinical type. Although it has been said that transitions of lupus into cutaneous tuberculosis are frequent, I maintain that such a transition into true tuberculosis of the skin never takes place. We recognize, however, a true tuberculosis of the skin, which also possesses well-defined clinical characteristics, and is distinguished from lupus as well as from other processes.

In *diagnosis* the essential differential feature is the character of the nodules, which are embedded in the cutis tissue and do not disappear under the pressure of the finger. Whenever the appearances are very complicated (confluent nodules or ulcers, with or without crusts) we should look for the primary efflorescences, which are found, as a rule, in the vicinity of a diffuse focus. The greatest difficulty arises, as a rule, in the diagnosis of lupus from syphilis nodosa serpiginosa et ulcerosa.

Apart from the character of the individual recent lupus nodules, much depends, in diagnosis, upon the condition of the lupus ulcers, their indolence, the flabbiness and vascularization of their base and edges, the abundant development of granulations, their slight tenderness. In addition, lupus nodules never spread so regularly from the centre to the periphery as do syphilitic nodules, hence they do not form kidney-shaped ulcers. A diagnosis can always be made after a certain length of time, because new lupus papules will appear at the end of two to four weeks. Furthermore, local and general antisyphilitic treatment (especially the use of emplastrum hydrargyri, which is so effective in syphilitic nodules) will be useless in lupus.

Finally, lupus runs a much slower course than syphilis, and in many years hardly produces as much destruction as ulcerative syphilis in a few weeks. In lupus the nose diminishes in size mainly from retraction, while in syphilis the individual parts are sharply separated from the healthy whole, and, furthermore, losses of bone (vomer, hard palate) do not occur in lupus. In the same way, for example, elephantiasic thickening of the leg, of the kind described, which is combined with the presence of papules, can only be regarded as lupus. Lupus alone lasts so many years in a papular form that it may lead to elephantiasis, while a papular syphilide lasts only months or a few years, and those forms of syphilis which may give rise to elephantiasis belong to the gummatous variety and form characteristic ulcers. Finally, it must not be forgotten that, however long the lupus may have lasted, relapses are always attended by the production of the characteristic nodules. If all these

circumstances are carefully considered, it will always be possible to distinguish lupus from syphilis, even in difficult cases.

Lupus erythematosus presents an entirely different appearance from lupus vulgaris (page 508), and I must decidedly oppose the attempt which has been made to assume a relationship between the two or the possibility of a transition from one to the other. The condition to which Leloir applies the term lupus vulgaris erythematoides is, according to his own description, a lupus vulgaris which appears atypical on account of the intense inflammatory infiltration of surrounding parts.

LECTURE XLIII.

LUPUS (*continued*).

ANATOMY—TREATMENT—SCROFULOSIS—TUBERCULOSIS OF THE SKIN.

In order to obtain a correct idea of the very complicated *anatomical* conditions found in lupus, it is necessary to examine recent nodules which are still deeply embedded. Upon sections of such a part of the skin (Fig. 51) we notice, under low powers, larger and smaller roundish (nest-shaped) masses of tissue embedded in the corium (lupus nodules). They are situated irregularly and at various depths in the corium, whose upper and papillary layer and rete appear normal. They contrast by their yellowish-red color and sharp

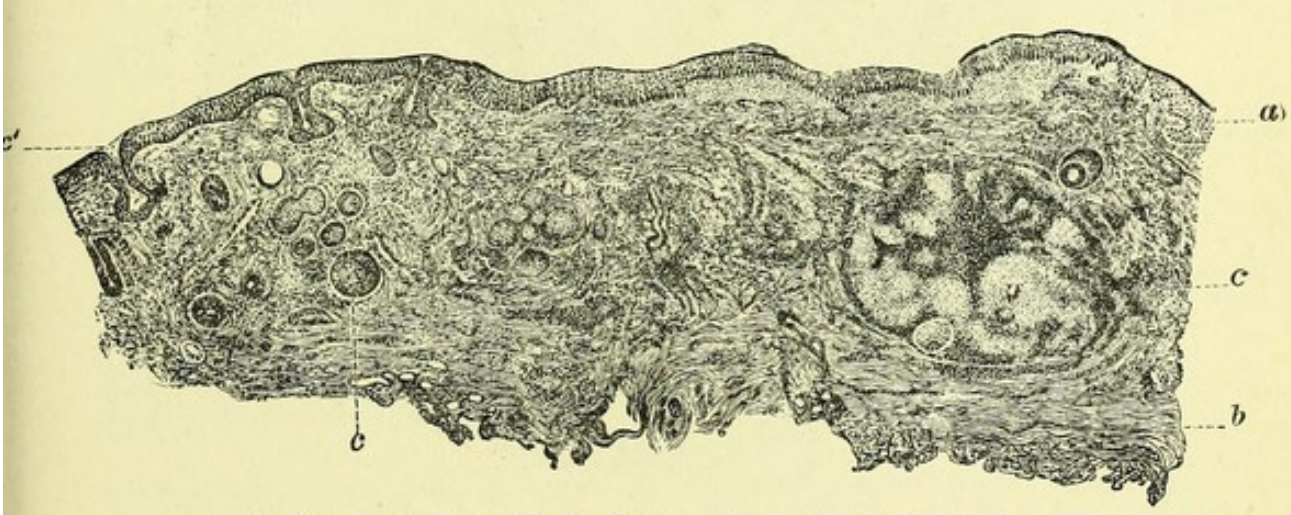

FIG. 51.—LUPUS OF THE NOSE (VERTICAL SECTION). (LOW POWER.)

a, epidermis; *b*, normal corium, scattered irregularly through which are small round and one large lupus nodule (*c*); *b*, overlying layer of corium, rete, and follicle (*d*) normal.

boundaries with the surrounding cutis tissue, especially when carmine staining is used (they stain less than the other tissues).

Under high powers the lupus nest (Fig. 52) is sharply defined from the healthy connective tissue, which surrounds it in dense bundles, and its structure is found to correspond to that of granulation tissue. It consists of a network of coarse fibres with finer ramifications and numerous large blood vessels, the spaces formed by the intersections of the large fibres containing cells with large, strongly refracting nuclei. The narrow meshes contain similar cells, and also

numerous smaller cells with sharply defined nuclei. In a few parts are found epithelioid cells, and also, according to Unna, the large "plasma cells" of Waldeyer, to which he ascribes a special importance in lupus. Jadassohn denies their histological importance in lupus, and refers to their occurrence in other inflammatory processes and their frequent absence in lupus. On shaking, the embedded small cells are apt to fall out, so that the network with its nodal cells alone remains. In some places the entire nest falls out, so that its place is occupied by a round hole (Fig. 51, *c'*).

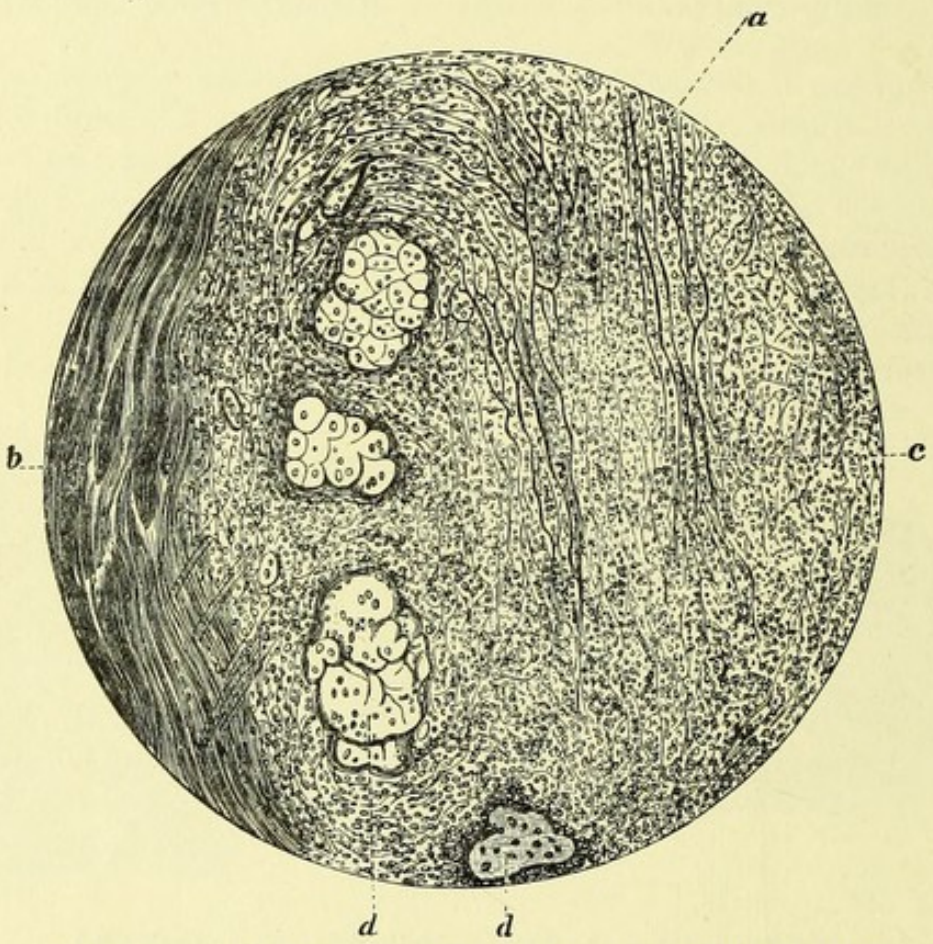

Fig 52.—Section of a Microscopic Lupus Nodule under High Power.
b, the same, surrounding healthy corium; *a*, reticulum with cell infiltration (*c*); *d*, giant cells.

The further development of the lupus tissue leads to very complicated changes. According to the most recent investigations, the blood vessels and lymphatics appear to produce the network and vessels of the lupus nodule by outgrowths from their protoplasmic walls and proliferation of their adventitia. Proliferating cells are also derived from the connective-tissue corpuscles and wandering elements of the inflamed stroma of the cutis.

The recent lupus nodule is therefore a vascular, actively proliferating growth. After it has lasted for some time resolution begins,

manifested first by the disappearance of the vascularization of the centre of the nodule and by necrobiosis of the cellular elements. Some cells are distended and enlarged, so that they have been compared to epithelial cells. The majority undergo cloudy swelling, no longer stain with carmine, disintegrate, and are collected into granular, crumbling heaps. In places there appear large, irregularly shaped, homogeneous or finely granular, protoplasmic masses with five to twenty or more oblong, shining nuclei (Fig. 52, *a*). These are the giant cells which were formerly regarded as peculiar to tubercle. It is now known that they occur in many other conditions, in gumma, sarcoma, and even in granulations, and that their presence in lupus nodules is not peculiar.

The main part of the lupus nodule is thus incapable of organization. After the retrograde metamorphosis of its elements, it under-

FIG. 53.—SECTION OF SKIN ATTACKED BY LUPUS HYPERTROPHICUS PAPILLARIS ET VERRUCOSUS. (LOW POWER.)

a, epidermis; *a'*, collection of horny cells; *b*, hypertrophied rete with papillæ, ten times their normal size; *d*, sclerotic and hypertrophic connective tissue of the elephantiasic corium, within which are seen lupus nodules (*c*), one of which (*c'*) has fallen out.

goes absorption or elimination (if situated superficially), and the inflamed tissue in which it is embedded then undergoes cicatricial retraction. In my opinion, however (Lang and Leloir appear to entertain the same idea), a part of the lupus tissue with its vessels and cells undergoes organization into young connective tissue, which retracts at a later period. In this way lupus appears to me to differ essentially, from a biological standpoint, from the nodules of syphilis and leprosy.

In addition to this course of certain of the nodules, others enlarge by a continuance of the new formation along the corium and papillary vessels, even as deep as the adipose layer, and coalesce with foci which have started from other centres. At the same time the interstitial connective tissue undergoes inflammatory infiltration,

the original arrangement (which resembles that of alveolar tissue) disappears, and an irregular, diffuse cellular infiltration of all layers of the skin is the result. After a long time this may also disappear completely, leaving cicatricial contraction of the skin and its glands. The inflammatory element of the infiltration supplies the basis for that hypertrophy of the connective tissue which develops during the course of years, particularly in the limbs, and which has already been described as elephantiasis arabum glabra et papillaris. Upon an hypertrophied and degenerated cutis of this kind, and in which the lupus may still persist, we may find, in places, enormously hypertrophied papillæ with correspondingly enlarged rete cones and colossal layers of horny cells (*lupus verrucosus s. cornutus*, lupus scléreux; see Fig. 53).

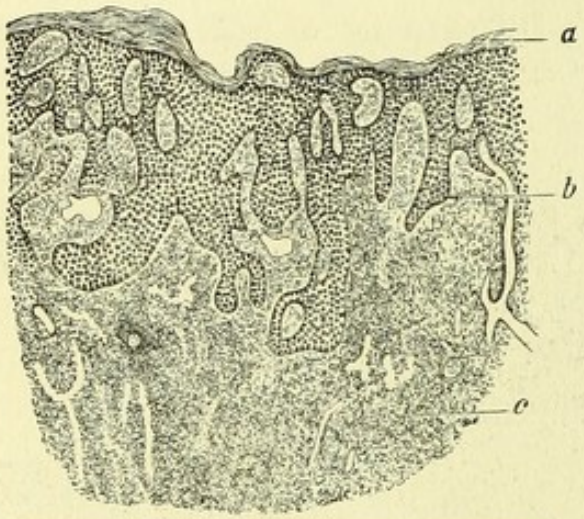

FIG. 54. ATYPICAL OUTGROWTH OF EPITHELIUM. SECTION FROM NATES.

a, epidermis; *c*, corium, diffusely infiltrated with lupus, into which the rete pegs (*b*) have pushed in the form of simple and branched cones.

Among the other elements of the cutis, the epithelial structures are implicated at a very early period. As soon as the lupous infiltration, which is originally superficial, has extended into the papillary layer, proliferation, cloudy swelling, and vacuolization begin in the rete cells. The boundary between the papillary and mucous layers is effaced by the extension of the lupous infiltration into the latter, and if the rete is cast off by suppuration or exfoliation the lupus nodule is laid bare (ulceration). The cells lining the sebaceous and sweat glands and the hair follicles also hypertrophy and degenerate as the result of cloudy swelling and early cornification; the hair follicles are obliterated after degeneration of the papilla, loosening and falling of the hair. After their excretory duct has shrivelled the acini of the sebaceous glands often remain filled with epidermis arranged like the layers of an onion (pearl bodies).

Another noteworthy form of epithelial hyperplasia is the outgrowth of the rete into the corium in the shape of simple and ramifying epithelial cones (Fig. 54), associated with similar outgrowths from the cells of the sheaths of the sweat glands and the hair roots. These form a network of epithelium, ramifying in all directions. As I showed in 1879, these form the histological basis for the development of epithelial cancer in recent or old lupus lesions.

In the majority of cases the combination of cancer with lupus is rapidly destructive, and in only a few cases was temporary improvement or permanent recovery secured. In my experience recovery occurred only when the base of the carcinoma was formed, not by loose, inflammatory, but by cicatricial, callous tissue, evidently because the latter offered more resistance to the advance of the epithelial processes.

In lupus of the mucous membrane (Fig. 55) the anatomical conditions are essentially the same, except as they are modified according to the difference of location.

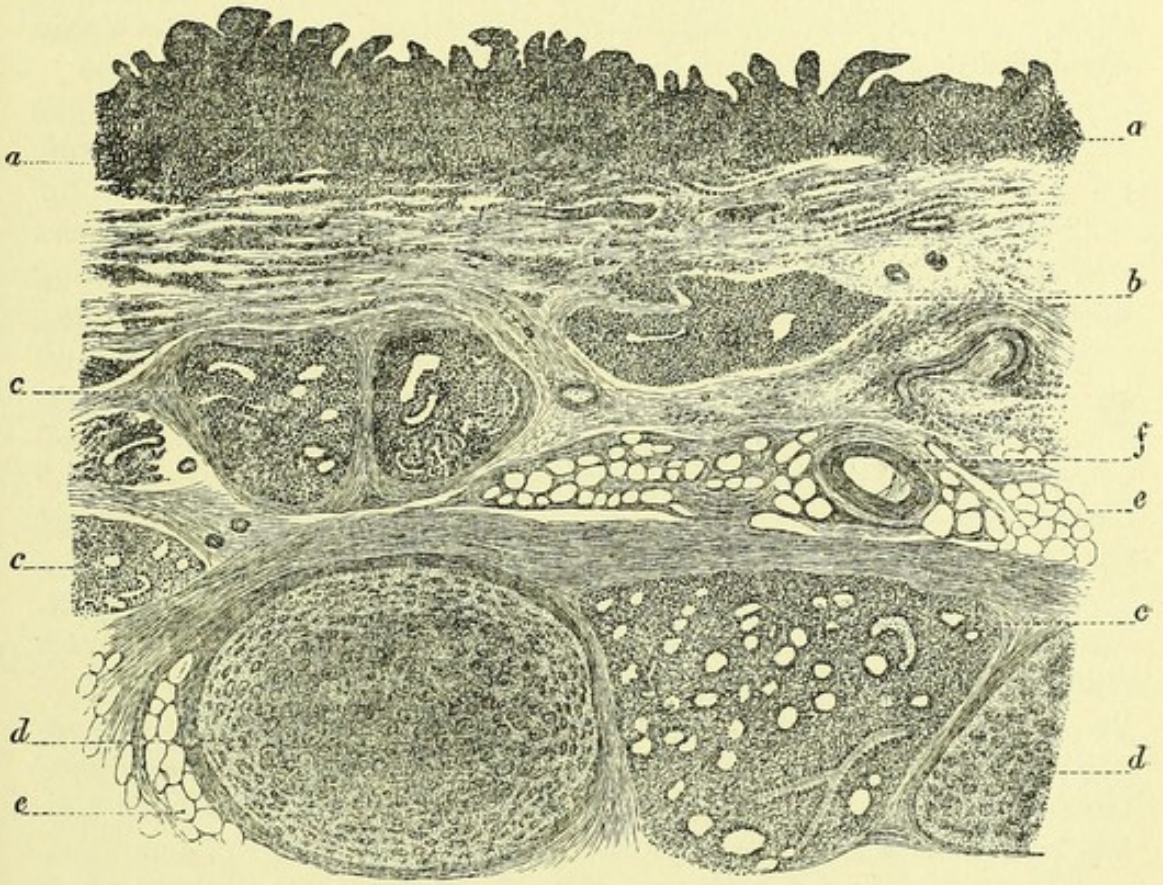

FIG. 55.—LUPUS OF THE MUCOUS MEMBRANE (EPIGLOTTIS).

a a, mucous membrane, showing cell infiltration and papillary outgrowths; *b*, lupus nodules; *c c c*, mucous glands infiltrated with lupus; *d d*, cartilage; *e e*, submucous fat; *f*, artery (cross-section), thickening of intima.

We now turn to the *treatment* of lupus. No internal remedies will cause resolution of existing lupus or prevent a relapse. Cod-liver oil, iron, arsenic, etc., are given in order to improve the general nutrition if the patient is scrofulous, anæmic, or poorly nourished.

In November, 1890, great expectations of the rapid and radical cure of lupus by means of Koch's tuberculin were awakened. The brown, clear fluid, when injected, in doses of 0.01 to 0.02 cubic centimetres, into any part of the body, gave rise, at the end of five to

eight hours, to violent general toxic symptoms (fever, vomiting, pains in the joints and abdomen, collapse, delirium, etc.) and to an acute inflammation of all lupus foci in the body, as well as of young and old lupus cicatrices. This was attended with redness, swelling, and extensive serous exudation, which detached the scales and crusts of the lupus. In twenty-four to thirty-six hours the inflammation had run its course and the lupus foci everywhere appeared flattened and pale. At intervals of several days the subcutaneous injections were repeated, in increasing doses, for several weeks.

Experience showed, however, that the inflammation-producing action of the tuberculin gradually grew weaker in the later injections and finally ceased, and that in the meantime the lupus again developed or even appeared in acute regional metastases.

We are therefore, in view of this failure, compelled to treat lupus by local measures. These include (1) mere adjuvants of treatment, (2) measures which directly destroy the lupus nodules.

The former category includes all fats, oils, ointments, plasters which are employed to macerate crusts and to cover suppurating wounds, or to macerate the lupus nodules themselves. They may be used in various ways. For example, compresses of cod-liver oil are useful in lupus tumidus which is partly covered with crusts; after one to two weeks' application the crusts are softened and fall off, and the nodules themselves are in a state to undergo more rapid destruction. Compresses of soft soap act in the same way, except that they have also a slight caustic effect.

The necessary maceration is generally effected by ung. simplex, ol. olivar, emplast saponat. salicylat., rubber cloths, and other bland remedies. The pressure exerted by the plasters also facilitates the removal of the inflammatory infiltration and swelling. I am unable to confirm the statement made by some writers that emplastrum hydrargyri causes very rapid disappearance of the lupus nodules. It merely acts like any other mild plaster.

The removal of lupus can only be effected by mechanical or caustic interference. The most rational and simple method would be that of excision of the entire process, followed immediately by transplantation according to Thiersch's method or the ordinary methods of plastic surgery. Unfortunately, the localization and extent of the foci are poorly adapted to this plan of treatment. In addition, experience shows that relapses may occur even in the transplanted flaps. Nevertheless this method merits more frequent use. It should be kept in mind, however, that a flat, thin cicatrix of the nose, eyelids, and face, obtained by prolonged treatment, causes less deformity than a plastic operation which is not brilliantly successful.

The mechanical treatment of lupus with the sharp spoon, accord-

ing to Volkmann, has met with well-deserved approbation. The lupus tissue is so soft and yielding that it can be easily scraped out, together with the adjacent infiltrated part of the integument. It is difficult to cause any injury in this way, inasmuch as the healthy skin offers great resistance to the entrance of the spoon.

This method is best adapted for large, confluent nodules and for flabby, diffusely infiltrated, ulcerated tissues. The hæmorrhage, which is always profuse during the scraping, ceases upon applying a tampon and compress and bandage. At the end of two or three days the gray deposit—*i.e.*, the layer of destroyed tissue which is still adherent—is detached, and healthy granulations appear forthwith.

Mechanical treatment also includes puncture and scarification with a sharp-pointed bistoury, with Veiel's instrument composed of several lancets, with Hebra's lance-headed punctor, or, better still, with Vidal's knife. The puncturing and scarification not alone incise the lupus tissue directly, so that it undergoes local destruction, but they also destroy a large part of the nutrient blood vessels. These operations may be employed in diffuse infiltrations as well as in single, scattered ones which cannot be enucleated very well with the sharp spoon.

Before each puncture the lance may be dipped, as recommended by Auspitz and Schiff, in a feebly caustic fluid, such as a solution of iodine, carbolic acid, or chloride of zinc, and in this way the caustic is introduced into the midst of the little lupus nodules. This may also be done by means of a closed rubber tube (into which the fluid has been aspirated) armed with a fine canula; or by Auspitz and Kohn's sharp spoon, which is also provided with a puncturing canula and a fluid-holder.

Apart from mechanical treatment, the use of caustics is important. The most practicable is solid nitrate of silver. It has sufficient resistance to penetrate the individual lupus nodules, thus uniting mechanical and caustic action. It also possesses the advantage that it does not enter healthy tissues. Large nodules of lupus tumidus, and particularly superficial infiltrations, may be bored out as thoroughly as with the sharp spoon. Since the solid stick not only destroys the vessels of the borders and base mechanically, but also causes thrombosis of the vessels, the cauterization furnishes all the necessary requirements for effecting a cure. This caustic is especially useful when the lupus occurs in the form of a few scattered primary nodules. Apart from the galvano-cautery, there is no better than the solid stick in lupus of the conjunctiva and cornea.

A concentrated solution of nitrate of silver (argent. nitrat., aq. destil. āā 10.0) is not used in lupus if the skin is unbroken, because it does not penetrate the epidermis. It is used only in partly degene-

rated lupus, upon loose granulations, or to prevent the relapse of recent papules. In the latter event the cutaneous surface is first washed with soap, and a solution of caustic potash 5.0, aq. destil. 10.0 is vigorously rubbed in by means of a pledget of lint. This detaches the epidermis over the lupus nodules, and the latter are exposed as red, raw dots. The excess of potash is now washed off with a weak solution of carbolic acid, the parts dried, and the solution of nitrate of silver then applied.

Pure chloride of zinc deliquesces very rapidly in the air, and may be applied in this way on a brush or it may be dissolved in a little alcohol and water. Bruns and Köbner have melted it into rods by mixing with potassium nitrate and potassium chloride, according to the following formula: zinc. chlorid. 1.0, potass. nitrat. 0.5 to 1.0, potass. chlorid. 0.5 to 1.0. The rods must be covered with tinfoil, because they are very hygroscopic. They are not as resistant as lunar caustic, melt during cauterization, and they do not coagulate the blood, so that the latter floods the surface of operation. Finally, the pain is not much less than that produced by lunar caustic, and the formation of the cicatrix does not proceed more favorably.

Canquoin's paste is made by the mixture of chloride of zinc with treble the amount of starch. When smeared on linen and applied it cauterizes both healthy and diseased skin, so that it is to be used only on the trunk and limbs.

This is also true of the modified Landolf's paste. It is made in the following manner: Zinc. chlorid. 10.0. D. S.: Put in a bottle.—Butyr. antimon. 10.0. D. S.: Put in a bottle.—Acid mur. conc. pur. 5.0. D. S.: Put in a bottle.—A little pulv. rad. liquir. The chloride of zinc is now placed in a mortar, and hydrochloric acid added until it is entirely dissolved. Then the chloride of antimony is added, and the whole rubbed together into a thick paste, while gradually adding the pulv. liquiritiæ. This is spread upon linen cut into strips of the desired size. The strips are tied in position and allowed to remain twenty-four hours. At the end of five to six hours pain sets in and continues several hours. After removal a yellowish-brown scurf is found, which falls off in a few days and leaves a granulating wound. The paste also affects healthy skin, so that it may only be employed when the preservation of the healthy bridges of skin is not important. Hence it is best adapted to the borders of lupus serpiginosus, upon the trunk and limbs, and never on the face. On account of the deep cauterization, the subsequent cicatrices are very large.

A preferable preparation is the pulvis cosmi modified by Hebra (arsenici albi 1.0, cinnabar. fact. 3.0, ung. emoll. 24.0). The paste is smeared thickly on linen, applied for twenty-four hours, and then replaced by fresh paste. Pain sets in during the second day. On

the third day the paste is again renewed, and, as a rule, the pain usually lasts several hours. After the removal of the paste the pain ceases forthwith. It is then found that only the lupus papules have become blackish gray and necrotic, while the intervening skin remains intact. This is extremely advantageous in treating lupus of the face, because small wounds alone remain after exfoliation of the scurf, and these heal in a few days with good-looking scars. In ulcerating lupus the effect will be obtained in two days; in lupus tumidus, in four days.

We have never observed any toxic symptoms due to arsenic, but no surface larger than that of the palm of the hand should be cauterized at one time. Poisoning, with a fatal termination, we have seen once from the use of a paste composed of equal parts of arsenic, opium, and creosote.

Dupuytren's powder (acid. arsenios. 0.1, calomel 8.0) is strewn upon ulcerating and proliferating parts, but possesses very slight caustic effects. Caustic potash vigorously cauterizes healthy tissue as well as the lupus, and hence may only be used against large lupous infiltrations, due consideration being paid to their location.

This is true also of Vienna paste. We prescribe: Kali caust. pulv. 5.0. D. S.: Put in bottle.—Calcar. caust. pulv. 5.0. D. S.: Put in bottle.—Spir. vin. rectif. 10.0. D. S.: Put in bottle. The caustic potash and lime are rubbed up in a mortar and made into a thick paste with a small amount of spir. vini. The part to be cauterized is carefully separated from surrounding parts by the application of strips of adhesive plaster. Then the freshly prepared paste is applied with a spatula and covered with Bruns' cotton. Violent pain is produced in a few minutes. The paste is allowed to remain for ten minutes, then the cotton is removed and the paste washed off by a stream of water, or the part is dipped in water. A black scurf is exposed, and is exfoliated in four to eight days. The paste should not be applied to the face.

Carbolic acid cauterizes very superficially, attacks the healthy skin, causes violent pain, and is very uncertain in its action. Acid. pyrogallic. 5.0, ung. simpl. 50.0 (Jarisch), smeared on linen, has an excellent, painless caustic effect and spares the healthy bridges of skin. It may be strongly recommended.

The galvano-cautery and Paquelin cautery are very useful. The individual lupus papules may be touched with the glowing platinum tip, large infiltrations may be burned with a porcelain tip, or large proliferations may be removed with a loop. The pain is not very severe.

Electrolysis has less caustic effect, but is almost entirely free from pain. Lustgarten employs an almost painless superficial cauterization, produced by the application, for ten minutes, of a slightly con-

cave silver plate (negative pole) about two centimetres in diameter. The current has an intensity of five to ten milliampères.

Methodical applications of iodized glycerin, tincture of iodine, iodoform, empl. hydrargyri are useful auxiliaries in softening extensive cicatrices and diminishing hyperæmia which has been left over. Various dressings, mild cauterization, etc., must be employed, according to circumstances, in the treatment of the wounds, because the greatest attention should be paid to securing thin, superficial cicatrices, especially in the face. The sharp spoon or lunar caustic is best suited for lupus of the conjunctiva and cornea.

In all cases of lupus, especially in very extensive lesions, it will be necessary to employ in succession all of these various remedies and methods. In one place we will be obliged to cauterize, in another to macerate ; here combat erysipelas, there carefully guard the formation of granulations. In short, the treatment must be carried out with great care and skill, and it must not be forgotten that in order to obtain success the complete action of our destructive methods must be secured.

With regard to *scrofulosis* of the skin, I must refer the reader to the well-known works on surgery and pathological anatomy, and to my remarks in the chapter on the etiology of lupus. We have to deal, in this condition, chiefly with inflammation which extends to the skin from inflamed hyperplastic lymphatic glands and from gumma-like nodules of perilymphangitis. The products of the inflammation exhibit slight tendency to organization, and a great tendency to cheesy degeneration and to the formation of flabby, undermining ulcers which heal with stellate and interlacing cicatrices.

Cases of *tuberculosis* of the skin developing independently in its tissues are reported by Wagner, O. Weber, and others, but the majority of these reports refer to inflammatory and ulcerating morbid products in elephantiasic skin or in foci of lupus, which have been regarded as tuberculous on account of the finding of giant cells.

The first case of undoubted true tubercle of the skin was found (1877) in the dead body by H. Chiari. A second one was found at our clinic, in the living subject, by Chiari and Jarisch. The patient, a man of forty-two years, had a large ulcer upon the left ear, with reddish-yellow, moderately firm granulations on the base and a jagged border. He died a few weeks after admission to the hospital, after numerous miliary, rapidly degenerating papules had developed on the velum. In addition to pulmonary tuberculosis, Chiari found, at the rim of the ulcer and in the subcutaneous submucous tissues, isolated and aggregated, roundish papules, about 0.3 millimetre in size, with cheesy centres. These exhibited the undoubted histological characteristics of tubercle. Since that time fourteen cases of

tuberculosis of the skin have been received into our hospital. In all, more than thirty cases have come under my observation.

The symptoms of tuberculosis of the skin are very distinct. It appears in the shape of flat, fissured, very painful ulcerations of irregular shape and finely serrated borders. The base and borders are pale red or grayish and secrete a little thin pus. A flat cicatrix forms upon the ulcer, while at the margins appear new-formed, moderately firm papules, as large as a pinhead or a little larger. The centre of these papules soon acquires a grayish, discolored appearance, and in a few days drops out, leaving a punched-out ulcer. In this way the ulcer enlarges by the peripheral growth of miliary tubercles. The centre may cicatrize completely, and likewise certain parts of the edges, while the disease extends in other parts. Complete recovery hardly ever occurs spontaneously throughout an entire focus.

Tuberculosis of the skin is usually located on the upper lip, the rim of the nares, tip and bridge of the nose, alæ nasi, angles of the mouth, vicinity of the anus, the labia majora and minora. From the lips the process extends to the mucous membrane of the cheeks, from the labia minora to the vagina. I have seen one focus over the tuber ischii, one upon the ankle, one on the back of the neck, and one upon the left temple, involving the ear and parietal region.

Tuberculosis of the palate and cheek was associated in some cases with tuberculosis of the integument. I have also seen a number of cases of tuberculosis of the tongue, palate, and pharynx without coincident tuberculosis of the skin. Fuchs has observed tuberculosis of the palpebral conjunctiva without cutaneous tuberculosis.

Pulmonary tuberculosis was present in all cases, but the cutaneous lesions do not always develop during an exacerbation of the pulmonary process or toward the end of life. All of my cases, with one exception, occurred in males.

Duncan and Thin have described processes upon the female genitalia, the vagina and portio vaginalis, which are attended by ulceration, papillary hypertrophy, and stricture. I am unable to decide as to their character, but they do not appear to be lupoid

The *diagnosis* of tuberculosis cutis is easily made from the symptoms. The secretion of the ulcers and the tissues of the papules contain numerous tubercle bacilli, but in certain stages of the process the bacilli cannot be found.

The *prognosis* in regard to the local affection is not absolutely unfavorable. Spontaneous recovery often takes place in the centre and at a part of the margin. Local cure may also follow elimination by mechanical means or by caustics. We have also seen recovery and immediate cessation of pain following treatment with iodoform. But relapses occur in the vicinity sooner or later, the pulmonary condition grows worse, and finally a fatal termination ensues.

Under the term *tuberculosis verrucosus cutis* Riehl and Paltauf have described chronic inflammatory, papillary patches on the hands, which, in addition to the warty structures on an infiltrated base, also exhibit papules which undergo purulent degeneration and leave pit-shaped scars. In addition to giant cells the papules always contained tubercle bacilli. The writers believe they are due to inoculation with tubercular material, because the majority of cases occurred in individuals (butchers, dissecting-room attendants, prosectors) who come in contact with animal products. Cadaver warts (*verruca necrogenica*) are also regarded by these writers as tubercular in character, and this view has been adopted by the majority of writers.

The inflammatory infiltration and papillary proliferation which distinguish these forms of inoculation tuberculosis from typical tuberculosis of the skin are explained by Riehl and Paltauf as the result of the action of cocci which, in addition to the tubercle bacilli, have gained an entrance into the injured skin.

CLASS X.

MALIGNANT NEOPLASMS.

LECTURE XLIV.

LEPROSY.

LEPROSY (*syn.*, lepra Arabum, elephantiasis Græcorum, spedalskhed [Norw.]) is a constitutional disease which, at the present time, occurs endemically in certain regions. From the fifth to the fourteenth century, especially during the Crusades, it numbered hundreds of thousands of victims throughout Central Europe and along the Asiatic and African coasts of the Mediterranean. In the eighth century thousands of leper houses were erected in Germany, England, and France.

The number of victims began to diminish at the beginning of the fifteenth century, and toward the end of it the disease appeared to be extinct. Indeed, the knowledge of the disease was almost entirely lost from that time until the beginning of the present century.

The first clear knowledge of leprosy was furnished by two Scandinavian physicians, Boeck in Christiania (1842) and Danielssen in Stockholm (1848). Since then numerous writers have investigated this subject.

At the present time leprosy occurs endemically upon all the coast and islands of the Mediterranean, the Black and Caspian Seas, in Norway, Lapland, upon the entire coast of Africa and the adjacent islands, in Asia Minor, Syria and Palestine (Lebanon), upon the coasts and islands of the Indian Ocean and China Sea, in the Australian Archipelago, in certain parts of North America, in Central and South America, and is unusually prevalent in Iceland. In a special form (macular leprosy) it is also found in the southeastern part of Europe, in Moldavia, Wallachia, Turkey, Southern Russia. Scattered cases have also been observed in Hungary, the Tyrol, Bosnia, Roumania.

Leprosy is a chronic constitutional disease which gives rise, upon the skin (and mucous membranes), to yellow, reddish-brown, or dark-brown patches, to flat, diffuse, and nodular infiltrations which

undergo desquamation or ulceration, or, more rarely, to vesicles. It also gives rise to hyperæsthesiæ and anæsthesiæ and to various affections of the internal organs. With rare exceptions it leads to a fatal termination, either directly or indirectly, by a specific marasmus.

Sometimes all the symptoms mentioned are found in the same individual. As a rule, however, they appear in a certain combination and order, so that the disease may be divided into varieties. In accordance with my experience I have distinguished three forms: (1) nodular leprosy, (2) macular leprosy, (3) anæsthetic leprosy.

In all forms the disease is preceded by prodromal symptoms resembling those observed in other severe diseases—viz., anorexia, insomnia, general malaise, moderate fever, diarrhœa. In some cases a few pemphigus vesicles crop out occasionally. The prodromes may last weeks, months, or even several years. They are similar in all forms of the disease.

1. *Nodular leprosy* begins with the development of round or irregularly shaped patches, varying from the size of a finger nail to that of the palm of the hand. At first they are red, but grow pale under the pressure of the finger; later they assume a grayish or sepia brown or bronze color. The integument is smooth, shining as if covered with paint, or it is bronzed and thickened (infiltrated), flat or slightly prominent, and painful on pressure. The patches are scattered irregularly over the entire body.

The process remains in this stage for weeks and months, the patches changing their shape and size. They may coalesce with one another, they may disappear in part, or they may assume an annular shape as the result of central involution and peripheral extension.

At the end of several months, even two or three years, nodules appear in various parts of the body. They vary from the size of fine shot to that of a hazelnut, project slightly or are hemispherical, have a dirty brownish-red color, and are firmly elastic or soft to the touch. They are covered with shining, sometimes moderately desquamating epidermis. In places they may be closely aggregated and then form either irregularly nodular plaques or (rarely) regular, circular figures.

The face is the chief site. Here they form ridges above the eyebrows, running parallel to the latter; upon the nose, cheeks, and chin they appear in dense, irregular masses which remind us of lupus tumidus or acne rosacea. The lips become thickened, either diffusely or in nodules; the lower lip is pendulous, giving the face a malicious, stupid expression. At the same time the frontal ridges give the face an expression of moroseness. The eyelids are often pushed down or everted by nodules; the lobes of the ear form thick, shapeless, gelatinous, transparent masses.

Upon the trunk and limbs the nodules are irregularly distributed ; in many places the finger feels them extending into the subcutaneous cellular tissue. Upon the palms of the hands and the soles of the feet in one case, and upon the face and arms in another case, we have seen small papules which closely resembled those of syphilis or lupus. The flat and nodular infiltrations of the hands and feet, with the accompanying œdema, thicken these parts and cause severe pain, so that walking and handling objects are interfered with.

Upon the conjunctiva palpebrarum and the cornea may be found trachoma-like papules and retractions (pannus crassus s. leprosus). Ulceration of the cornea may follow ectropium and lagophthalmus. In one of our cases nodules developed upon the sclerotic coat and ciliary body of both eyes, and their extension gave rise to gray infiltration of the cornea and blindness.

The individual nodules run an extremely slow course, though they may develop quite rapidly. They do not undergo a retrograde metamorphosis until the end of many months. Many are absorbed completely, leaving dark, pigmented, atrophic patches ; others extend peripherally while they disappear in the centre ; others undergo degeneration, chiefly as the result of mechanical causes, such as blows or pressure (especially over the elbows, knees, and feet), and give rise to leprous ulcers. These are flat, extremely indolent, secrete a moderate amount of thin fluid, and repeatedly become covered with skin which again breaks down. They rarely extend more deeply, and may then give rise, especially in the lower limbs, to complicating inflammations, lymphangitis, erysipelas, suppuration, and opening of the joints (ankle, metatarsus, phalanges of the fingers and toes). Parts of the bones may thus be lost, entire joints may drop off, and various mutilations may be produced (lepra mutilans).

Numerous gray papules also develop upon the mucous membrane of the nose, the conjunctiva (in Breuer's case a nodule as large as a nut was situated in this locality), but especially on the bucco-pharyngeal mucous membrane and the epiglottis and larynx. These give rise to thickening and fissuring of the tongue, shrivelling of the epiglottis, loss of voice, and a sweetish sick odor from the breath. Sooner or later anæsthesia also develops in various parts of the body.

In some cases these symptoms are attended by fever and develop very acutely. Within a few months they attain as great severity as that reached in other cases at the end of many years. Such cases are complicated by diseases of internal organs (cerebral symptoms, diarrhœa, pneumonia, pleurisy), and death soon takes place. But, as a rule, the symptoms run a chronic course, interrupted by acute exacerbations, especially by fever. The latter may be followed by rapid involution of a majority of the nodules, by a renewed out-

break, or by the involution of old lesions and the production of new ones, so that the fever exhibits the characteristics of a metastatic process.

The general condition suffers considerably during such complications, while it may be entirely normal in the apyrexial stages, unless the local cutaneous affections make the condition uncomfortable. After an average duration of eight to ten years, death will result from general marasmus or a complicating disease of the internal organs, the lungs, pleura, kidneys. The fatal termination often results from an acute febrile eruption which lasts several weeks or months. In other cases anæsthesiæ and pemphigus forms first develop ; these cases are known as mixed leprosy.

2. *Macular leprosy* is characterized by the development, with or without prodromata, of patches which are red, violet, various shades of brown, with or without infiltration ; or they appear in the shape of punctate, striped, or diffuse dark pigmentations which are intermingled with non-pigmented, white dots, patches, and stripes, so that the integument assumes a dappled appearance.

Some authors (E. Wilson) divide this form into subvarieties, such as morphœa (rubra, alba, lardacea, atrophica, nigra), in which there are patches of various sizes, with red borders and the centre white, lardaceous and firm, or atrophic and pigmented ; also vitiligo forms, in which there are extensive sepia-brown pigmentations.

Macular leprosy often passes into the nodular form, and it is also often associated with the symptoms of anæsthetic leprosy.

3. *Anæsthetic leprosy* is characterized by the occurrence of cutaneous anæsthesia. It occurs in spots which are the site of nodules or patches ; or it is preceded, with or without other symptoms of leprosy, by pemphigus vesicles (pemphigus leprosus), which on resolution leave white, shining, anæsthetic spots or are followed by superficial or deep ulcerations ; or the anæsthesia occurs upon parts which appear to be perfectly normal, so that it is found only on examination. The anæsthesia is sometimes preceded for months by redness and hyperæsthesia. The epidermis of the anæsthetic parts is sometimes wrinkled and has a senile look ; this contrasts strongly with the adjacent skin, from which it often appears separated by a reddened, hyperæsthetic zone.

The anæsthetic parts do not correspond to the tracts of definite cutaneous nerves. In the beginning the anæsthesia often changes its situation, and only becomes stable in previously reddened or infiltrated spots which have undergone atrophy. The anæsthesia is complete ; a needle may be pushed into the muscles without producing a sensation, and the patients are often burned without knowing it. At the same time the deeper structures, the fascia and bones, may pain spontaneously, as the result of a blow, or during inflammation.

Subcutaneous nerves—for example, the ulnar between the olecranon and internal condyle, or the cervical plexus—may be swollen and very painful on pressure.

In this form no striking changes occur upon the mucous membrane of the mouth and pharynx, but the patients often complain of dryness and annoying thirst. The hyperæsthesiæ and their secondary diseases now become intensified. Formication in the limbs and tenderness of the nerve trunks appear; the patients are unable to remain in the same position for any length of time; they must even be fed because they are unable to handle anything, or to walk or stand, without suffering the most violent pain and occasionally clonic spasms. After a time the hyperæsthesia diminishes and is followed by anæsthesia over larger areas. This is followed by atrophy of the skin and the underlying tissues, especially the muscles, imparting a flabby, senile appearance to the integument. The face looks old, dull, stupid, on account of the wrinkles and the cessation of the play of the features. Paralysis of the orbicularis palpebrarum causes drooping of the lower lid, epiphora, xerophthalmus; the lower lip is also pendulous and the saliva is constantly escaping. The unequal power of the partially acting, partially paralyzed groups of muscles causes deformities of the face as well as of the limbs, particularly the hands. The flexors are more powerful than the extensors and keep the fingers semiflexed, while the palm of the hand is convex and depressions are seen at the sites of the atrophic interossei muscles. At the same time the tips of the fingers are thickened like a club, the nails are cap-shaped and thinned, the hand is rotated inward at the wrist. The hair of the scalp and body gradually becomes dry, thin, and falls out (lepra alopecia). Finally, ulcerations occur or the tissue is simply consumed, so that, with the increasing loss of the skin, the fascia and tendons, one or another joint is exposed, and a finger, a phalanx, a whole hand or foot may suddenly fall off (lepra mutilans). Here and there dry or moist gangrene may set in. Such changes are probably due to lesions of the trophic nerves, inasmuch as they are also found in places where there can be no question of tension or pressure—for example, in perforation of the nasal septum, iridocyclitis, bleaching of the iris.

Some have thought that the sexual function is abnormally increased. This is not true. On the other hand, the function is not always abolished, even after the integument of the genitalia is anæsthetic.

With the increase in the anæsthesia and the tissue atrophy the production of heat is considerably diminished, the action of the heart is slowed, and the cerebral functions impaired. The patients grow dull, sit for days in an apathetic condition, must be fed and put to bed. Gradually the secretions and excretions are disturbed. The

patients die from an attack of tetanus or from various complications —diarrhœa, pneumonia, pleurisy, Bright's disease, pyæmia.

Lepra anæsthetica et mutilans is usually the terminal form, even if the disease begins as the nodular or macular variety, provided the patient's life is sufficiently prolonged. The nodular form may terminate in eight to ten years, while the average duration of the anæsthetic form is eighteen to nineteen years.

Among the interesting complications which may appear on the skin we may mention : favus, general eczema, molluscum fibrosum, elephantiasis arabum, and especially scabies.

The *prognosis* of all forms of leprosy is absolutely unfavorable. It always terminates fatally with symptoms of trophic or neurotic marasmus or from complicating diseases. A. Hansen believes that some cases remain stationary and are thus practically cured. This is also true of those forms which, as a sort of residuum of former epidemic leprosy, now appear in countries which are free from leprosy, as circumscribed morphœa. This may heal spontaneously, and, even if it persists, does not involve the entire organism.

The *diagnosis* is not difficult, even to the inexperienced, unless the disease is not far advanced. At the onset the macular and nodular forms may be mistaken for syphilis, especially if a nodule is present on the prepuce or upon the lobe of the ear, but the case will be made clear as soon as extensive dark-brown patches appear or when antisyphilitic treatment has proven useless.

Nodules in the face may be mistaken for acne rosacea, those in other parts for lupus and idiopathic multiple pigment sarcoma. The macular form must be distinguished from pigment anomalies of other kinds, especially vitiligo. The anæsthetic form must be distinguished from syringomyelia. Great importance attaches, in diagnosis, to the habitat of the patient. If he comes from a leprous region or has lived there for a long time, the diagnosis of leprosy is more probable.

Since the time of Virchow's studies all investigators have regarded the lepra nodules as "granulation tissue," very similar to that of lupus, except that it does not develop in separate nests and that the constituent formed elements—the leprosy cells proper—are larger and more persistent. Among the analogous granulation tissues of syphilis, lupus, and leprosy, the latter run the slowest course, although they finally undergo involution and absorption or degeneration. The new tissue develops in the corium, sometimes more superficially, sometimes more deeply, starting around the vessels and from their walls, chiefly in the vicinity of the vascular glands and follicles. It extends along the vessels into the rete and between the fat lobules, so that diffuse leprous cellular infiltration results. In the section shown in Fig. 56, from the living skin of an excised

nodule of leprosy, it is seen that the infiltration is subdivided into larger and smaller foci. The bands of interstitial connective tissue are normal in places, and in other places they contain deposits of cells, as in inflammation. Outgrowth and infiltration of the walls of the vessels, and endothelial proliferation, have been repeatedly described (Fig. 57), also cone-shaped and reticular outgrowths of the rete cones and the cells lining the glands. These changes, together with the later obliteration of the glands and follicles, the occasional

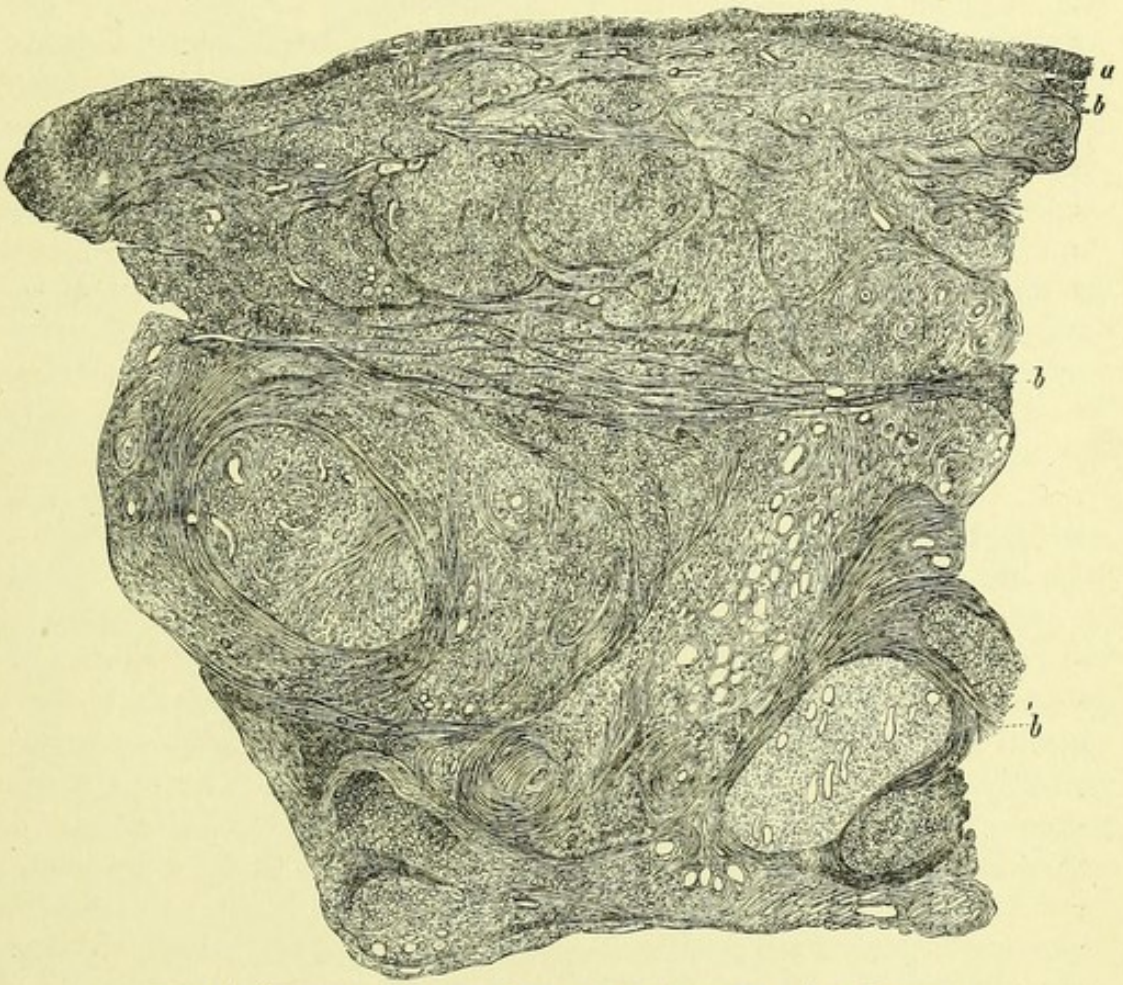

FIG. 56.—SECTION OF A LEPRA NODULE FROM FOREARM. (LOW POWER.)

a, deeply pigmented epidermis; *b*, papillæ and corium with infiltration of small cells, which appears separated by connective-tissue bands (*b'*) into alveoli.

hæmorrhages, the signs of retrograde metamorphosis (fatty degeneration, distention of the embedded cells, formation of giant cells)—all these changes correspond to those which occur in lupus. The incapacity of the leprosy nodules for higher organization is probably explained by their slight vascularity compared with lupus, and the final retrograde metamorphosis and blocking of the scanty vessels by the proliferation of their lining endothelium.

The affected nerves disclose a true lepra neurorum. We find a

chronic inflammatory process which involves, in small, microscopic foci, at first the connective tissue of the external nerve sheaths, then the neurilemma, and later the septa separating the individual nerve bundles ; this is associated with an infiltration of cells corresponding to the inflammatory foci. The process may undergo involution in places, or it may finally lead to fatty degeneration or complete atrophy of the individual primitive fibres of the nerves.

This condition is not peculiar to leprosy, but it explains the par-

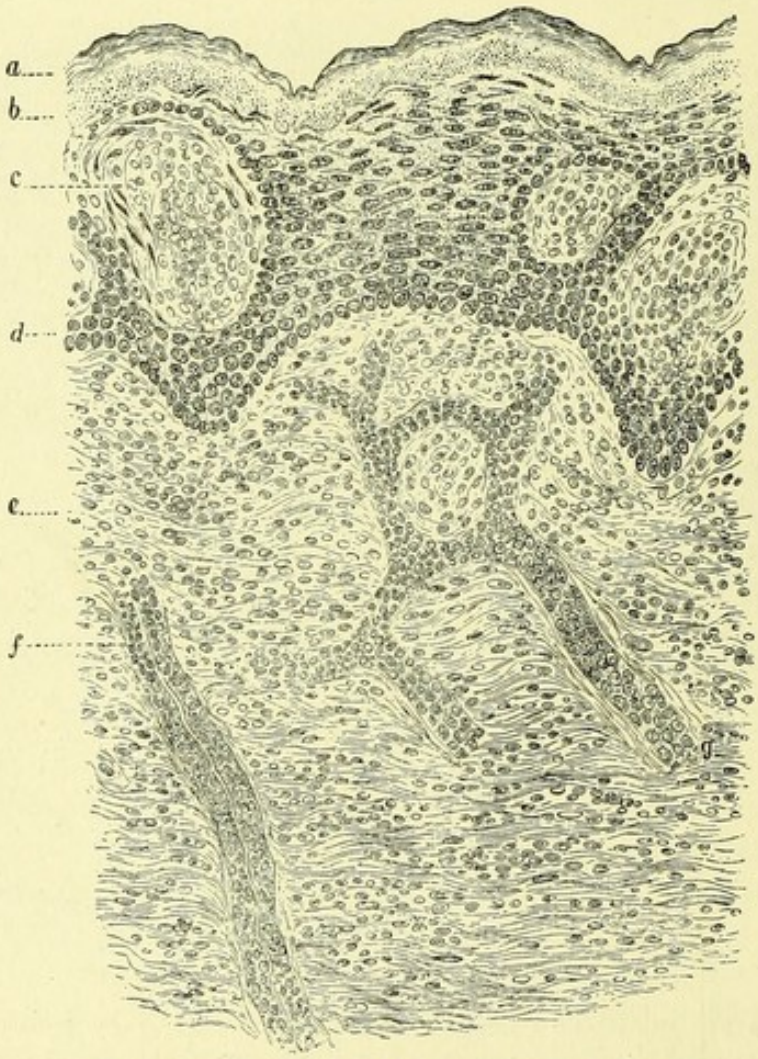

FIG. 57.—SECTION OF A LEPRA NODULE. (HIGH POWER.)

a, horny layer; *b*, granular layer of epidermis; *d*, swollen rete; *c*, papilla, greatly increased in size, like corium (*e*), by cell infiltration; *g*, blood vessel with cell proliferation of its wall; *f*, lymphatic (sweat duct ?) with proliferating endothelium.

æsthesiæ, hyperæsthesia, anæsthesia, and trophic changes, as well as the fact that the sensory changes may be temporary at the start, so long as the inflammatory products are still capable of absorption. It also explains the peculiarity that the anæsthetic regions are so irregular anatomically because only certain primitive fibrillæ are injured while adjacent ones may be intact. A large part of the anæs-

thesia is also due to direct implication of the most peripheral nerve fibres by the infiltrations of the cutis (nodules, maculæ).

The view that the sensory and trophic disorders of leprosy are due to a leprous affection of the central nervous system (Rosenthal) has no objective foundation. Spinal lesions are rarely found. Steudener, Langhans, and Tschirjew have observed pronounced myelitic softening of the posterior horns and Clark's columns, advancing to the formation of cavities.

According to recent investigations, focal cellular infiltrations of the connective-tissue framework, with secondary atrophy of the parenchymatous tissues, have been found in the lungs, intestines, testicles, liver, spleen, and kidneys. The bacillus lepræ will be discussed later.

The *etiology* of leprosy has been repeatedly investigated by commissions and individual observers. Leprosy is an endemic disease, confined, on the whole, to certain localities. Until the last few years it has been assumed that leprosy is due to climatic-telluric conditions or to the physical condition of the soil, or to a malaria-like agent resulting therefrom. This view does not harmonize with the fact that the disease is found in the icy north and under the burning sun of the tropics; upon the heights of the Lebanon, remote from the sea, and in the swamps of the Crimea.

For a long time it was held that leprosy was an hereditary disease. From the statistics of Danielssen and Boeck it appeared that the disease developed in the same family for generations, even if the offspring had migrated in earliest childhood to regions which were free from leprosy. This doctrine is opposed by the fact that many persons, whose ancestors had never suffered from the disease and had lived in regions free from it, acquire leprosy after migration to countries where it prevails. I am acquainted with an entire series of such cases. One woman went to New Orleans from her native town in Alsace, and returned in a few years a victim to leprosy; men born in South Germany acquired leprosy in Buenos Ayres, in Montevideo or Mexico. J. C. White has also shown that leprosy is rapidly spreading in parts of North America in which it had formerly been unknown, and that it is imported mainly by the Chinese. The reports from Hawaii tend in the same direction. According to Gibson's official report, leprosy had attained an enormous diffusion in 1886, although the disease only entered the islands in 1859 (imported by two Chinamen).

A positive basis for the doctrine of the contagious character of leprosy was first furnished by the discovery—at first by Hansen and later by Neisser—of bacilli in the tissues of leprosy. These bacilli are rod-shaped (Fig. 58). They are often narrowed at the ends, their length equals one-third to one-half the diameter of a red blood

globule, and they are seen in short rows, usually in bundles, in the large leprosy and giant cells, rarely in the intercellular spaces.

Unna believes that he has proven by means of a special method (desiccation) that the bacilli are not situated in cells, but are free in the lymph spaces. All other observers agree that they are situated mainly in the cells. On the other hand, there is no doubt that free microphytes are also present in the lymph spaces.

A pearl-necklace-like structure of the bacilli has been demonstrated by various methods. Lustgarten showed this by placing freshly excised pieces of the nodules in two-tenths per cent chromic

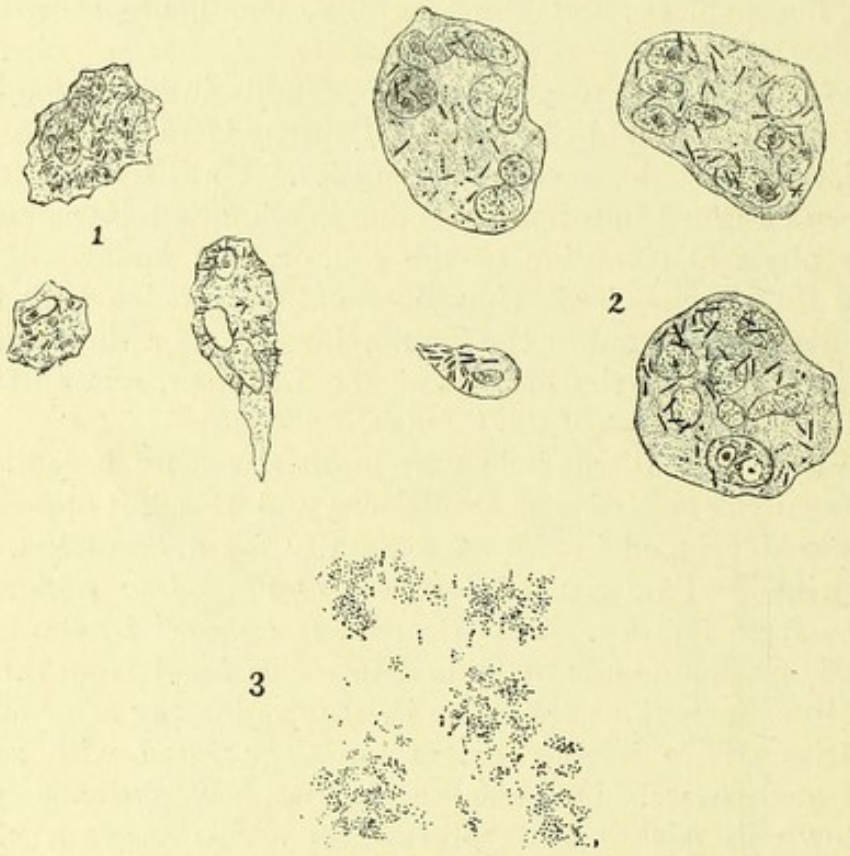

FIG. 58.—BACILLUS LEPRÆ.

1, lepra cells with bacilli ; 2, giant cells with bacilli ; 3, free bacilli and spores.

acid for twenty-four hours and then treating in the ordinary way (Fig. 58, 3). The bacilli are also found in the leprous infiltrations of the internal organs, and rarely in the blood.

In view of all the facts of the disease and of the constant presence of the bacillus in leprosy, it is not surprising that many pathologists regard it not alone as a specific infectious disease, but also as contagious—*i.e.*, that the disease can only be acquired by direct contagion. This point requires further elucidation.

Experiments on animals do not support the theory of the contagious character of leprosy. Köbner, Campana, and Thin made

unsuccessful inoculations of pure cultures of the bacillus in cold-blooded and in warm-blooded animals; Neisser produced local inflam-

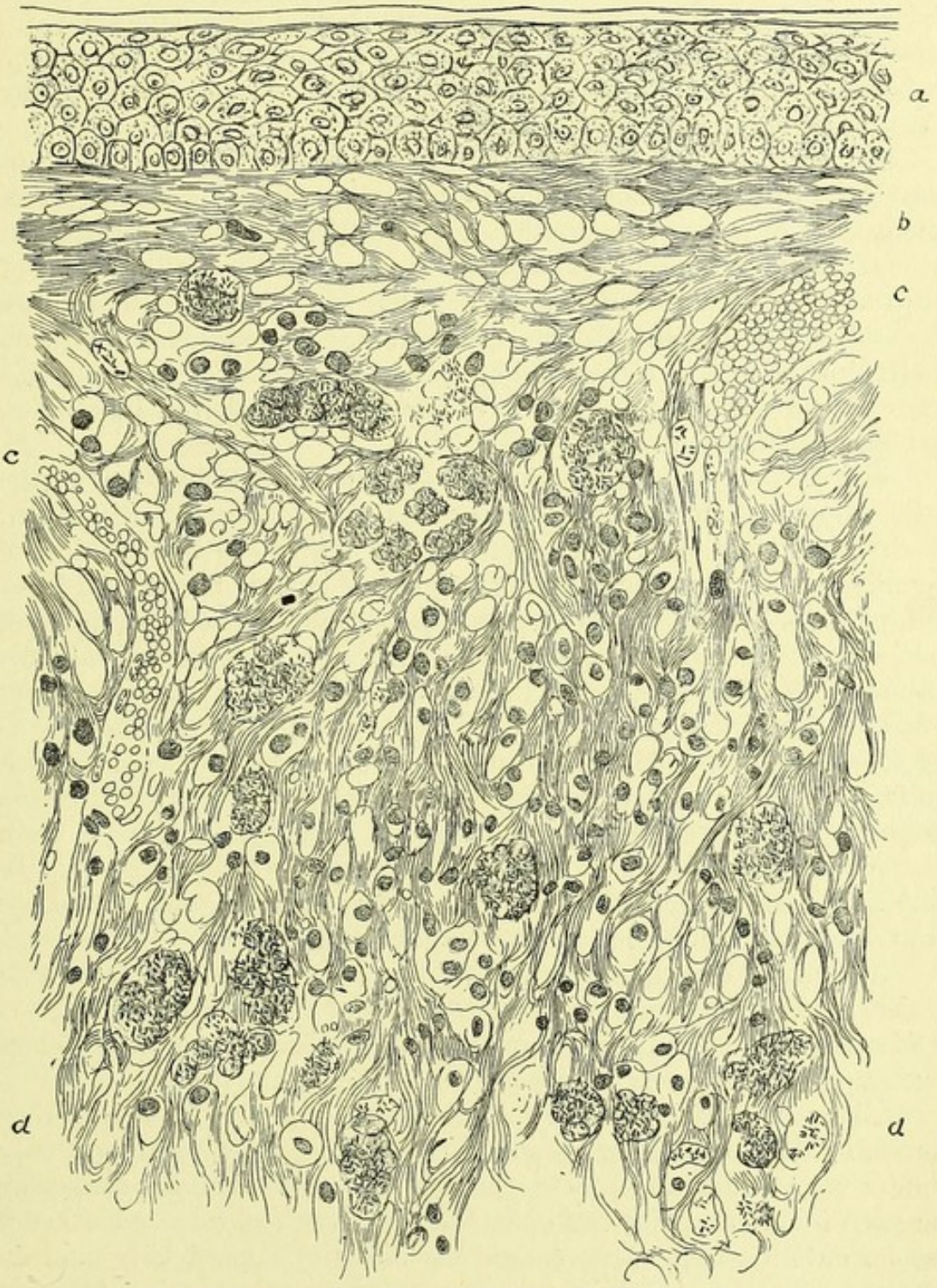

FIG. 59.—SECTION OF A LEPRA NODULE EXCISED DURING LIFE.

a, epidermis; *b*, subpapillary layer of corium, free from cell infiltration, with dilated lymphatics; *c c*, blood vessels. In centre of the nodule, from *c* to *d*, small cells and giant cells containing bacilli.

matory nodules containing bacilli by introducing pieces of leprosy tissue beneath the skin of rabbits; Damsch by introducing them into

the abdominal cavity of dogs; Bizzozero inoculated them into the cornea, giving rise to keratitis (the micro-organisms retained their vitality for two months), but general infection did not result. These facts, however, do not disprove contagiousness, inasmuch as the animals may be refractory to leprosy as they are to syphilis. Even the experimental inoculation of eight men by Profeta and of one man by Arning furnished negative or inconclusive results.

The assumption that vaccination from arm to arm may have conveyed the disease in some places is controverted by Ashmead's statement that in Japan vaccination is performed by tattooing without transmission of the disease so far as known. No undoubted case of transmission of leprosy is known. Even when individuals who are not tainted by heredity have been attacked in leprous regions, no individual source of infection has been discovered. Of course this is rendered difficult on account of the long duration of incubation which is assumed for leprosy.

Still, if an incubation period extending over many years is the rule, then we cannot understand rapid propagation by contagion, such as occurred in the Sandwich Islands within the brief period of twenty years. The contagious character of leprosy is also rendered improbable by other experiences. Boeck and Danielssen mention examples of married couples, one of whom was leprous while the other was not infected, despite the fact that they slept together (*sensu stricto*) for many years. Still more interesting are the statements of Prof. Baetz, of Tokio. In Japan, where leprosy is extremely common, especially in the country, the people sleep together in large numbers, naked, under one cover; and although many of them suffer from ulcerations, no case of contagion has been observed. In the hospitals the lepers live with the other patients, but the nurses and other patients do not acquire the disease.

The official Hawaiian report (1886) on leprosy is also unfavorable to the theory of contagion. The infection of the population by two Chinamen (Hillebrand) appears to be mythical, and the diminution of the number of cases as the result of isolation seems illusory.

All experiences favor the opinion that leprosy, although very probably due to bacillary infection, is not contagious, or at least only under very special and rare circumstances. Infection and contagion are not identical. For example, pityriasis versicolor is undoubtedly a dermatomycosis. The fungi are situated superficially and in masses upon the integument. Nevertheless no clinician includes pityriasis among the contagious diseases. The question of the contagion of leprosy is important, because upon it depends the question of the compulsory isolation of the patients. This question we must answer in the negative.

The *treatment* of leprosy does not offer much prospect of suc-

cess. Nevertheless certain processes may be brought to a standstill, or even made to disappear, the general condition may be improved and life prolonged.

Not alone may nodular and diffuse infiltrations and pigmentations of the skin be improved or undergo involution, but we may even notice the recovery of anæsthesiæ and paræsthesiæ. Nodular eruptions, ulcers, and patches of the mucous membrane of the mouth, pharynx, and larynx may also be improved. In two cases Koch's injections had no effect, in two others they produced reaction and exacerbation. Unna is the only one, however, who reports the cure of two cases of leprosy, but even in these cases we think we are warranted in assuming merely improvement. Experience shows that in such cases the disease finally runs its course, and that death occurs partly from new leprous symptoms, partly from complications such as pleurisy, pneumonia, Bright's disease, marasmus. Hansen believes in the possibility of the cure of anæsthetic leprosy —*i.e.*, in complete standstill of the process.

Leprous disorders of the tissues and nerves often undergo involution for a time, partly spontaneously, partly as the result of febrile and inflammatory complications in the skin (erysipelas and dermatitis) and internal organs. If we remember this fact we will not be apt to overestimate the effects of medication.

The numerous specific and secret remedies which have been recommended are entirely useless. Langerhans and Perez claim to have had excellent results in three cases from the internal administration of creosote (1.25 in 50 pills, three to five pills daily), and Danielssen and Köbner report satisfactory results from the use of soda salicylate (two to six grammes daily). Unna claims to have cured two cases by the internal administration of natrium ichthyosulphuricum and the local application of ichthyol and pyrogallic acid (five to ten per cent ointment). I can only repeat that these remedies may have a temporary effect under certain circumstances. This is also true of the local application of emplastrum hydrargyri and the internal and subcutaneous administration of arsenic and iron. One of our patients took natrium ichthyosulphuricum constantly for fourteen months without effect, but the nodules repeatedly disappeared in many places under local treatment.

We come to the conclusion, then, that the treatment must be carried out according to general therapeutic principles. When practicable the patients should be sent to regions free from leprosy. The general nutrition must be improved by mountain air, nourishing diet, baths; the local symptoms (nodules, ulcers, inflammatory phenomena) are treated according to general surgical principles. Hyperæsthesiæ are treated by opiates, anæsthesiæ by electricity. Local applications of tincture of iodine, emplastrum hydrargyri,

emplastrum saponatum salicylicum, unguentum pyrogallicum, chrysarobin, may result in resolution of the nodular and superficial infiltrations of the skin and the pigmented patches. Creosote or camphor ointments should be applied to the atonic ulcers.

LECTURE XLV.

GENERAL CHARACTER OF THE SYPHILIDES—CLASSIFICATION ACCORDING TO MORPHOLOGICAL APPEARANCES—SPECIAL FORMS—SYMPTOMATOLOGY —DIAGNOSIS—RELATIONS TO CONSTITUTIONAL SYPHILIS— GENERAL AND LOCAL TREATMENT.

CUTANEOUS SYPHILIS—SYPHILIDES.

SYPHILIDE is the term applied to the cutaneous symptoms of constitutional syphilis, whether hereditary or acquired.

Whatever the initial lesion may have been, the syphilitic affection of the general integument, the syphilide, is as a rule the first undoubted symptom of the general infection. Specific forms of disease of the skin may then continue to appear for months, years, or even for life, as signs of the still existant syphilis. The syphilides will be here considered chiefly in regard to the peculiarity of their clinical characteristics. This will furnish a basis for their absolute diagnosis, differential diagnosis, and for their treatment.

The morphological characteristics are the same as those of non-syphilitic dermatoses (maculæ, papules, nodules, pustules, ulcers, with the formation of scales and crusts).

Their undeniably specific clinical character does not depend upon their morphological properties, nor, as is often taught, upon certain other physical peculiarities, such as the dark brownish-red (copper) color ; their localization, in the main, upon the flexures of the joints and around the openings which lead to the various hollow viscera ; their symmetrical appearance, polymorphism, absence of itching, etc. All these phenomena are often found in non-syphilitic eruptions (brownish-red color of acne rosacea and acne disseminata, the grouping of lichen scrofulosorum and lichen ruber planus, the occurrence of psoriasis vulgaris on the palm of the hand, etc.).

Careful examination shows that the peculiarities of syphilides are the expressions of a number of phenomena which form the anatomo-pathological course of the individual efflorescences, and are manifested by three characteristic factors.

1. The syphilides are sharply defined, dense, and uniform (cellular) infiltrations of the papillary body and corium, and differ from one another only in size.

2. These cells are not fitted to undergo permanent organization (into connective tissue), but always undergo involution and disappear either by absorption or purulent degeneration.

3. The syphilitic infiltration of the skin always enlarges and disappears in the same direction—viz., centrifugally. Hence the peripheral parts are relatively the most recent and exhibit all the characteristics of the fresh infiltration. The oldest parts are in the centre and are the first to disappear.

In regard to these fundamental characteristics of the syphilides, the papule may be regarded as the prototype. It constitutes the acme of the syphilitic production as a sharply defined, dense, cellular infiltration of the corium and papillary body. In tracing its symptoms from the moment of its development to its disappearance, we will at the same time indicate the path followed by all syphilides.

If we imagine a vertical section through the epidermis, rete, papillary layer, and corium, the cellular infiltration constituting the papule is found bounded by two sharply defined, lateral boundaries in the corium and in the papillæ.

From this fact we can infer the clinical characteristics of the papule. It projects above the level of the skin, it glistens because the epidermis is stretched by the dense cellular infiltration, it does not disappear under the pressure of the finger, gives a sensation of firmness to the touch, and has a brownish-red color on account of the escape of the coloring matter of the blood, due to the stasis in the compressed vessels.

If an efflorescence does not exhibit all these signs it is not a syphilitic papule, or at least not a recent one.

After a longer or shorter interval, retrograde metamorphosis of the cells and absorption take place. This occurs first in the centre, resulting in a depression there.

The formerly tense and in part proliferated epidermis begins to wrinkle, and later breaks into scales as the underlying infiltration disappears. The peripheral parts still retain their firm consistence and brownish-red, tense, shining appearance. Hence we have a constant appearance : a central, depressed scale, or a central depression due to atrophy of the skin, surrounded by a brownish-red, firm, glistening zone of infiltration.

The complicated forms which appear, for example, on the palm of the hand as the result of an accumulation of such papules undergoing involution, can only be recognized and differentiated from similar non-syphilitic affections by taking into consideration these elementary processes in the individual papules.

Psoriasis syphilitica palmaris et plantaris diffusa never develops except by the juxtaposition of individual papules. Later their peripheries come in contact and scales appear at the centre of

the different papules—a very characteristic sign. Even when a uniform deposit of scales accompanies the advancing atrophy of the papules, a continuous brownish-red rim of infiltration is found at the extreme periphery. In non-syphilitic psoriasis, chronic eczema, and non-syphilitic keratosis of the palm of the hand, the horny, thickened epidermis does not exhibit this rim of infiltration at the point of transition into the healthy epidermis.

The syphilitic infiltrations may undergo purulent instead of fatty degeneration. If the pus is present in small amounts it dries and forms, with the overlying débris of epidermis, dirty yellowish-brown crusts. These take the place of the scales just described. Otherwise the relations are the same. The crusts always correspond to the central, oldest part of the infiltration, and are always separated from the surrounding healthy skin by the peripheral, non-degenerated portion of the infiltration. Similar appearances are presented by the arrangement of the degenerating papules in rows and circles.

Under the same conditions we may note the development of pustules and vesicles (herpes and pemphigus syphiliticus). When these are encrusted the characteristic picture is shown by the infiltration of the base and periphery.

Syphilitic ulcers of the skin have a well-known characteristic appearance, due to the constancy of the three factors already alluded to. There is no syphilitic ulcer without a preceding nodule. As this always ulcerates first in the centre, the ulcer is surrounded by the peripheral mass of the nodule. While degeneration is taking place toward the centre, the base and edges of the ulcer look as if covered with lard; the edges are sharply defined but jagged, somewhat undermined, and firm.

A crust forms upon the ulcer. The mass in which the central ulcer is embedded now undergoes degeneration. Fluid is secreted which elevates the central crust and later itself forms a crust. This is situated beneath the first one, but overlaps it at the periphery. Next to this crust, on the outside, a new zone of infiltration has developed in the meanwhile; in this the second ulcer is excavated, etc. This constitutes *rupia syphilitica*. Its external characteristics are: a central elevated crust, which is surrounded by larger rings of crusts, sloping like a roof and situated more deeply; at the extreme periphery is a rim of infiltration. After lifting the crusts a characteristic ulcer is revealed.

In non-syphilitic rupia the crusts also develop from peripheral extension of the destruction of tissues—for example, in a varicose ulcer; or from peripheral extension of a superficial exudation—in excoriations or pemphigus circinatus. But these forms lack the peripheral ring of infiltration—an essential feature in syphilis.

After a syphilitic ulcer has reached a certain size the peripheral specific infiltration occurs, as a rule, only in a part of the disc. Hence it is possible that, in the part which is not subjected to new infiltration, cicatrization will result from granulations starting in the adjacent healthy tissue. In the other direction, in which a new specific infiltration has formed, degeneration takes place, resulting in the well-known kidney shape of the ulcer.

If a number of such ulcers are grouped together we will find central cicatrices, next to a continuous series of ulcers, whose steep, convex margins are situated to the outside, because it is here that the most peripheral parts of the individual infiltrations lie (serpiginous syphilitic ulcers).

It is thus made evident in all forms of syphilitic skin disease that the syphilide consists of a sharply defined cellular infiltration of the corium and papillary body—*i.e.*, it is a papule or nodule—and that all the external manifestations of syphilis are based upon the regular course of the cellular infiltration.

In roseola syphilitica alone is the infiltration wanting, because it forms but an early stage of the papule ; and in the small pustular syphilide, whose middle portion is occupied by a follicle, the clinical demonstration of the infiltration is more difficult on account of the slight dimensions of the compact part of the efflorescence.

After this general survey of syphilides, the description of the individual forms may be made briefly.

Roseola syphilitica (syphilis cutanea maculosa, maculæ syphiliticæ) consists of round oval, pale-rose to bluish-red, distinct but not sharply defined spots as large as a lentil or finger nail. They grow pale under the pressure of the finger, are colored more deeply in the centre than at the periphery, and in the former situation are occasionally papular. These patches do not itch, and are localized mainly on the trunk and the flexures of the limbs. They continue in their original size for days, weeks, even two to three months, and disappear without scaling and without leaving a trace, with the occasional exception of pigmentation. Non-syphilitic roseola is distinguished by the rapid changes in the size and shape of the efflorescence ; herpes tonsurans maculosus by the distinct formation of scales ; pityriasis versicolor by the possibility of scraping off the patches (the fungus is found in the two latter conditions).

Roseola syphilitica usually appears, as the first manifest symptom of constitutional syphilis, six to twelve weeks after infection, or as a relapsing symptom within the first year, rarely in the second or third year. It then appears in large patches or very rarely in an annular form, as persistent red circles varying from the size of a quarter to that of a dollar (roseola syphilitica annularis).

The roseola of early periods is often mixed with papules (maculo-

papular syphilide), or is combined in certain patches with a central papular elevation.

The PAPULAR SYPHILIDE (syphilis cutanea papulosa) occurs in a large and small papular variety.

The *large papular* or *lenticular syphilide* consists of sharply defined, brownish-red, firm, somewhat projecting, shining papules, as large as a lentil or even larger. They enlarge from the centre toward the periphery, form scales and crusts, and disappear after leaving a small atrophic depression which is at first pigmented, later white and shining. As a rule efflorescences of all stages of development and involution are present (polymorphism), so that the diagnosis of lenticular syphilide is quite easy.

It is often the first eruption (combined with roseola) of constitutional syphilis, and is the most frequent form of relapsing disease within the first five to ten years (or even later). The nearer the earlier period the more diffuse is the eruption ; the nearer the later period the more it is confined to certain regions.

When diffuse it is scattered quite uniformly, but is grouped more closely in certain localities : upon the forehead (corona veneris), in the naso-labial fold, around the mouth and nose, upon the flexures of the joints, the mammary fold, groins, genitalia, and anus. These parts, together with the scalp, are the most frequent site of the local relapsing eruptions of the later period of syphilis. In the latter the papules are often arranged in groups or circles.

The differential diagnosis from lupus is based especially upon the constant central atrophy and the absence of deeply embedded papules. Some of the papules may grow to the size of a dollar or even larger, and, as the central atrophy keeps pace with its growth, they form a ring shape (syphilis papulosa orbicularis). This is often distinguished with difficulty from herpes tonsurans, eczema marginatum, and psoriasis annularis.

Among the special forms of localization may be mentioned :

Papules at the angles of the mouth and in the folds between the toes, which are converted into speckled, steep-margined, characteristic, painful rhagades. Papules of the palms of the hands and soles of the feet (psoriasis palmaris et plantaris).

The early forms, which are combined with a general eruption, consist of disseminated papules which are often arranged in circular lines. The late forms, which occur as relapses and often last for years, are characterized by diffuse coalescence of the papules, deep infiltrations, and the formation of thick callosities and rhagades.

Condylomata lata, papulæ latæ, mucous patches, are disc-shaped, plateau-like, firm tumors from the size of a penny to that of a dollar, covered with gray detritus and secreting a viscid fluid. They develop from papules in those parts at which folds of skin lie in

contact (the labia and surrounding parts, groins, perineum, anus, scrotum, penis, mammary fold, and axilla). Their secretion is extremely infectious.

Broad condylomata are not alone a symptom of constitutional syphilis, and very often a relapsing symptom, but they are sometimes an initial lesion, because they are contagious, as such, like the papule. Hence, if one or a few condylomata are present, for example, at the angle of the mouth or the anus of an infant, on the nipple of a nurse, it cannot be decided forthwith whether we have to deal with a relapse of an older syphilis or with the initial lesion of syphilis which was contracted three to six weeks before.

The *small papular syphilide* (lichen syphiliticus) forms firm papules, from the size of a poppy seed to that of a pinhead, almost always arranged in groups and circular lines, and often tipped with small pustules. They undergo involution after pronounced desquamation, and leave shallow, atrophic depressions in the skin. It rarely occurs diffusely as the first eruption or as an early relapse, and then is usually combined with lenticular papules. This facilitates its diagnosis from lichen scrofulosorum and lichen ruber. As a relapsing form it is confined chiefly to the joint flexures and the neighborhood of the mouth and orbits. The diffuse, small papular syphilide is extremely obstinate, often relapses as such, and is found chiefly in cachectic individuals. It may lead to marasmus.

The PUSTULAR SYPHILIDE repeats all the forms of the papular syphilide, from whose papules it develops by purulent destruction of the infiltration. Hence it appears in a large pustular and a small pustular form.

The *large pustular syphilide* (variola, acne, impetigo syphilitica) consists of pus-containing efflorescences varying from the size of bird shot to that of a bean and usually combined with papules. The pustules are flat and surrounded by a brownish-red, firm, shining, raised margin—*i.e.*, by the most recent part of the papule which forms its base. They dry into crusts, and after these fall off the characteristic papule, depressed in the centre, is apparent.

When diffused generally, the large pustular syphilide constitutes the first (as a rule febrile) eruption, or a relapsing eruption of early syphilis. It is not infrequently mistaken for variola, but this is only possible if we overlook the character of the pustules, their mixture with papules, the absence of the vesicular stage, and the protracted course. The relapsing forms of the later period of syphilis are always circumscribed and are grouped like the corresponding papular forms. In localization, upon the nose and forehead they are distinguished with difficulty from acne and lupus; upon the scalp, from eczema impetiginosum; upon the lower limbs (where their base often has a livid brown color), from acne cachecticorum.

Peripheral papules, which enlarge to the size of a quarter or a dollar and become successively pustular, result in forms which are known as pemphigus syphiliticus when there is a large central pustule; as rupia syphilitica when there is a central formation of crusts with a zonal arrangement of crusts and pustular rings; and as syphilis annularis pustulosa when the centre has healed. In all these forms the pustular formation without previous vesiculation, the appearance of the ulceration or atrophy after removal of the crusts, and the sharply defined marginal infiltration, furnish the differential diagnosis from similar non-syphilitic processes, viz., pemphigus vulgaris circinatus et rupiaformis, eczematous and excoriation pustules, herpes iris, and herpes tonsurans vesiculosus.

The *small pustular syphilide*, like the small papular syphilide which gives rise to it, appears always in the form of pustules (called miliary if the size is that of a pinhead), arranged in groups and circular lines. The differential diagnosis from lichen scrofulosorum is only possible, in some cases, from a consideration of remote circumstances; the diagnosis is easiest when lenticular papules are present.

The prognosis of the large pustular syphilide is better than that of the small pustular.

The NODULAR SYPHILIDE (syphilis cutanea gummatosa) consists of large nodules which may be distinguished as cutaneous or subcutaneous gummata. With rare exceptions they appear in late stages of the disease and their localization is circumscribed. The cutaneous nodules are as large as a pea or bean, or even larger, and are in part discrete, but usually arranged in groups (syphilis corymbosa) or in circular and curved lines (syphilis serpiginosa). These forms are very similar to lupus serpiginosus, but are distinguished by their positive characteristics and by the absence of the lupous infiltrations in the central cicatricial area.

The subcutaneous nodules (gummata) form longish, round, firmly elastic, painful nodules, at the start as large as a pea, hazelnut, or even larger. At first they are movable, but later, after proliferating into the cutis itself, they become fixed. The gummata disappear, after the lapse of weeks or months, by atrophy and absorption; in the subcutaneous variety the central part sinks in and they assume a biscuit-like shape.

The ULCERATIVE SYPHILIDE develops from purulent degeneration of the gummata. Syphilitic ulcers are characterized by their great tenderness and the previously described (page 578) specific form and structure; according to the course of the infiltration, they are round, kidney-shaped, serpiginous, or rupia-like. The ulcers following subcutaneous gummata are less typical, because the latter are not arranged so regularly as the cutaneous nodules.

On account of the rapid destruction of the tissues, the ulcerative syphilide is very important, especially when it affects the face. Here, as upon the scalp, it often leads to necrosis of the underlying cartilage and bone; upon the hands and lower limbs complicating inflammation may lead to chronic œdema, elephantiasic hypertrophy, and mutilations. Otherwise the prognosis is not more unfavorable than in other syphilides.

Syphilis cutanea vegetans (frambœsiaform) occurs as papillomatous, red, warty outgrowths which rise above excoriated or ulcerated papules or gummata. Their most frequent sites are the nasolabial folds, the angles of the mouth, the opposed cutaneous folds of the inguinal region and mammary folds, more rarely other parts of the body. These outgrowths have the same significance as non-syphilitic inflammatory processes, such as the vegetations in elephantiasis arabum, sycosis, lupus, etc., and they can only be regarded as syphilitic in so far as they develop upon a syphilitic infiltration. When the latter has disappeared such a diagnosis is not possible, because the warty outgrowths appear, clinically and histologically, like connective-tissue new formations.

It is these papillomatous forms of syphilis which have been reported in literature as endemic diseases under the terms radesyge (Norway), siwwens (Scotland), falcadina (Istria), yaws, frambœsia (West Indies), etc., but whose manifold significance has already been referred to (page 476).

With regard to *frambœsia*, however, many writers (particularly English and American), and especially Pontoppidan and Charlouis (Java), have attempted to furnish proofs of its pathological independence. The latter describes it as a disease which occurs in the West Indies among the African negroes (yaws); in East India, on the other hand, mainly among the natives. At first disseminated, later confluent, moist and papillomatous, or deeply ulcerating nodules (mama-pian) are produced, which usually disappear spontaneously at the end of a year. The disease is contagious, and is rapidly cured by antisyphilitic treatment, but is nevertheless said to be distinct from syphilis

All these factors evidently favor the view of the syphilitic character of the forms of disease referred to But one fact is extremely noteworthy. In a man who was suffering from frambœsia, Charlouis produced constitutional syphilis by inoculation from a hard chancre. This proves positively that, in this case at least, the frambœsia was not identical with syphilis. Charlouis proposes for the disease the term "polypapilloma tropicum."

In HEREDITARY SYPHILIS there appears, either at birth or within the first three weeks of life (rarely later), a syphilide which does not

differ materially from that of acquired lues. It is usually a maculo-papular eruption, with formation of rhagades at the angles of the mouth, the anus, the interdigital folds. More rarely it is a papular syphilide, under the form of large pustules, which develops on ulcerating, flat papules (pemphigus syphiliticus). A characteristic feature of hereditary syphilis is a diffuse infiltration of the sole of the foot and the palm of the hand; the skin is uniformly brownish red, dry, shining like satin, and covered here and there with rhagades.

Gummatous nodules and their ulcerative forms appear in the later years of hereditary syphilis as in the acquired disease.

LECTURE XLVI.

CUTANEOUS SYPHILIS (*continued*).

TREATMENT.

THE treatment of syphilides is identical, on the whole, with that of constitutional syphilis. Hence it will be advisable to present to you the principles of a rational treatment of syphilis.

The treatment of syphilis has not kept equal pace with the course of pathology, and does not differ materially from that practised at the end of the fifteenth and beginning of the sixteenth century. At that time inunctions with mercurial ointment were the sole means of treatment. In the second decade of the sixteenth century the vegetable remedies of the West Indies were introduced; and inasmuch as relapses occurred despite repeated mercurial treatment, but rapidly recovered under the use of the former, the view gained ground that the relapses were due, not to syphilis, but to the mercury itself.

This was soon disproved, however, and at the end of the first half of the sixteenth century mercury had been restored to its old place. The only addition to the therapeutics of syphilis since that time has been the introduction of the use of iodine and of Lewin's method of subcutaneous injections of mercury.

In opposition to Bärensprung and others, we proclaim emphatically that we regard syphilis as a curable disease—indeed, as one of the most curable of all the infectious constitutional diseases.

Certain questions of principle play a decisive part in practical medical treatment and will here be briefly considered.

First: Can the prevention of general infection be expected from any mode of treatment of the initial lesions?

In the decision of this question it is immaterial whether we stand, as I do, upon the unitarian standpoint and believe that every form of primary lesion—hard or soft chancre, erosion, sclerosis, papule—may furnish the source of the general infection, or whether, with the dualists, we ascribe this part solely to the typical sclerosis. It is only essential that we do not regard the sclerosis as a manifestation of the already completed general infection.

If we adhere to the view that the specific virus remains for a cer-

tain length of time at the site of the primary lesion and is then absorbed into the lymph and blood channels, we should, from a theoretical standpoint, attempt to prevent this absorption and the consequent general infection. In order to achieve this end three methods are possible.

1. The destruction of the virus at the site of inoculation—*i.e.*, together with the primary lesion. This may be done in two ways—by vigorous cauterization and by excision of the initial lesion.

The second method has been extensively practised. In a small proportion of cases the results were apparently satisfactory, but in the large majority they were negative.

With regard to the assumed results of preventive treatment, it must be remembered that the absence of syphilis after the cauterization or excision of the initial lesion cannot be regarded *per se* as a positive success. In the first place, not every sclerosis is necessarily followed by syphilis (such cases have been reported by myself, Boeck, Leloir, and Dubois-Havenith); and secondly, it depends upon the judgment of the operating physician whether the initial lesion in question is to be regarded as a typical sclerosis. Furthermore, the number of cases of positive success will be greatly reduced the longer the period of observation after the operation.

Moreover, we possess no positive basis for determining the period at which local extinction or elimination of the virus is still theoretically possible. Sigmund has seen distinct sclerosis as early as the third day. We do not know whether absorption is not possible at a much earlier period. On the other hand, the frequent alterations in the constitution of the sclerosis, observed in many cases as late as the third week, seem to favor the view that the virus perhaps remains localized for a long time.

The absence of distinct swelling of the adjacent glands is a negative criterion, because absorption is conceivable without such swelling. The presence of adenitis is not a positive sign, because it may be due to any lesion, especially suppuration.

The demonstration of Lustgarten's bacillus would be a sure indication if its relations to syphilis were ascertained in all directions. Since the demonstration of bacilli in the smegma of the prepuce, Lustgarten's syphilis bacilli have lost in importance, but it is to be remembered that they are found particularly in tissues of syphilitic origin—in the tissue of scleroses, gummata, recent non-ulcerated papules of the trunk, etc. Lustgarten demonstrated the bacilli within the cells of such tissues, where they have also been seen by myself and others.

Excision depends practically on certain anatomical conditions. For example, it may be performed thoroughly upon the preputial margin, while this is hardly possible on the glans and in the coronary sulcus.

The question also presents itself whether the methods of treating the chancre proper have any effect in the prevention of syphilis. I think I am able to claim that mercurial ointment has an undoubtedly favorable effect upon the resolution of the sclerosis, with which the source of infection is removed. Indeed, it has often appeared to me as if the formation of the sclerosis itself were prevented.

2. The second method of preventing general infection is the elimination of the absorptive tract. As it must be assumed that the virus is absorbed by the lymphatics and first stored in the glands of the first zone, then (in affection of the penis) we must merely divide (or excise) the dorsal lymphatic and perhaps remove the glands. But if we examine a penis whose subcutaneous lymphatics have been injected, we will convince ourselves that the entire organ is everywhere traversed by a network of lymphatics. According to Max Zeissl and Horowitz, lymphatics also pass through the inguinal canal to glands within the pelvis. Hence inferences made with regard to the assumed success of such operations will not possess much value. This is also true of Lipp's method of injecting mercurial preparations, iodine, etc., in and around the lymphatic glands of the first order, and thus destroying the virus along its path of absorption.

3. The third method of preventing general infection is preventive general treatment. If an initial lesion warrants the assumption that general syphilis will follow in four to six weeks, will it not be rational to begin forthwith the general treatment which is known to be effective in evident syphilis ?

In my opinion this is not practicable. I have always deprecated beginning treatment before the outbreak of the eruption. Hebra, Sigmund, and Zeissl have also opposed preventive treatment because their experience was similar to mine. Experience teaches that such premature treatment merely delays the general symptoms ; that they then appear in an irregular course, and that we no longer find mild eruptions, but very early severe symptoms, such as periosteal lesions. It is therefore harmful to the patient and confusing to the physician to begin general treatment before the symptoms of constitutional syphilis are fully pronounced. Indeed, it is well to allow the eruption to develop more fully, and not to begin treatment at the first indication of roseola.

The second question for our consideration is: What advantages are offered by the drugs which are found to be effective in syphilis, and against which pathological forms ? We must first lay down the principle according to which we will measure the effectiveness of a drug or a method of treatment, and will then proceed to a critical review of these drugs.

The efficacy of a remedy must be measured according to the

promptitude with which it causes the resolution and disappearance of local and visible syphilitic changes, and according to the prevention of relapses. If a papular syphilide, for example, has lasted three months, and we find that under inunctions it exhibits all the signs of resolution on the sixth day, we are justified in regarding the remedy as efficient. But if we find that under vegetable treatment or the cold-water cure the symptoms disappear in the order of development and in a period of three to six months, I would attribute the recovery to the physiological course and not to the influence of the remedy.

It is more difficult to arrive at a conclusion in regard to the prevention of relapses. The statistical data on this point which are furnished by hospitals are useless. The patients are dismissed as soon as the visible symptoms have disappeared. It is only in private practice that we are able to observe the effects of treatment for years in the individuals themselves and in their children. Private material, however, is not used for official statistics, so that the judgment concerning the permanency of the effects of a remedy really depends upon the subjective experience of the physician. I may formulate my own opinions in the following way :

I know quite a number of patients who have been permanently cured after a single course of treatment lasting a few weeks or months.

There is also no doubt that many suffer one or more relapses, are subjected to corresponding courses of treatment, and then remain cured.

Finally, a relapse may occur, in a smaller number of cases, after the lapse of years and decades, even after the patient has procreated a number of healthy children.

It is therefore impossible to fix a limit to the period in which the curative action of a drug can be definitely decided. Hence it seems to me that the conditions must be considered in a different way. If the symptoms of the first acute period of eruption disappear acutely under the use of a certain drug, then the latter is effective, and to a greater degree the more rapidly and generally (*i.e.*, affecting all tissues and symptoms) the symptoms disappear.

On the other hand, we must require, after such a course of treatment, first, that at least several months must elapse without visible syphilitic signs, during which time the patient looks and feels well. The resolution of the polyadenitis is important in this regard. Secondly, the relapses, so far as they involve the skin, must be regional and thus exhibit the character of late forms. But if, shortly after the first eruptive period, a general eruption reappears, similar to the former one, then we are justified in believing that the previous treatment has been ineffective.

We will now consider the drugs and modes of treatment which are regarded as efficacious against syphilis.

A. The curative remedies proper :

1. Mercury.
2. Iodine.
3. Vegetable remedies.

B. The so-called after-cures :

Sulphur baths.

Hydrotherapy (sea baths).

(Fournier's method—protracted cure—traitement successif).

Mercury is administered endermatically, hypodermatically, and internally.

Endermatically it is given in the form of ointments or baths. Among the ointments the most important is the official unguentum hydrargyri, which is used in the methodical inunction cure. The inunction cure is the most effective and reliable remedy in regard to the local affections as well as to the dyscrasia, and it is the only rational method in all cases in which delay is dangerous.

Instead of simple fat, lanolin (Liebreich) and ten per cent ol. olivar. have been recently employed in making ung. hydrargyri. Mercurial soap may also be used, but I prefer the official ointment.

We prescribe ung. hydrarg. 30.0, div. in dos. æqu. no. x. or xii. ad chart. cerat. This is rubbed (at night and in a warm room) into different parts of the body in a regular order, so that the intervals between the applications to the same part of the integument should be as long as possible. In this way we may guard against the danger of mercurial eczema, one of the most violent of all the artificial eczemata. When practicable the patient himself should perform the inunctions.

Emplastrum hydrargyri of all kinds has an extremely good local effect It cannot be recommended too highly in gummatous, ulcerative affections of the skin and periosteum. It may also be used for general treatment by applying to large surfaces of the skin—for example, in infants and also in adults—when extensive ulcerative syphilides require protection by a plaster.

The mercurial bath is made with corrosive sublimate (ten grammes to the bath in the adult, one to two grammes in infants). This is extremely effective in syphilis of the new-born and also in extensive gummatous, ulcerative forms in adults.

Fumigations with cinnabar or calomel are very little used.

The *hypodermatic method* of administering mercury has been much in vogue during the last few years. It is more direct and exact than the endermatic method, and it possesses the further advantage that we can thereby administer chemically well-defined combinations in definite amounts.

Lewin first employed corrosive sublimate injections, but since then other salts have been tried, the attempt being made to administer the mercury in the form in which it is supposed to circulate in the blood. These remedies are :

1. Soluble salts :
   Corrosive sublimate, according to Lewin.
   Sublimate-sodium chloride (Stern-Auspitz).
   Mercury albuminate (Bamberger).
   Mercury peptonate (Martineau).
   Hydrargyrum bicyanatum (Martineau).
   Hydrargyrum formamidatum (Liebreich).
   Blood serum-mercury (Bockhart).
   Glycocollasparagin and alanin-mercury (Wolff and Nega).
   Mercury chloride-urea (hydrargyrum bichloridum carbamidatum), Schütz.
   Mercury succinamate (hydrargyrum imido-succinicum, Wolff), etc.
2. Insoluble salts :
   Calomel in suspension (Scarenzo).
   Hydrargyrum tannicum oxidulatum in suspension (Lustgarten).
   Hydrargyrum oxidatum flavum (Watraszewski).
   Oleum cinereum (Lang).
   Hydrargyrum salicylicum (De Silva-Aranjo).
   Hydrargyrum benzoicum oxidatum (Stoukowenkoff).
   Hydrargyrum thymolicum (Neisser).
   Hydrargyrum sozoiodolicum (Schwimmer).

If I compare my own experiences with those reported by many other writers and with the opinions expressed at the Copenhagen International Congress, I can agree, on the whole, with Bockhart in his statement that those preparations of mercury which remain longest in the body have the most prolonged effect and hence are most apt to prevent relapses.

As a general thing, injections are made to the amount of 0.01 to 0.1 gramme. The following formulæ may be used : hydrarg. bichlorid. 0.1, aq. dest. 10.0; or hydrarg. chlor. mite 1.0, glycerin. 10.0.

Neisser, Kopp, and Chotzen were the first to use suspensions of calomel; Lustgarten used tannate of mercury in suspension. Since then many physicians have used insoluble compounds of mercury in various vehicles—for example, hydrarg. tannic. oxidulat. 2.0, aq. destil., sol. acid. sulph. (one per cent) āā 10.0 (Lustgarten); or calomel vap. pur. 5.0, sodii chlor. 1.25, aq. dest. 50.0 (Kopp and Chotzen). An injection was made every week or two, subcutaneously or intramuscularly, four to eight injections being made in all. It is supposed that soluble chlorides of mercury will be formed gradually from the insoluble salts which have been injected subcutaneously,

and that thus small amounts will be carried constantly into the circulation. When larger amounts of soluble salts are introduced a large part will be rapidly excreted.

It has been repeatedly found, however, that as the result of the acute absorption of large amounts of mercury from the subcutaneous and intramuscular depots of the insoluble combinations, the most intense and even fatal gangrenous stomatitis and enteritis have developed.

Lukasiewicz has combined the advantages of both methods and avoided the dangers by employing a five-per-cent sublimate solution for injection (0.05 to a syringe). One injection is made weekly, and six to eight suffice for a cure, as is shown by the experience at our clinic.

When given *internally* the ordinary preparations of mercury act more slowly, although they are sometimes quite prompt. Calomel and tannate of mercury, although they generally produce diarrhœa, cause less gastric distress and may also be administered to children. The following prescription may be used : hydrarg. tannic. oxidulat. 5.0, sacch. lact. 7.0, div. in dos. no l. in capsules. S. 3 capsules daily. A dose of 0.05 to 0.03 t. i. d. may be given to children. The iodine combinations, the protiodide and biniodide of mercury, are also adapted to internal administration.

Treatment with mercury is indicated, according to my experience, in all forms of cutaneous lesions of the early and late periods, and also in affections of the bones, the parenchymatous organs, the cerebro-spinal system in the acute stages. Later stages of the latter affections, joint affections, and cephalalgia yield much more rapidly and certainly to iodine.

In my opinion, relapses and a protracted course of the disease are prevented with so much more certainty the more persistently and actively the treatment is carried out during the very first acute period of the disease.

For this reason I favor inunctions from the start, and it is only when this is impracticable (this has never occurred in my experience) that I would favor injections of sublimate, calomel, peptonate of mercury, oleum cinereum. Neisser begins the calomel injections immediately after the inunctions with unguentum cinereum.

I regard the slowly acting methods—milder injections and mercury internally—as unjustifiable during the first period, because they prolong the symptoms. In the late forms, unless the symptoms are urgent in character, slowly acting medication is permissible. But as soon as the symptoms become more dangerous—for example, iritis, ulcerative nasal and laryngeal affections, cerebral and spinal disease—I am in favor of the more vigorous medication.

According to my experience, mercurial treatment is unattended

with any disadvantages if we take care to prevent annoying incidental effects It is well known that some preparations produce salivation more readily than others. but I have seen no salivation in years, because I exercise care in this matter. It has already been said that injections with insoluble combinations are the most dangerous in this regard.

Iodine is the second specific remedy against syphilis. This is given as potassium or sodium iodide, one to two grammes daily, in solution or pills. It is most effective in bone and joint affections, and nocturnal pains in the bones and head, but is also very useful in other conditions.

Iodine may not be given, however, as a corrective against the abuse of mercury, although it is often effective when other remedies prove useless. The first period of disease should not be treated by iodine alone, as it will certainly prolong the process. As a general thing it acts very slowly against eruptions.

The third remedy, *Zittmann's decoction*, I regard as extremely effective in late forms, especially in ulcerative affections of the pharynx and skin, and I often give it in combination with mercury, particularly in the form of inunctions. I do not believe that its action is due to the slight admixture of mercury which is possibly present in the official preparation, because it often acts excellently when inunctions, etc., are useless.

As Zittmann's decoction affects the intestinal tract by producing diarrhœal evacuations, the diet must be regulated during its administration. Decoct. Zittm. fort. 250–300, warm, should be taken in the morning, and an equal amount of the mild decoction in the afternoon.

The so-called after-cures, such as sulphur baths, sea baths, hydrotherapy, do not have the slightest direct influence on syphilis. They may be recommended for stimulating the activity of the skin and the excretions and to improve the general tone of the body.

How long and how often should antisyphilitic cures be prescribed? Fournier states that every patient should be treated for at least two years, and believes that he may then guarantee him against relapses.

I am acquainted with many patients whom I have treated only once and who have remained healthy. I have also known others who had frequent courses of treatment and always suffered relapses, who had healthy children, but finally died of cerebral syphilis. Of what avail was the periodical repetition of the treatment in such cases?

I would recommend, in general, that the first course of treatment should be carried out sufficiently long and carefully. The cure should be repeated as often as characteristic symptoms of syphilis (affections

of the skin and mucous membranes, enlargement of the glands, etc.) reappear, but only at such times. When signs of syphilis are not present I regard treatment as superfluous. A rational course of treatment will not, however, do any harm in an otherwise healthy individual, so that I am not opposed, on principle, to an occasional repetition of the treatment within the first couple of years. But the protracted and periodical repetition of the treatment, as a general principle, is unjustifiable in my opinion, and in many respects, indeed, it is disastrous.

Rational treatment is not confined to the proper use of remedies against the constitutional disease. Local treatment of a syphilitic affection of the skin is often indicated irrespective of general antisyphilitic treatment. For example, in ulcerative syphilide of the nose and other parts of the face we cannot wait for the curative effect of general treatment. Before this sets in so much time may have elapsed that important parts, such as the nasal septum or alæ nasi, undergo destruction. When there is local danger the process must at once be circumscribed locally. This can be done in almost every case by cauterizing, into the healthy tissues, with lunar caustic or caustic potash ; in less threatening cases, by the application of a mercury plaster which will adhere well. The latter is particularly serviceable in the treatment of old psoriasis palmaris el plantaris, condylomata lata, painful rhagades, paronychia ulcerosa, obstinate lichen syphiliticus, nodular syphilides, and gummata, even when the latter are already undergoing softening.

Corrosive sublimate also has a rapid local action against psoriasis palmaris et plantaris, in the shape of foot baths and hand baths (5 : 500); against mucous patches, in slightly caustic concentration (1.0 : 50.0 alcohol or collodion), or in the shape of Plenck's solution (sublimat., aluminis, camphoræ, cerussæ, spir. vini, aceti vini āā 5.0).

Tincture of iodine, iodized glycerin, iodoform, iodol and iodoform-collodion, and salve favor the absorption of periosteal and osteitic gummata and joint affections, but not of other forms of syphilis.

LECTURE XLVII.

MYCOSIS FUNGOIDES (FRAMBŒSIA)—LYMPHODERMIA PERNICIOSA—SARCOMATOSIS CUTIS.

SARCOID TUMORS.

UNDER this term I would include, if only for a short time, the diseases which are now to be considered. Their pathologico-histological character is undecided, some regarding them as inflammatory tumor formations, others as tumors in the stricter sense and included among the sarcomata. At one time they appear to be primary affections of the skin, at another time a symptom of visceral affections or of a change in the constitution of the blood, such as leukæmia and pseudoleukæmia.

Whether the internal connection and genetic unity of these forms of disease will ever be demonstrated can only be guessed at in the present state of our knowledge. Until a more thorough understanding is reached they should be classified in pathology according to the types in which they come under observation. From a clinical standpoint they are extremely interesting and important because almost all prove fatal.

I. MYCOSIS FUNGOIDES (ALIBERT).

This term is still employed for a disease which begins with slight, eczema-like symptoms attended with violent itching. After a longer or shorter period flat and nodular infiltrations appear upon the skin, and develop later into extensive, fungous, ulcerating, and papillary tumors. As a rule the disease ends in fatal marasmus.

Alibert (1835) described, under the term mycosis fungoides, a peculiar tumor development of the face, but did not define it as a special morbid process. This was first done by Bazin (1862–1873), Gillot, and Ranvier. For some time after this the majority of cases were reported by French writers (Gaillard, Landouzy, Demange, Vidal, Besnier, Hallopeau, etc.).

Köbner, the first author of any other nationality, described the disease as "multiple sponge-like papillary tumors"; Geber and Duhring, as "inflammatory fungous tumor"; Engelstedt and others, as lymphadenoid tumors; the more recent writers have described

it in part as mycosis fungoides, in part as granuloma fungoides. I and a few others have always regarded it as a form of sarcomatosis.

The differences of opinion manifested by the multiplicity of names are due to the varying interpretations of the clinical symptoms, anatomical lesions, and the supposed etiology of the affection.

In almost all cases mycosis fungoides begins with the symptoms of eczema. Upon the trunk, the flexures of the limbs, the face, especially on the forehead, appear disc-shaped, red patches from the size of a dollar to that of the palm of the hand. The overlying epidermis is finely scaly; in rare cases there is moderate moisture in places, resulting in the production of thin, gum-like crusts. These changes correspond completely with those of squamous eczema in anæmic individuals. They are attended by violent itching and insomnia.

This eczema may continue many months, even one to two years (*erythematous and eczematous stage*), during which some patches grow pale and disappear, others develop or extend centrifugally and coalesce with adjacent patches. In this way a universal, usually very pale eczema occasionally develops. Certain areas heal in the centre while they spread at the periphery and thus form circular patches of eczema. In addition to the scaling and scanty moisture, excoriations, a few larger or smaller furuncles and abscesses, together with diffuse brown pigmentation of the skin, are found as the result of the vigorous scratching.

After some time has passed there is thickening of the skin in places, due to inflammatory, œdematous infiltration of the corium (*second stage*, according to Köbner), especially noticeable upon the orbicular patches, whose borders are somewhat prominent, tense, smooth, and shining.

Thicker infiltrations appear next in the shape of hemispherical, firm, pale brownish-red or bright-red projections of the surface, or distinctly circumscribed nodules, in the middle or various parts of the border of the eczematous patches. Round, reddish-brown, very firm nodules or wheal-like, firm prominences, from the size of a pea to that of a bean, also appear irregularly upon non-eczematous parts of the skin (*période lichénoide*, Bazin). Many disappear completely within a few days or weeks, or they are followed by pigmentation or shallow. atrophic depression, while new lesions appear in other places. Owing to central depression and atrophy with coincident peripheral extension, some patches develop into flat, firm rings of infiltration from the size of a dollar to that of the palm of the hand. The nodules are usually scattered. In one case I saw them closely grouped over the entire trunk, chest, abdomen, and back, so that the skin was as hard as wood and extremely painful.

Finally, red, nodular *tumors*, from the size of a nut or egg to

that of a child's head, develop. These grow either directly from the flat eczematous patches or by the rapid spread of the infiltrations and nodules. They have an unequal, firm or flabby consistence, usually bright red, sessile or constricted at the base, or they have the shape of a flat cake and exhibit shallow lobes. At the top they soon degenerate into ichorous, partially necrotic ulcers with a fungous, freely bleeding base and viscid secretion. I have seen such tumors upon the scalp, mamma, patella, abdomen, thorax, and arm.

A peculiar feature is the rapid spontaneous diminution (within a few days), or even complete involution, of the largest of these tumors, leaving the skin unchanged, except that it is more or less pigmented. Such a portion of the skin may either remain permanently healthy or it may again become the site of a new growth.

Papillary (framboesia-like), red, lobulated, moist, and bleeding proliferations spring either from the base of the ulcerated tumors or directly from the flat infiltrations and eczematous parts of the skin.

There are also cases in which, without any eczematous or lichenoid prodromal stage, a small number of the large, ulcerating, papillary tumors suddenly appear ("d'emblée") and run the same course as the others. Vidal regards these as a second variety of mycosis fungoides.

Enlargement of the glands is always absent.

Paltauf regards as a variety of the disease those cases which are complicated with enlargement of the lymphatic glands, swelling of the spleen and liver, a leukocythæmic condition of the blood, and the presence of nodules on the hard palate and tonsils, thus exhibiting the appearances of a pseudoleukæmia or "lymphadénie."

As a rule the patients are run down considerably, even during the eczematous stage, owing to the intense itching, the consequent insomnia, and the frequent loss of appetite. Some of them exhibit no change in their general condition for several years.

Usually fever is entirely absent, except in the paroxysms due to local ulceration and the resulting erysipelatous and lymphangitic inflammation. But the acute and subacute development of nodules is accompanied by remittent or intermittent fever.

After a period of advancing marasmus (*fourth stage* of writers), which may begin as early as the eczematous stage, the patients die, even at this time from local complications—for example, suppuration of the cellular tissue—or during the period of nodular development, or not until the stage of ulceration and papillary proliferation is reached.

With the exception of four cases, post-mortem examination has shown no changes in the internal organs, associated directly with the process. In a few instances cocci have been found, but they did not appear to be connected with the disease, inasmuch as they were absent in other carefully observed cases.

In one of my cases the left lung contained a few quite hard, not very sharply defined nodules as large as a pea; a grayish white, quite hard, diffuse infiltration was found in places around the vessels; the latter condition was also noticed in the right lung. The peritoneal lining of the stomach and intestines and the greater and lesser omentum contained numerous soft nodules the size of a millet seed; here and there were whitish, prominent lobules or diffuse whitish infiltrations. The liver contained numerous hard, white, sharply defined nodules; the spleen was doubled in size, dense, brownish red in color. The enlarged kidneys contained numerous soft, whitish tumors, some as large as a pigeon's egg. There were numerous nodules in the medulla of the somewhat thickened femur. All of these nodules exhibited the same histological structure as those of the integument.

In one case Paltauf found infiltration of the tonsils, palate, and upper half of the larynx, general enlargement of the lymphatic glands, interstitial infiltration of the liver similar to that found in leukæmia and pseudoleukæmia, considerable enlargement of the spleen, grayish-white interstitial infiltration of the testicles, and grayish-red color of the medulla of the bones.

Finally, Hallopeau and Jeanselme report, in addition to the tumors of the skin, similar products on the velum palati, in the larynx, axillary and inguinal glands, the spleen, liver, and kidneys.

Microscopic examination discloses, in the corium and subcutaneous tissues, an infiltration starting from the vessels, with typical small round cells. These are embedded in a close network of fine fibrillar connective tissue. At first the epithelium appears to proliferate, growing into cones, but later it undergoes atrophy above the nodules. The small-celled infiltration forms larger and smaller foci in places, but at their periphery it again assumes the character of an infiltration.

I found a similar infiltration in the kidneys, in the interstitial tissue between the urinary tubules. The kidney also contained nodules which dissolved at the periphery into distinct cellular infiltration, but in no part of the skin or kidney was the autochthonous tissue replaced by the round-cell infiltration, and the urinary tubules retained their normal position and histological characteristics.

On account of the reticulated structure and round-cell infiltration, Ranvier and numerous other writers applied the term "lymphadénie cutanée" to this process. Other observers also recognize a relation to leukæmia and pseudoleukæmia, inasmuch as diminution in the number of red blood globules and enlargement of the glands were found in addition to the anæmia which is peculiar to all cases.

It has been hitherto impossible to arrive at a definite conclusion in regard to the pathological significance of this disease. Paltauf, one

of the ablest investigators of the subject, opposes the view that mycosis fungoides is an inflammatory granulation tumor, an infectious inflammatory neoplasm (Neisser), lymphadénie cutanée, sarcomatosis, or a disease *sui generis* (Vidal). But, while he opposes these views of the various writers, he confesses that they are justified to some extent. While classing mycosis fungoides among the "vegetative disorders," Paltauf thinks it is probably allied to pseudoleukæmia and certain forms of lymphosarcoma.

This agrees with my own opinion. In view of its similarity to sarcoma or lymphosarcoma, I have always regarded mycosis fungoides as a form of sarcomatosis; but, on the other hand, I have merely intended to point out its similarity to sarcoma. At the same time I have claimed that it is a type of the diseases which are included by me among the sarcoid tumors, and to which lymphodermia perniciosa cutis, leukæmia cutis, and sarcomatosis also belong. There seems to me to be no doubt that there are clinical and histological transitions between the different forms. There is also a possible relation between these diseases, pseudoleukæmia, and leukæmia.

The doctrine of the possibility of the transition of pseudoleukæmia into leukæmia (Mosler) is constantly gaining ground. It is not until we possess convincing proof of the significance of cutaneous inflammatory and vegetative processes in hæmatogenesis that a step will be taken which will enable us to understand them. For years I have been inclined to this opinion, and I believe that in anæmic and predisposed individuals the eczematous dermatitides may furnish the starting point for these atypical vegetative forms.

In the majority of cases the process is confined throughout to the skin alone. It may, however, also involve the underlying tissues, the fasciæ and muscles. In rare cases it extends to the lymphatic vessels and glands; in still rarer instances to the parenchymatous and internal organs and to the marrow of the bones. There is also no doubt that the opposite condition may obtain.

Inasmuch as these relations are obscure, and mycosis fungoides in the majority of cases runs its course without glandular enlargement, pseudoleukæmia, leukæmia, or implication of the internal organs, it seems to be indicated, from a clinical standpoint, to regard it as a special clinical type.

The *diagnosis* of mycosis fungoides of the pure type is not difficult, except in the initial stages when the lesions often appear and disappear. The differential diagnosis from the eczema of anæmic skin may be difficult, especially when it is combined with flabby furuncles and abscesses. The diagnosis from the two processes which will next be considered is very difficult or even entirely arbitrary.

If we compare the frequent change of symptoms in mycosis fungoides with the occurrence of flabby fungoid and papillary abscesses and granulations in the eczema of anæmic individuals and in pemphigus, and also with the symptomatology of frambœsia or yaws (page 476), it will be evident that many mistakes are possible and that much is still involved in obscurity.

Treatment has been of no avail. Köbner alone has seen recovery in a recent case. Doutrelepont reports improvement in advanced cases under arsenic treatment, but I have seen no effect from this method. Bazin reports a spontaneous recovery after erysipelas. Nothing remains, then, except symptomatic treatment.

II. LYMPHODERMIA PERNICIOSA (MIHI).

In 1885 this disease was first described by me, and has since been observed by a few other writers. It began with the symptoms of a partly diffuse, partly localized, scaling, moist, and intensely itching eczema, which gradually resulted in diffuse, soft swelling and thickening of the affected parts. Then cutaneous and subcutaneous, doughy or firm, in part ulcerating nodules developed, the glands and spleen enlarged, with severe affection of the entire organism, leukæmia set in (absolute increase of the number of white blood globules) and was followed by a fatal termination. There was general pallor of the skin, and the face, ears, forehead, lips, and integument of the thorax and arms exhibited shapeless nodular thickening. At the autopsy the spleen was found to be enlarged fourfold, the marrow of the sternum, vertebræ, metatarsi, and long bones was grayish from the excess of leucocytes, and leukæmic nodules were present in the pleura and lungs. The nodules of the cutis, which were situated mainly in the adipose layer, were also leukæmic tumors.

Although I attempted to fix the clinical distinctness of this form by its nomenclature, still I suspect that an etiological and nosological relationship will develop between the different forms of mycosis fungoides, lymphodermia perniciosa, and certain forms of sarcomatosis cutis.

This view is favored by the occurrence of certain forms of eczema-like disease which have come under my observation. In anæmic females the integument of the face, trunk, upper and lower limbs, with the exception of larger or smaller islets of healthy skin, became pale red, doughy, smooth, with a satiny gloss ; there was also a development of a uniform, firmer mass extending from the deep corium into the subcutaneous cellular tissue. The face, limbs, neck and thorax appeared enlarged, the wrinkles and folds of the forehead and neck were more distinctly marked. The parts were tender on pressure and occasionally pained spontaneously. They itched intensely, but no notable effects from scratching were produced,

because the contact of the nails caused severe pain. The insomnia was extremely distressing.

The resemblance to the early stage of lymphodermia perniciosa was great, but the conditions are not identical. In the latter affection the skin was dry from the start, hard, lobulated; in the other forms it was constantly smooth, despite the partial fine desquamation, and satin-like to the touch.

These forms, which have been described by me in other places, present a very grave prognosis, because they may pass into leukæmia or pseudoleukæmia, or even into mycosis fungoides. One patient died in a few months from increasing weakness and numerous lymphatic abscesses; another died after increasing emaciation and constantly recurring firm but indolent (perhaps leukæmic or pseudoleukæmic) furuncular nodules and abscesses; a third patient died after the development of leukæmic tumors and true leukæmia; a fourth exhibited upon the hypogastrium and genitalia recent and gangrenous tumors like those found in mycosis fungoides.

We thus find sufficient transitional forms to uphold the idea of genetic unity, although no transition to my lymphodermia perniciosa has yet been found.

Many writers have already expressed decided opinions in regard to these affections, and hence the diagnosis of the individual cases and forms of disease vibrates to and fro in a confusing manner. Thus, Vidal regards my lymphodermia perniciosa as a form of mycosis fungoides. This is also the opinion of Hallopeau and Paltauf. The latter considers only the histological findings, although they are not entirely clear. At the same time he is compelled to acknowledge the great difference in the clinical history of the two diseases. This is shown chiefly in the deep location of the nodules in lymphodermia, and their superficial situation in mycosis, as well as by the general clinical characteristics

Here, again, the indecision in the question of leukæmia and pseudoleukæmia constitutes an element of doubt in regard to the pathological position of these diseases, added to which is the difficulty of deciding whether the skin or internal organs are the primary site of the disease.

Biesiadecki observed undoubted leukæmic tumors of the skin in a case of true leukæmia. Numerous flat, raised, pale-red, smooth little nodules, from the size of a millet seed to that of a lentil, were found in the face and upon the back. They consisted of lymphoid cells. Hochsinger and Schiff observed a similar condition in a child of eight months; the case was described as "leukæmia cutis."

III. SARCOMA AND SARCOMATOSIS CUTIS.

Sarcoma cutis is rare, especially when it occurs in the shape of

isolated sarcomata which have developed from slight injuries and cicatrices or upon apparently healthy skin. Thus, I have seen a spindle-cell sarcoma develop, within a few weeks, upon the site of a healed furuncle on the anterior surface of the thigh; another occurred in the shape of an umbilicated tumor, as large as a dollar. between the shoulders. Both growths were extirpated, and no relapse occurred during the many years which have intervened.

Great clinical and pathological importance attaches to the somewhat more frequent general sarcomatosis of the skin.

These forms of disease have recently been interpreted in various ways. The long-known, typical pigment sarcoma corresponds in great measure to the generally accepted notion of sarcoma, but in regard to the other forms the same doubt arises as in the case of mycosis fungoides and lymphodermia. In some cases the structure of the tumors does not correspond exactly to that of pure sarcoma or lymphosarcoma, or it closely resembles that of lymphoma, or the growths are associated with similar ones on the nasal and buccal mucous membrane or with pseudoleukæmia and leukæmia. According to the standpoint from which these growths are considered, their classification will differ greatly.

According to my experience, we are justified in distinguishing three types of general sarcomatosis of the skin:

(*a*) *Typical melanotic sarcoma*, of general diffusion, starting from a primary focus in the skin—for example, on the toe, labium majus, forehead, or trunk. The latter usually develops from a warty nævus in the shape of a blackish-blue, mushroom-like, spongy tumor. The general outbreak occurs within a few weeks or months, often immediately after the extirpation of the primary nodules. The secondary tumors appear as bluish-black, firm, moderately tender nodules from the size of bird shot to that of a cherry or larger. At first they are regional in regard to the primary tumor, or arranged in rows corresponding to the lymphatic vessels. These coalesce in a few months (after enlargement and homologous degeneration of the lymphatic glands and ulceration of some of the nodules) into bluish-black, nodular, firm, painful infiltrations which occupy large areas of the abdomen trunk, and limbs. Upon autopsy a similar degeneration of the internal organs is found.

Otherwise the sarcomatous growths occur as metastases of a pigment sarcoma which has already developed in the internal organs—for example, in the enormously enlarged liver, the intestines, etc.

Histologically they form a vascular round- and spindle-cell sarcoma, in places a giant-cell sarcoma, with very abundant intracellular and intercellular, granular and diffuse deposit of pigment.

In a case now under observation at my clinic there are, in addi-

tion to the bluish-black nodules, flat nodules as large as a bean or hazelnut, which resemble the nodules of the second type by their red color, their consistence, and the tendency to involution.

As a rule the process proves fatal within a few months from its appearance on the skin, and is unaffected by medication.

(*b*) A second typical form of sarcomatosis of the skin was described by me in 1879 as "*idiopathic multiple pigment sarcoma.*" I have seen sixteen cases, all in men, and others have been reported by various writers. It always begins upon both feet and hands, and advances by separate growths along the legs and arms until, at the end of two to three years, it appears upon the face and trunk. We find reddish-brown, later bluish-red, round, moderately firm nodules, from the size of a pea to that of a bean, which are in part separate and irregularly situated, in part arranged in groups and diffuse infiltrations varying from the size of a quarter to that of the palm of the hand. The flexor and extensor surfaces of the feet and hands are puffed up, nodular, shapeless, very painful on pressure and also spontaneously, the fingers thickened in a spindle shape, separated from one another. Walking and manipulations with the hands are impeded to a marked degree on account of the rigidity of the skin. After lasting several months the older nodules sink in and disappear in part, or even entirely, leaving darkly pigmented, cicatricial depressions. The plaques composed of groups of nodules also atrophy in the centre and then form a firm, brownish-red wall, covered with hard, dry scales, and surrounding the central pigmented cicatricial depression. Many nodules grow soft, but ulceration or necrosis *en masse* rarely takes place. In one of my cases the disease began upon the backs of both hands alone, in another upon the sole of the foot, in the shape of a few bluish-red nodules as large as a pea. In a case now under observation the feet and hands are the site of a diffuse, puffed-up, very hard and painful, bluish-red infiltration with very few nodules, while a few older nodules and plaques are scattered over the rest of the body. At the end of two to five years or more, nodules from the size of a pea to that of a nut, appear upon the eyelids, the nose and its mucous membrane, the cheek, lips, and various parts of the trunk. These are dark bluish red in color, spongy to the touch, and, as they ulcerate upon the surface, expose a tissue suffused with blood. Glandular enlargement does not appear to be peculiar to this type of sarcoma, apart from occasional sympathetic enlargement, as, for example, in gangrene of the foot. Fever, bloody diarrhœa, hæmoptysis, and marasmus soon set in at this stage, and are followed by death. At the autopsy similar nodules are found in large numbers in the lungs, liver, spleen, heart intestinal tract; in the descending colon they are especially dense and apt to be necrotic.

Histologically we find a round-cell sarcoma, except that in a few places the characteristic spindle-cell sarcoma is seen. A peculiarity of this type is the presence of capillary hæmorrhages, which explain the later bluish-black pigmentation of the originally bluish-red nodules, and also the excessive hardness of the diffuse infiltrations around the groups of nodules (deposit of fibrin).

So long as this form is confined to the hands and feet it may be mistaken for papular syphilide, and later for gummata, lupus, or leprosy.

The *prognosis* is unfavorable, even in those cases which come under observation as soon as the first nodules develop. In my experience the further development and fatal termination could not be prevented by extirpation, local or general treatment, or the administration of arsenic. Köbner states that he obtained temporary improvement in one case. The disease runs a slower course than other forms of sarcoma (three to eight years or more).

Treatment, which is urgently indicated on account of the great pain in the firmly infiltrated hands and feet, is confined to meeting the symptomatic indications: emollient ointments, plasters, baths, compress and bandage. Ulceration and necrosis of the nodules are treated according to general surgical principles.

(*c*) The *third type of sarcomatosis cutis* is characterized by multiple cutaneous tumors, which form quite a distinct clinical picture, but pathologically and histologically are less sharply defined. Their relations to leukæmia and pseudoleukæmia are the subject of much contention.

As illustrations we may mention two cases under my observation in vigorous men, and one case in an old woman.

In the former the trunk and limbs exhibited more than one hundred bluish red, flat or somewhat prominent patches as large as a finger nail. Upon feeling them, firm, elastic nodules were found deep in the corium and extending into the panniculus; their lateral borders were not sharply defined. In addition, palpation revealed some nodules over which the skin was neither prominent nor injected. In one patient the nodules were very painful; in the other they were merely a little tender on pressure. Both recovered, in three to four months, under methodical arsenic treatment, the nodules disappearing completely. No glandular enlargement, abnormality of the blood, or other anomaly of nutrition could be detected in these patients.

In the old woman, who has suffered from the disease for three to four years, about a hundred tumors are found upon the shoulders, posteriorly and anteriorly, over the clavicles and upper part of the thorax. The nodules are hemispherical, firmly elastic, of the size of a pea to that of a nut; the smaller ones are bright red, the larger

ones dark bluish red, smooth and shining on the surface, moderately painful on pressure. There are also flat, raised plaques, as large as the palm of the hand, with a central depression and indentations of the borders, so that there is a striking resemblance to the tomato forms of mycosis fungoides. An equal number of nodules were found scattered over the dorsum and sacrum, the chest and abdomen. and a few upon the limbs. On the whole, they were merely a further development of the variety described above. This patient was also treated with arsenic. After six to eight injections of arseniate of soda the majority of the nodules were flattened and collapsed by one-half or two-thirds. Since that time, however, most of the nodules have continued to grow, despite the constant use of arsenic.

A large number of analogous cases have been recorded by various writers. Many were completely cured by arsenic, but others ran a fatal course.

The *microscopical* appearances resemble those of round-cell sarcoma, but not completely. The picture is not exactly that found in leukæmic tumors of lymphomata. Leukæmia has been present in some cases, pseudoleukæmia in one case. In Arning's case lymphomata were found on the palate, gums, and tongue. In all of our patients the blood findings were normal. The same relations are evident here as in the case of mycosis fungoides and lymphodermia perniciosa. The question will only be cleared up after the observation of a larger number of cases and further examinations of the blood.

The *diagnosis* of this form of sarcomatosis from the previously described tumors, which it resembles most closely, is, however, not so impracticable, histologically, as Paltauf believes, and clinically the differentiation is imperatively indicated. In the former regard importance attaches to the location of the cellular infiltration in the deeper layers of the corium and the subcutis, while the papillary layer long remains intact. This renders possible the clinical diagnosis from mycosis fungoides, in which the uppermost layer of the cutis and the papillary layer are affected from the start and sometimes exclusively. From a clinical standpoint the curability of these forms by arsenic, and their more favorable prognosis, will also serve to distinguish them from the other affections. In making a differential diagnosis from lymphodermia perniciosa, I may state that the dryness, scaling, diffuse infiltration of the skin, and intense itching observed in the latter are entirely wanting in the form of sarcomatosis under consideration, except that the nodules may itch upon their first appearance. It is hardly possible to mistake the condition for idiopathic multiple pigment sarcoma.

Treatment by subcutaneous injections of arsenic (sodium arseniate 0.02 every second day) should be employed in every case.

LECTURE XLVIII.

CARCINOMA —THE THEORY OF CANCER—ITS FORMS—EPITHELIOMA —CONNECTIVE-TISSUE CANCER—PIGMENT CANCER.

CARCINOMA.

CANCER is a malignant neoplasm, the essential characteristic of which is now assumed to be certain atypical, proliferative growths of the epithelium, the so-called cancroid cones and spheres, associated with an inflammatory loosening of the embedding tissues.

This conception of the nature of cancer has only developed in late years after manifold changes of theory. In former times cancer was regarded as a tumor which began as a hard nodule and later was converted into the cancer nodule proper, undergoing fungous proliferation, then ulceration, and finally resulting in death by general marasmus (cancer cachexia). When the anatomical epoch of medicine began, the attempt was made to furnish a positive anatomical basis for the changing clinical theory of cancer. Some regarded the cancer cells as the characteristic element, but it was soon found that they could not be distinguished from physiological structures, from proliferating epithelial cells. Rokitansky took a step backward to the old ideas; although he erected an anatomical scheme, he regarded the malignancy—*i.e.*, the clinical character—as a co-ordinate element. Anatomically he held that cancer is composed of proliferating and rapidly perishing nuclei and cells (the cancer mass) embedded in a connective-tissue stroma.

Epithelioma proper was included among the cancers by Rokitansky and Schuh, evidently on account of its occasional malignant character. Others excluded it from this class because it persists for a long time as a local affection, and called it pseudocancer or cancroid (Lebert) or epithelioma (Hannover).

In the following period the clinical character was almost entirely neglected and the structure of the tumor alone determined its significance. Under Virchow's teaching a large series of tumors formerly placed among the cancers were classed with the sarcomata. An alveolar structure with epithelial contents was regarded as necessary to the notion of cancer, and epithelial cancer became the cancer, *κατ' ἐξοχήν*, although there had previously been a tendency to exclude it from the category.

Attention was now concentrated upon the histogenesis of the epithelial proliferations. Virchow and Förster regarded as cancers only those epithelioidal tumors whose elements were derived from proliferation of the connective-tissue corpuscles, independent of preexisting epithelium of the rete and glands.

Thiersch, on the other hand, attributed every cancerous proliferation to preformed epithelium. The Remak-His developmental theory, according to which the formation of all physiological tissues could not proceed within the limits of the three germinal plates, was extended to pathological new formations. These views were soon adopted by the majority of pathologists. According to Thiersch, the morphology and histogenesis of carcinoma reach their height in the demonstration of the proliferating epithelial cones, *i.e.*, the embedded cells, the cancer mass. The second element, the stroma, was entirely neglected. Hence cancer looked, according to Thiersch, the same as many benign structures which were due to epithelial proliferation, such as molluscum verrucosum and certain adenomata.

Billroth then laid stress upon the infiltration of the connective tissue—*i.e.*, the existence of the cancer stroma—as necessary to the character of cancer. Indeed, Billroth returned, in part, entirely to the old views, inasmuch as he held fast to a connective-tissue cancer dependent on epithelial origin. Others also returned to the views of the first period of Rokitansky, and assumed all sorts of combinations of stroma and cells in cancer, which were called sarco-carcinoma, fibro-sarco-carcinoma, adeno-carcinoma, etc. The pure epithelial character of the cancer mass was thus no longer regarded as indispensable, although it might not be entirely wanting.

We have also returned to the Rokitansky period in another sense: we hold that a purely histological characterization of cancer is no longer sufficient. Even Thiersch finally acknowledges that the conception of cancer must be clinical.

We may define cancer, therefore, as a malignant neoplasm which consists of a proliferating epithelial cell mass, arranged in an alveolar, cone-like or tube-like manner, together with a connective-tissue stroma in a condition of inflammatory infiltration.

This definition applies to epithelial cancer. There are other forms to which it does not apply completely, and of which, indeed, a general definition cannot now be given.

Some of these forms of cancer attack the skin idiopathically, others only secondarily. I will describe epithelioma, connective-tissue cancer, and pigment cancer, as especially important in dermatology.

EPITHELIAL CANCER.

Syn. *epithelioma, cancroid,* skin cancer, pavement epithelial cancer, rodent ulcer. This is frequently seen in dermatological

practice, and is located upon the skin and the mucous membrane. It may be divided into (1) flat, (2) deep-seated or nodular, and (3) papilloma-like epithelioma.

The *flat epithelioma* generally develops upon apparently normal skin in the shape of one or more shining, pale-red or waxy, very firm papules as large as a pinhead. They are occasionally linear, but are usually grouped in an irregular, wart-like protuberance. They soon excoriate or fissure spontaneously, or are scratched on account of moderate itching, and are then covered with a little crust composed of viscid secretion and blood. Several years may elapse before this condition is appreciably changed; then the focus enlarges, somewhat more rapidly, by the appearance of new peripheral papules. These papules are very characteristic and may be readily removed with a blunt instrument. Each one looks like a white, smooth sphere with a mother-of-pearl gloss, and is easily broken between the fingers. It is composed of epithelial cells (nucleated, round, spindle-shaped, and caudate pavement cells, containing one or more nuclei and daughter cells), collected in clumps or arranged around a central mass. Various terms have been applied to this product, such as cancroid corpuscles, cancroid spheres, pearly bodies, globes épidermiques, etc.

Gradually deepening exfoliation finally exposes a raw surface, the flat cancer ulcer (rodent ulcer). It forms a round (triangular or polygonal, when it assumes larger dimensions), shallow loss of substance with sharp edges; the brown or yellowish-red, finely granular, irregular surface secretes a viscid fluid which dries into a varnish like coating. The base and edges are hard, slightly movable; the edges are partly smooth, partly strewn with firm pearly nodules which glisten like vesicles. In the middle there is often complete exfoliation of the epithelioma by the formation of a cicatrix, starting from the inflammatory infiltration of the cutis in which are embedded the cancer cones. On account of its firmness the cicatrix offers greater resistance to the latter than the inflammatory papillary layer of adjacent parts. Finally a flat cicatrix results and the cancer ulcer is reduced to a narrow groove enclosing it. As this process continues with the spread of the epithelioma at the periphery, it finally extends over large areas (forehead and temples, the cheek) in the shape of a narrow ring enclosing the entire cicatricial area (epithelioma serpiginosum). Occasionally the rim as well as the cicatricial area contains slate-gray pigment (chimney-sweep's cancer), although the affection does not assume the malignant character of pigment cancer. Finally the formation of new pearls at the periphery may cease and the cancer may thus heal locally at the end of fifteen to twenty years. As a rule, however, a new focus appears at an adjacent spot.

Flat epithelioma often begins as a flat ulceration of a seborrhœic area of the skin, or of a verruca senilis, or of a papillary wart.

During its course of ten to twenty years or more the flat epithelioma does not produce any bad effect upon the general system nor any enlargement of the neighboring glands.

In many cases, however, it is converted into the *nodular or deep-seated epithelial cancer*. The latter, moreover, often appears as a primary affection. It develops in the shape of closely aggregated, flat or moderately prominent, very firm and somewhat translucent nodules, from the size of bird shot to that of a pea, which pass through the entire cutis and extend into the subcutaneous cellular tissue or start from the latter. In the course of months and years it grows into a spherical or flat, hard tumor, whose surface is shining, waxy or rosy, traversed by vessels, irregularly nodular. As the result of spontaneous retraction the centre is often drawn in like an umbilicus; the edges are steep and smooth, or dotted with cancroid corpuscles, or they are everted and pass sharply into the adjacent healthy parts. Later the growth extends into the deep layers of the skin as a hard, rigid mass which is only recognized on palpation. Here and there it appears in the vicinity of the central tumor as apparently isolated nodules. After a variable period ulceration occurs. This may give rise to flat ulcers, such as were previously described, or it may result in rapid softening of the deeper parts, above which the skin is bluish red and thin, and finally is lost, exposing a deep cancerous ulcer. The ulcer is crater-shaped, irregular, with steep, hard, dentated edges, from which cheesy, comedo-like plugs (the epithelial pearls) emerge on pressure. It secretes a viscid fluid, occasionally ichorous if the tissues undergo rapid destruction. With the spread of the cancerous infiltration to the underlying tissues, is leads in a variable period (a few months, several years) to destruction of the cartilage, muscles, and bones. In parts the tissues drop out by necrosis *en masse*, healthy granulations sprout from the edges, and even a new epidermis may form. But the process extends in other directions, possibly acquiring the character of medullary cancer, and death occurs in a shorter or longer space of time after enlargement of the adjacent lymphatic glands.

Papillomatous epithelioma (malignant papilloma) runs the most rapid course. It appears as a broad, hard tumor, or is seated on a pedicle like a mushroom and projects one-half to one and a half centimetres from the surface of the skin. Its surface is smooth, slightly umbilicated in the centre, and bordered by upturned edges. At first it is red or slate gray, shining, dry like parchment; later it exfoliates, becomes excoriated and fissured, and breaks down gradually into ulcers, at first flat, later deep and ichorous. If the malignant papilloma is situated on cutis which is not much infiltrated

(upon a flat epithelioma) it may run a favorable course, but it is rapidly destructive when located on an infiltrated cancer.

These three types of epithelioma often occur singly, but they may also be combined in the same individual.

All forms of cancer of the skin occur most frequently upon the face, chiefly the eyelids and adjacent parts, the integument of the cartilaginous and bony parts of the nose, the lips, forehead, and lateral parts of the cheek. The disease is sometimes confined for years to the same locality, or several places are affected at the same time or by gradual extension from the original focus. The eyelids, temples, and bridge of the nose are often covered for years by a flat epithelioma, which, after central cicatrization, may extend to the cheeks, lobe of the ear, and upper lip. In other cases the cancer extends from the lid to the conjunctiva and thence into the orbit, without involving the globe for a long time. Xerosis corneæ occurs secondarily as the result of cicatricial ectropium. Upon the forehead the epithelioma often becomes serpiginous and retains the character of flat epithelioma for fifteen to twenty years; or it soon becomes nodular and extends to the bones. When it attacks the cutaneous part of the nose, the latter becomes eroded and shrivelled, and the cancer mass then infiltrates the vomer, superior maxilla, and alveolar processes. Upon the lips we often find flat, papillomatous cancer at the start, but it soon becomes infiltrated and extends to the buccal mucous membrane. After the bone has been attacked the degeneration spreads more rapidly and results in perforation of the hard palate, loss of the teeth and alveolar processes, perforation of the antrum of Highmore, the frontal sinuses, the cranial bones, exposure of the brain. In places we find a conversion into medullary cancer, extensive ichorous ulcers, necrosis *en masse*, fungous proliferations from adjacent tissues, involvement of the neighboring lymphatic glands, marasmic fever, and death.

The genitalia are a less frequent site of epithelioma. It begins as flat epithelioma upon the glans, the edges of the meatus, the integument of the penis, after a relatively short period infiltrates, with a very hard swelling, the dorsal lymphatics, the corpus cavernosum, and the inguinal glands. It terminates fatally in two to three years. The process is still rarer upon the female labia. Central cicatrization and peripheral extension sometimes convert a flat epithelioma of the genitalia into an extensive rodent ulcer which extends over the mons veneris and the inner surface of the thigh.

A much less common event is primary epithelial cancer of the umbilicus, nipple, or any part of the trunk. It is somewhat more frequent on the upper and lower limbs, where it generally starts from exuberant granulations (in elephantiasis arabum and lupus).

Upon the mucous membrane of the mouth and nose, conjunc-

tiva, vagina, and rectum epithelioma occurs either primarily, or secondarily as an extension of disease of the adjacent integument. Cancer of the tongue and mucous membrane of the cheek is much more frequent. Here it develops very often from the gray epithelial callosities, the so-called leucoplakia, which may or may not be due to syphilis. Upon the tongue it begins as a flat, red, raw surface, from the size of a pea to that of a bean, occasionally covered with whitish dots, painful spontaneously and on pressure ; it also occurs as a fissure of the border or dorsum of the tongue, with a soft base. At a later period a firm nodular infiltration develops beneath the flat ulcer. In other cases this infiltration precedes the superficial ulceration. Shooting, lancinating pains which radiate toward the ears, and enlargement of the submaxillary glands, set in after the lapse of one to three years and presage the lethal termination. Upon the mucous membrane of the cheeks epithelioma is rarer ; it is usually flat, but often mushroom-like, with turned-up edges.

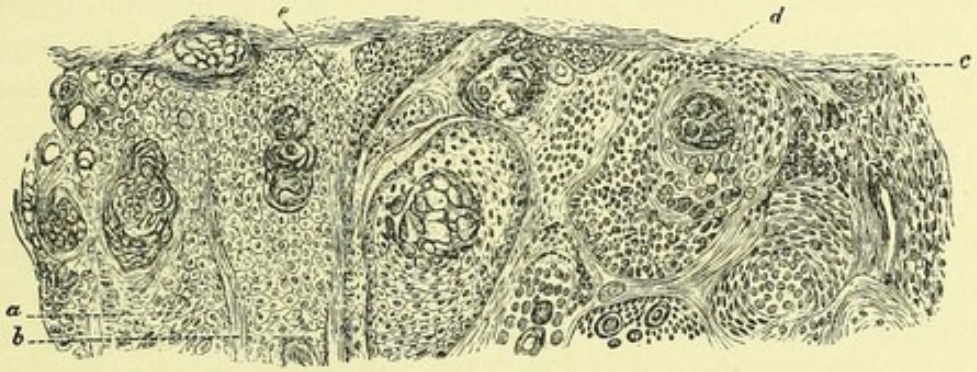

FIG. 60.—EPITHELIOMA (VERTICAL SECTION).

a, *d*, rete pegs greatly prolonged into deeper parts ; between them shrunken papillæ, *b* ; in each (*c*, *d*, and elsewhere) cancroid corpuscles ; *c*, horny cell layer.

Primary epithelioma of the portio vaginalis is often seen in dermatological practice, especially in reference to differential diagnosis from syphilis. It is often cauliflower-like or looks like a finely granular raw surface.

The *anatomy* of epithelioma has not been established definitively. It consists of inflammatory cutis tissue (infiltrated with lymphoid and proliferating cells, traversed by dilated vessels, and soaked with serous lymph in the distended meshes), which is traversed by a network whose bands contain epithelial proliferating cells and cancroid corpuscles (Fig. 60). The rete cones force the bands of the network deep inward, like the fingers of a glove, and these prolongations are connected with adjacent ones, forming an epithelial network.

The origin of the proliferating epithelial cones is an important point in regard to the histogenesis. Virchow attributed them to

proliferation of connective tissue corpuscles; Thiersch, Recklinghausen, and others, to outgrowths from the rete pegs and the glandular epithelium; while Köster regards them as a product of the proliferation of the endothelial cells of the lymphatics. In my opinion Thiersch's theory is undoubtedly true in regard to flat epithelioma. In sections taken from the borders we can follow the successive prolongations of the rete pegs into the cutis, and can also observe that this is associated with the formation of lateral proliferations and cancroid corpuscles, probably in the same way that the epithelial processes from the lining cells of the glands are put out. It is not until a later period that inflammatory infiltration of the corium develops, and there are times in the formation of cancer when it is impossible to determine histologically whether we have to deal with a benign, atypical epithelial proliferation, such as I have mentioned under the heading of lupus (page 552, Fig. 54), an actively growing papillary wart, or real cancer. The cancerous character only becomes more distinct with the increase of the inflammatory infiltration and the more general advance of the epithelial growths. The inflammatory loosening of the tissue, the dilatation of the lymph spaces, the cleavage of the tissues according to a scheme mapped out by the vessels (Rindfleisch), pave the way for the advance of the epithelial cones, while cicatricial formations oppose an obstruction and thus render possible the local spontaneous recovery of the cancer. It has been rendered certain, however, by a series of investigations that, apart from the endothelial and epithelial cells, all the other formed elements—connective-tissue corpuscles, the elements of the walls of the vessels, muscle cells, lymph cells—may undergo proliferation and produce epithelioid (*i.e.*, cancer) cells, thus aiding in the increase of the cancer mass.

The further symptoms of tissue consumption, suppuration, gangrene, are merely secondary to the retrograde metamorphosis (fatty, mucoid, colloid degeneration) undergone by the epithelial elements which are incapable of higher organization. There is also necrosis *en masse* in consequence of the elimination of larger particles of tissue, especially bone, from the nutrient surroundings.

The *etiology* of cancer in general is still obscure, while considerable light has been shed on that of epithelioma. Heredity plays no part, while advanced age is a general, and certain anatomical conditions special, predisposing factors. We have seen epithelioma, however, in several patients between the ages of eight and eighteen years. Males furnish a larger contingent of cases than females (100 : 30 according to Von Winiwarter).

Certain local, congenital or acquired, histological conditions of the skin undoubtedly furnish the exciting cause for the development of epithelioma, as soon as they involve a change in the nutritive

relations between the papillæ and connective-tissue stroma on the one hand and the rete and pigment on the other. Among such conditions may be mentioned : pigment, papillary, and sebum warts, which first undergo epithelial proliferation, either spontaneously or after repeated irritation (tobacco juice on the lips, repeated mechanical injury), and are then transformed into epithelioma by sending out epithelial processes into a corium which is in a condition of inflammatory softening or has become less resistant by senile atrophy ; furthermore, granulations, above which the normal formation of epidermis is delayed and prevented by mechanical or local nutritive conditions, as in ulcers of the foot, lupus, xeroderma pigmentosum ; finally, the gray epithelial callosities of the mucous membrane of the tongue, lips, and cheeks, known as psoriasis mucosæ oris (leukoplakia buccalis, Schwimmer), which may or may not be syphilitic, and which often give rise to epithelioma.

The demonstration of various cocci and bacilli as causes of carcinoma has not been successful. The same is true of psorosperms, which Pfeiffer has lately (1893) put forward as the cause of the disease, but which Noeggerath and others have recognized as only atypical and degenerated epithelial forms.

It has also been claimed that the cancer juice possesses a " specific, toxic, and infectious character," and this has recently been isolated by Adamkiewicz in the shape of a watery extract, " cancroin." As this poison, according to Adamkiewicz, is the product of the cancer cells, and the latter are independent living beings, protozoa (" meat-eating sarcolytes "), the development, destructive action, and metastasis of cancer would be thoroughly explained, if Adamkiewicz's theory were confirmed by others.

The *diagnosis* of the fully developed forms of epithelial cancer is evident from the symptoms. A decision is sometimes difficult in regard to the initial forms when situated on the genitalia, where they may be mistaken for chancre. The diagnosis will be cleared up by the early lancinating pains and the induration of the glands. It may also be difficult to differentiate cancer of the buccal mucous membrane or tongue from syphilitic gumma, so long as the characteristic hardness of the base is not recognizable. This is also true in regard to tubercular ulcers of the tongue, especially if syphilitic symptoms are present at the same time. In such cases histological examination will furnish the best means of diagnosis, but the test of antisyphilitic treatment should not be omitted.

The *prognosis* of epithelioma of the skin is more favorable than that of all other forms of cancer in this locality. It is most favorable in flat epithelioma, which spares the deeper tissues for years, may heal spontaneously, and never results in infiltration of the glands or marasmus. Less favorable is nodular cancer, because it is much

more destructive locally, and in later years gives rise to enlargement of the glands, marasmus, and death. The fatal termination is accelerated when the growth assumes the character of medullary cancer. Even in the early stages of the last two forms the prognosis is favorable in regard to the results of proper treatment. After extirpation the epithelioma does not relapse at all, or, as happens more frequently, the relapse is local and is so moderate that it can be easily overcome. Even when there is widespread infiltration and destruction of tissues, suitable treatment will confine the disease locally or eliminate it, improve the general condition, and delay the threatened fatal termination for months and years.

Treatment with internal remedies has hitherto proven useless.

The method recommended by Adamkiewicz, viz., subcutaneous injections of "neurin," has met with no success. In my opinion its physiological action is to be classed with all those methods (injections of Koch's tuberculin, H. Hebra's thiosinamin, Mosetig's leucrin, etc.) which give rise to a more or less intense inflammatory process in the morbid focus. The use of these remedies has never resulted in recovery.

The only proper method of treatment is the direct removal of the cancer. The same methods may be employed as in the treatment of lupus (page 555). Flat and moderately deep nodular and warty cancer may be removed with the sharp spoon or with lunar caustic, chloride of zinc or caustic potash, or by the application of Vienna paste, Canquoin's paste, lactic acid (pure or in paste), arsenic paste, or ten to twenty per cent pyrogallic ointment. The two latter pastes, smeared on linen and applied continuously for three to six days, destroy only the diseased tissue. Moreover, the pyrogallic ointment is painless in its action. Deep-seated nodular cancer of the lips and other regions is best removed at once with the knife. The galvano-cautery and Paquelin cautery may also be employed. When the cancer is widespread and no longer adapted to surgical removal, the caustics are again indicated to prevent the further spread of the process and the destruction of the tissues. In addition to those mentioned above, we may also use creosote, either fluid or mixed with pulv. liquirit. and opium, or arsenic made into a paste. For example: creosot. 20.0, arsenic. alb. 0.3, opii puri 0.15. This paste may only be applied to small surfaces.

Relapsing nodules must be destroyed as soon as they appear. If we are active in this particular we may not alone avoid annoying deformity, but may also prevent death from cancer marasmus.

Of the CONNECTIVE-TISSUE CANCERS of the skin three forms of equal malignancy may be mentioned.

Carcinoma lenticulare develops upon a breast which is filled

with nodular cancer, or, after extirpation of the latter, as a relapse upon the integument, which is either infiltrated and hard as a board or is still soft. More rarely it develops primarily in the skin in the shape of firm, shining papules and nodules as large as a lentil or larger; these soon undergo ulceration. In this form the infiltration soon penetrates the cutis, where it spreads as the result of hyperæmia and induration (like a chronic lymphangitis of the cutis), so that the thorax looks as if surrounded by a cuirass. It consists of a dense fibrous network with a scanty deposit of cells in its narrow meshes. It occurs almost always in women, but I have recently seen it, for the first time, in a man of sixty years.

Carcinoma tuberosum occurs in old persons upon the face, hands, and other parts of the body, in the shape of lumps varying from the size of a pea to that of a hen's egg. They soon soften and ulcerate, and are associated with similar growths in the internal organs.

Carcinoma melanodes s. pigmentodes begins upon a circumscribed part of the skin—the back of the hand or foot, a finger, toe, labium—with nodules of the size of a shot to that of a bean. They have a graphite or blackish-blue color, and a partly firm, partly soft consistence comparable to that of a berry. A group of these nodules grows into a mushroom-like tumor, which soon ulcerates. Innumerable blackish-gray dots, papules, and nodules very soon appear; in part they are distributed irregularly, in part they follow the lymphatics; in places they coalesce into diffuse nodular infiltrations. The lymphatic glands become swollen; marasmus and death follow. The internal organs are abundantly infiltrated with similar but more hæmorrhagic nodules. They consist of a large-meshed, vascular, in places alveolar stroma, with nest-shaped or irregular heaps of small and large epithelial or spindle-shaped proliferating cells, and abundant pigment, derived in part from hæmorrhages, in part transuded directly from the vessels. The appearances correspond exactly to those of pigment sarcoma (page 601), and it is probable that mixed forms actually occur.

CLASS XI.

CUTANEOUS ULCERS.

LECTURE XLIX.

DEFINITION OF ULCER—GENERAL SYMPTOMATOLOGY—CLASSIFICATION—IDIOPATHIC INFLAMMATORY, SIMPLE, AND CONTAGIOUS ULCERS—ULCER OF THE LEG—CHANCRE—SECONDARY INFLAMMATORY ULCERS AND THOSE DUE TO NEOPLASIA.

A CUTANEOUS ulcer is a loss of substance in the corium, exposed directly or indirectly. Its secretion, as a rule, differs qualitatively from so-called laudable pus, and it recovers very slowly or not at all, because the tissues at the margins are undergoing progressive molecular disintegration.

According to this definition an abscess is not an ulcer, because it involves necrosis *en masse* and a tendency to recovery. This is equally true of a properly suppurating and granulating wound, and also of a loss of substance which, as in eczema and pemphigus, involves the epidermis alone.

An ulcer is not a primary formation. The place at which it originates must have been the site of an inflammatory or neoplastic formation, which either carries in itself the conditions necessary to progressive molecular disintegration, or in which the typical processes of recovery are disturbed by certain local or general influences. The former category include lupus, scrofulous and tubercular infiltration, leprosy, carcinoma and sarcoma, syphilitic gummata. The local factors which give rise to ulceration include : local obstruction to circulation, due to varicose veins, mechanical pressure, compression, scratching, chemical agents which destroy the young tissue, plasters and ointments, soiling of the granulations with fæces, saliva, and urine, necrosis and caries of the bones. The remote causes include heart disease and certain dyscrasic conditions, such as anæmia and marasmus, which either cause an infiltration of the skin predisposed to disintegration, or prevent the healing of the wounds on account of insufficiency of blood supply or imperfect metabolism of its juices.

If the inflammatory or neoplastic infiltration whose disintegra-

tion gives rise to the ulcer is eliminated spontaneously or artificially, or if those factors which interfere with the formation of granulations are removed, the latter will proceed to the completion of cicatrization as in every normal wound. Hence there is no sharply defined difference between an ulcer and a properly suppurating wound.

There is not the slightest reason, then, for ascribing to ulcers an ontological significance, as is still done occasionally at the present time. We do not understand how an ulcer of the leg may furnish a sort of vicarious outflow for suppression of menstruation or a hæmorrhoidal flux. In every case the development of the ulcer is thoroughly explained by the local and mechanical conditions, such as varicose veins, dermatitis, scratching, passive œdema, and hæmorrhage. A physiological connection between the ulcer and menstruation or hæmorrhoids cannot be detected. We are also unable to comprehend how otherwise intelligent pathologists will attempt, by inserting an issue in the arm, to antagonize the supposed disadvantages of the healing of an ulcer of the leg. No vital loss, whether due to inflammation or suppuration, is an element of health. Every loss of substance is an injury to the body, however it may have developed, and one which lasts months or years and is associated with copious secretion is certainly still more detrimental. Hence we should endeavor to heal all ulcers as rapidly as possible, and need not fear that the ulcer will "strike in" upon internal organs.

As the ulcers vary according to their kind, not according to their causation, they have certain general symptoms in common.

The base of an ulcer is grayish yellow, as a rule, infiltrated with pus because undergoing molecular disintegration, smooth or uneven. The edges are steep or slope gradually, smooth or jagged, sometimes more or less undermined; they may be movable or firmly adherent, soft or infiltrated by inflammation, easily bleeding. The immediate vicinity of the ulcer is in a condition of inflammatory swelling or is slightly changed; it may be almost normal, perhaps œdematous, sometimes hard, firm, and callous, or infiltrated by a specific new formation (lupus, miliary tubercle, carcinoma, syphilis). Small ulcers are usually circular or roundish; larger ones are irregular, crater-shaped, irregularly pitted or flat like an erosion. The size varies from that of a quarter to that of the entire circumference of a limb.

As a rule the secretion is unlike laudable pus. It is either abundant or scanty, thin, purulent, whey-like, mixed with a few cells, or transparent, viscid and sticky, odorless or foul-smelling, hæmorrhagic. It dries into crusts of various colors and dimensions. These may slope off like rupia, or, if the secretion is more scanty, merely form a sticky coating. It is also said that the secretion may contain

an excess of salts, especially phosphate or urate of soda, in arthritic ulcers. A blue color of the secretion has been observed occasionally and is attributed by some to the presence of blue vibrios. Others explain this color by the presence of pyocyanin and pyoxanthose. A specific odor has also been ascribed to ulcers.

Among subjective symptoms, it is to be noted that the ulcers are occasionally indolent or very painful (asthenic and erethistic ulcers).

In the course of an ulcer we distinguish a stage of destruction which may last weeks, months, or years, and a stage of repair into which every ulcer passes after removal of its direct cause; this corresponds to the condition of the normal wound. Some ulcers run a typical—*i.e.*, definite—course; others run an atypical course and their duration is indefinite. With few exceptions the local termination of all ulcers is the transformation into a healthy granulating wound and recovery by means of cicatrization.

The *prognosis* and significance of the ulcers to the affected part and the general organism depend upon the underlying anatomical change, and it is upon this alone that a rational classification is based. Ulcers may be divided accordingly into two classes: (1) those due to inflammation, and (2) those due to new formations. The inflammatory ulcers may be non-contagious or contagious, and each of these may be idiopathic or symptomatic.

Idiopathic, non-contagious, inflammatory ulcers result from idiopathic inflammations of the skin of all kinds, viz., acute and chronic dermatitis, abscesses, excoriations, eczema, vaccination pustules, and repeated disturbance of granulations by scratching, pressure, traction, retention of pus beneath crusts, irritating plasters, stasis of blood from constriction, varicose veins, etc.

Special practical importance attaches to the so-called *ulcus cruris*, or ulcer of the leg. In its development and course we may observe nearly all the conditions of ulceration. It occurs particularly in individuals suffering from varicose veins, such as many women after pregnancy, and individuals of both sexes who are compelled to stand many hours every day.

The first symptoms in such persons are occasional moderate œdema and pain in the ankle and sole of the foot, itching of the leg, and secondary scratching and excoriations. Small, superficial excoriations change into superficial, then deeper losses of substance. These lead to ulcers, the more rapidly the more the formation of granulations is disturbed by hæmorrhages, œdema, traction, mechanical injury by pressure, blows, complicating lymphangitis or dermatitis. In the subsequent course of the ulcers we find hardening of their edges from the frequent inflammations, and the tendency to the new formation of (cicatricial) connective tissue, constriction of the nutrient vessels by the cicatrices themselves, and the continuance of the original

etiological conditions. These convert the ulcer into a very painful and annoying disease which lasts for years or becomes incurable.

Ulcers of the leg are found mainly upon the middle lower third of the leg, on one or both sides, chiefly upon the anterior surface, in rare cases encircling the limb. The smaller ulcers are round, the larger ones irregular in shape and, according to their age, complicated with callous edges and more or less pronounced elephantiasic thickening of the limb.

It is distinguished from syphilitic ulcers, as a rule, by its superficial character, the slight degree of pain, and the absence of surrounding infiltration, corresponding to a gumma. Its mode of development is also shown by the chronic inflammatory condition of the surrounding parts.

Recovery will occur if the ulcer is not too deep and extensive, but is impossible when the involved surface is large and when the edges are hard and unyielding.

The *treatment* is conducted according to general surgical principles. When inflammatory symptoms are prominent they must be relieved by a horizontal or elevated position of the limb and the application of cold. The exfoliation of the disintegrated upper layers of tissue is hastened by the application of gypsum bituminatum pulverisatum (produced by rubbing oleum fagi with plaster of Paris), pulvis carbonis ligni tiliæ, Lister's dressing, sozoiodol powder, alumnol powder, etc. The formation of granulations must be carefully watched. They may be stimulated by a dressing of caustic potash 0.1, aq. dest. 50.0, or, if excessive, they may be removed by cauterization or scraping with the spoon, or restrained by caustic and astringent dressings, acetate of copper in solution, red precipitate ointment, alum, etc.

The continuous water bath is remarkably efficient in aiding the repair of the ulcers and in cutting short a complicating phlegmonous inflammation. Methodical compression by a flannel bandage, applied from the toes upward over the calf, aided by direct compression of the ulcer by means of emplastrum saponatum or a fixed plaster of Paris or gelatin dressing, renders it possible for the patient to walk around. The compression prevents dilatation and rupture of the small veins and capillaries, and thus also prevents hæmorrhages or an œdematous and exuberant condition of the granulations.

When the edges of the ulcer are extremely callous it heals with great difficulty. The edges may be brought closer to one another mechanically by loops formed of strips of plaster or by some other method of immovable dressing. Nussbaum recommends deep incisions outside of and parallel to the callous edges. In this way some of the vessels which supply constant hæmorrhages are destroyed, and the edges are brought nearer to one another. There is also fre-

quent opportunity of resorting to transplantation according to Reverdin's method, and, better still, according to Thiersch (complete covering of the granulating surface with flaps of skin taken from other parts of the body). As a matter of course, we should attempt to relieve the varicose veins, the demonstrable cause of the ulcer.

The operations for varicose veins include Schede's ligature method, and Englisch's plan of subcutaneous injections of alcohol, in order to produce adhesive inflammation and obliteration of the dilated veins. Apart from the occasionally dangerous complications (phlebitis, pyæmia, or embolism), these operations have a very limited effect, as they only affect certain of the veins. The only plan to be adopted, although it is merely palliative, is the constant wearing of a methodically applied flannel bandage or a well-fitting elastic stocking. The limb should be kept in a horizontal position as much as possible.

The *symptomatic, non-contagious, inflammatory* ulcers are the expression of a special dyscrasic or constitutional condition which either leads directly to inflammation and ulcerative disintegration of the tissues, or does so indirectly by preventing the normal termination of inflammation and suppuration which have developed in other ways.

This class includes the ulcers occurring in scurvy, gout, anæmia, general cachexia, acne cachecticorum, scrofula, and, in part, leprosy. Some of these ulcers reveal their etiology by their appearance. Thus, scorbutic ulcers are characterized by frequent hæmorrhages in the base of the ulcer, gouty ones by the gout stones (uric acid concretions); the anæmic (very rare) are characterized by their great pallor, slight reaction, slowness in the formation of granulations, and a scanty, thin, watery secretion. The most frequent and best known are the so-called scrofulous ulcers. These are generally called tubercular, but we think that, in view of their peculiar structure and course, the old term is preferable.

They are characterized by flabby, deeply undermined, easily bleeding, atonic edges, flabby granulations, thin, creamy secretion, slow repair, and give rise to queerly shaped cicatrices. They generally develop upon the inflamed integument over infiltrated, cheesy, suppurating glands (particularly in the neck), or over indolent and cheesy nodules from the size of a hazelnut to that of a walnut. The latter are usually grouped in twos or more along the lymphatic vessels, and display a great similarity to syphilitic gumma. Over carious bones the ulcers are funnel-shaped and surrounded by fungous granulations.

In the *treatment* of scrofulous ulcers the special local conditions must be taken into consideration, in addition to the general principles of treatment. We must also direct our attention to improving

the general nutrition (fresh air, suitable diet, cod-liver oil). Tubercular ulcers (page 558), due to true miliary tuberculosis of the skin, may be treated by caustics or by iodoform (iodoform. pur. 5.0, glycerin. 15.0) or iodol.

The only *contagious, inflammatory* ulcer is the chancre, the primary lesion of syphilis. According to the unicists, the chancre is the result of syphilitic products of all kinds; according to the French dualists, the soft chancre alone is produced by a special, so-called chancre virus, the hard chancre by the syphilitic virus; according to the German dualists, the chancre is produced by the chancre virus alone, has nothing in common with syphilis, and is preferably called a venereal contagious ulcer (Sigmund).

The typical *soft chancre* (chancre mou, simple, non inféctant, Ricord) is the most frequent form. It is a crater-shaped loss of substance, which looks as if bored out with a punch, has inflammatory, reddened, and swollen edges and base, suppurates freely, and is absolutely contagious. It runs a typical course of six to seven weeks, with a stadium destructionis which is also contagious, and a stadium reparationis in which it is converted into a healthy wound and is no longer contagious.

The so-called *hard chancre* (chancre dure, inféctant, Ricord) is also a typical form of ulcer. It develops from a soft chancre or from a nodule which forms at the site of infection after a period of incubation varying from several days to two or three weeks. The hard chancre is saucer-shaped, with slight secretion and a typical, sharply defined, almost cartilaginous hardness of the edges and base. It heals in a few days, is usually inoculable as such only upon non-syphilitic individuals, but persists as an induration many months after the ulcer has healed. We may also mention other forms of chancre, such as ulcus ambustiforme, phagadænicum, gangrænosum, serpiginosum.

The nosological importance of these different forms of chancre will vary according to the theoretical standpoint. It is here sufficient to say that all develop at the site of inoculation of the specific virus, and hence are idiopathic, specific, or contagious inflammatory ulcers.

Their course is typically circumscribed, so that the *prognosis*, so far as regards their character as ulcers, coincides with that of ulcers in general. But, as all the different types may or may not result in general syphilis, this must also be kept in mind in making the prognosis. As a general thing the typical soft chancre only gives rise occasionally to constitutional syphilis; the typical hard chancre does so almost constantly, and there is no rule in this regard concerning the other forms.

Apart from these circumstances, the indications in the *treatment* of chancres are the same as in the treatment of other ulcers. They

are treated according to general surgical principles; a dressing of emplastrum hydrargyri is particularly serviceable.

Among the second group of ulcers—viz., those due to new formations—attention has already been paid to the lupous, carcinomatous, sarcomatous, tubercular, and leprous ulcers. To these may be added the ulcerative syphilide, which is a symptom of constitutional syphilis (see page 577).

CLASS XII.

CUTANEOUS NEUROSES.

LECTURE L.

NEUROSES OF THE SKIN—THEORY—MOTOR, TROPHIC, AND SENSORY NEUROSES—PRURITUS CUTANEUS, UNIVERSALIS ET LOCALIS—PRURITUS SENILIS.

NEUROSES of the skin are diseases which are occasioned by a change in the function of the cutaneous nerves without demonstrable structural alteration in the skin.

These neuroses are threefold in character: (1) motor, (2) vasomotor (trophic and secretory), and (3) sensory neuroses. It is unnecessary to dilate upon the still undecided question of the existence of trophic nerves.

A type of the *motor neuroses* is *cutis anserina* (goose skin), in which the hair follicles are protruded in small, firm, pointed nodules, which are either covered with little scales or perforated by a hair. They are most numerous on the trunk and the extensor surface of the limbs. The phenomenon is due to contraction of the looped muscular fibres of the follicles.

The analogous condition known as lichen pilaris or a low grade of ichthyosis may be excluded from this category, because we cannot assume that the muscular bundles of the follicles are permanently contracted for years. By the term cutis anserina we refer merely to a neurotic contraction of the arrectores. It may follow direct or indirect irritation of the cutaneous nerves. The direct irritation is due to sudden changes of heat or cold, which likewise affects other muscles, as shown by tremor, deep or spasmodic respirations. Indirect irritation is due to cerebral stimulation following mental emotion, such as fright, delusions, etc. Strictly speaking, cutis anserina is a physiological process and occurs, under the conditions mentioned, in all normal individuals.

Trophoneuroses of the skin are nutritive disorders due to anomalies of the nervous system, such as angioneuroses, erythromelalgia, zoster, atrophy and hypertrophy along the course of injured nerves, gangrene in paralyses, etc.

Many other processes, such as erythema, urticaria, prurigo, ichthyosis, etc., have been classified as angioneuroses, trophoneuroses, neuropathic dermatoses, but, apart from the fact that the neuropathy really forms a part of very few skin diseases (even the diseases mentioned in the preceding paragraph—for example, zoster), do not constitute neuroses in the general pathological sense. They are characterized, each in its own way, by definite nutritive changes of the skin which, so long as their neuropathological relations are not clearer than at present, furnish the clinical characteristics for their diagnosis. For this reason it seems to me unwarranted, at the present time, to interpret and classify skin diseases differently from diseases of other organs—the lungs, heart, kidneys. The latter are undoubtedly, in part, of a purely neurotic character, but they are described and differentiated upon the basis of their anatomopathological changes and their clinical symptoms, even when they are associated with pathological conditions of the central or peripheral nervous system.

The true neuroses (idioneuroses, Auspitz) are those affections alone which correspond to the description given at the beginning of this chapter, *i.e.*,

Sensory Neuroses. These occur as excessive or diminished sensation—hyperæsthesia, paræsthesia, anæsthesia; or qualitative changes of sensation—pruritus, hyperalgesia, analgesia, diminution of the sensation of pressure or touch—which occur in hysteria and various diseases of the central nervous system and peripheral nerves —zoster, lepra, etc.

PRURITUS CUTANEUS.

This is a chronic disease of the skin, characterized by itching of a spontaneous nature—*i.e.*, without eruption or external causes. Hence the itching of eczema, prurigo, lichen ruber, etc., is not "pruritus" in the sense of an independent disease, but is merely a concomitant of the underlying disease. Pruritus is either diffused over the entire body or is confined to certain parts.

Pruritus universalis is manifested by the annoying sensation of itching, occurring in paroxysms a number of times during the day and night. An attack of itching is often excited by special circumstances—for example, heat, the warmth of the bed, violent movement, or forced rest. Mental emotions exercise an undoubted effect. The mere fear that the itching may begin, and that, as in a theatre, there will be no opportunity for scratching, is sufficient to excite an attack forthwith.

The itching begins irregularly here and there, at first a gentle tickling which the patient can resist for a time; then the sensation becomes more violent and the patient begins to exercise pressure or

to scratch moderately. Now the itching begins with enormous violence, and is more severe the longer the struggle against it has lasted. The itching is so violent that the sufferers are forced to disregard the proprieties. Scratching with the nails is often insufficient, and the patients use rough bodies and brushes in order to gratify the nervous sensation. At the start the scratching itself is the cause of increased itching or the appearance of urticaria, but the patients are unable to resist.

The itching does not cease until the skin is hyperæmic and bleeding in streaks here and there, and the sensation of burning is felt. The nights are especially distressing, because an attack of itching is often experienced while the patients are undressing. Sometimes they fall asleep at once, but are soon awakened by an attack, are tortured for several hours, leave the bed several times, apply cooling substances to the skin, etc., until morning, when they fall asleep exhausted.

The only objective appearances on the skin are those due to the scratching—*i.e.*, irregularly situated scratch stripes and patches, which are either fresh or darkly pigmented, according to their age. In other cases the pruritus is associated with a dry condition of the skin, or perspiration ceases almost entirely except at the joint flexures. Urticaria occurs almost constantly during the scratching.

The patient may suffer from impaired nutrition, psychical depression or exaltation, as the result of the insomnia and sometimes of the etiological factor of the disease. Occasionally there is danger of mental alienation, even of suicide, during a paroxysm of itching.

In some cases the *etiology* of general pruritus is tolerably well known. *Senile pruritus*, occurring in old age, may be regarded as the result of senile marasmus. The skin of such old people is very often flabby, dry, wrinkled, and shows brown pigmentation. There are also cases in which the skin gives no evidences of marasmus. Senile pruritus is incurable and continues until the end of life.

Pruritus also occurs in middle-aged adults of both sexes. In men it is often associated with chronic gastric disorders, poor digestion, pressure in the region of the stomach and liver, constipation; in women, combined with genital affections, dysmenorrhœa, the menopause, rarely with pregnancy. Albuminuria, Bright's disease, diabetes, tuberculosis, cancer of the stomach and liver, are sometimes found to be the cause of the pruritus. Indeed, the latter often precedes for a long time the development of one of the neoplasms mentioned. As a matter of course, the itching which accompanies jaundice is not to be regarded as a pure pruritus, but is produced mechanically by the biliary coloring manner deposited in the skin. Finally, depressing emotions, such as loss of fortune, grief, etc., are undoubted causes of general pruritus.

The *prognosis* is absolutely unfavorable in senile pruritus alone. Under all other circumstances the pruritus may cease spontaneously if the causal conditions are improved or removed. When this is impossible the disease may last many years or be incurable.

The *diagnosis* is by no means easy. We must first determine objectively that the patient has suffered for many months from itching (the marks of scratching, recent and old, upon the skin). Then the findings must be corroborated by the clinical history. All other chronic diseases which are associated with itching, such as prurigo, scabies, itching due to bedbugs and lice, must then be excluded. In the latter affection the large excoriations are found chiefly on the neck and in the sacral region. Chronic urticaria and pemphigus pruriginosus are excluded with the greatest difficulty. But as a matter of fact these processes are nosologically co-ordinate with pruritus—at least they often occur under the same etiological conditions.

Pruritus localis is divided into various forms, according to the locality involved.

Pruritus pudendorum muliebrium occurs chiefly as pruritus vulvæ et vaginæ. The patients scratch and rub the affected parts most violently. After a time the objective signs are found to be redness, catarrhal secretion of the vaginal mucous membrane, eczematous thickening of the mucous membrane and labia majora and minora, hypertrophy of the clitoris, with moderate excoriations and crusts. The patients sometimes suffer from all possible symptoms of hysteria and may even be nymphomaniacal, although an attack of itching is not checked by the orgasm or even by coitus. The *etiology* of this variety is the same as that of general pruritus. It sometimes occurs for years as a prodrome of uterine cancer.

Pruritus pudendorum marium affects mainly the scrotum, perineum, meatus, and urethral mucous membrane. On account of the violent scratching, it soon gives rise to scrotal eczema, which makes the diagnosis extremely difficult.

Pruritus analis affects the anus and its vicinity and also the first portion of the rectal mucous membrane. The frequent scratching causes symptoms of eczema, copious secretion of mucus from the rectum, swelling and inflammation of the mucous membrane. This condition is often associated with hæmorrhoids.

Pruritus palmæ manus et plantæ pedis, with or without hyperidrosis of these parts, is rarer, but is also very distressing.

Pruritus linguæ has been described by some writers, but has never come under my observation.

Duhring applies the term *pruritus hiemalis* to an itching which occurs during the winter months, even in young people, and is confined particularly to the limbs. In my opinion this is not a true

neurosis, but an itching due to dryness of the skin as the result of the cold atmosphere. In addition, cutis anserina is frequent in cold weather and is often associated with itching, especially on dressing and undressing.

The *treatment* of general and local pruritus promises most success when the cause of the disease can be removed. When associated with liver disease and chronic gastric disorders, recovery often follows a water cure at Carlsbad and Marienbad, the internal administration of soda, magnesia, and rhubarb, and suitable diet. When due to disorders of the female sexual organs the latter must be treated.

Otherwise we must resort to those measures which will mitigate the itching itself. It is a remarkable fact that, in general as well as in local pruritus, tar has very little effect, though it is an admirable remedy against the itching of prurigo and eczema. Temporary effects are obtained from those substances which produce the sensation of cold on the skin, such as ethereal and alcoholic fluids, with or without the addition of carbolic acid, salicylic acid (one per cent), sulphuric ether. These are applied to the skin as often as the itching is repeated. Favorable effects are obtained rarely from warm baths, more often from cold douches, wet packs, baths of sulphur, soda, alum, corrosive sublimate.

In pruritus of the vulva and vagina we may use medicated sitz baths combined with vaginal injections of lukewarm or cold water, also solutions of alum, zinc, or tannin, tampons dipped in these fluids or in opiate ointments, suppositories of cacao with laudanum, belladonna, morphine, cocaine, creosote.

Subcutaneous injections of morphine and chloral hydrate (the latter also internally), and inhalations of chloroform, are employed occasionally to mitigate the itching and secure sleep.

Internal remedies from which an alterative action on the nerve centres may be hoped include Fowler's solution, atropine (atropin. sulph. 0.02, gumm. tragacanth. 1.50, glycerin., pulv. liquir. āā q s ut f. pil. xx. Sig.: two pills daily), pilocarpine muriate (0.01 subcutaneously), quinine, cocaine. In our hands these remedies have had very slight or transitory effects. This is also true of the internal administration of carbolic acid (acid. carbol. 5.0, pulv. et extr. rad. gent. āā q. s. ut f pil. lx. Sig.: Take ten pills daily) or tinct. gelsemii (ten to fifteen drops every half-hour). It is claimed that nerve-stretching proved successful in one case (Langenbuch).

CLASS XIII.

PARASITIC DERMATOSES.

LECTURE LI.

VEGETABLE AND ANIMAL PARASITES—GENERAL REMARKS ON FUNGI AND THEIR BOTANICAL POSITION, ACTION ON THE SKIN. CLASSIFICATION OF DERMATOMYCOSES—SPECIAL: FAVUS, PATHOLOGY, TREATMENT.

PARASITIC diseases of the skin form the last class of Hebra's system, and constitute a natural morbid group, inasmuch as they are due to the influence of parasitic organisms upon the skin.

The pathological processes observed in these diseases consist mainly of hyperæmia, exudation, inflammation, desquamation, etc. Their localization, form, and course depend upon the vital and vegetative conditions of the causal parasitic organisms. For this reason it is necessary to study the properties of these germs when located outside of the body. They are divided into two classes, vegetable and animal parasites.

The *vegetable parasites* of the human skin belong to the class of fungi. They are distinguished from algæ by the absence of chlorophyll. They are unable to assimilate inorganic material and can only absorb prepared organic substances.

One group—saprophytes, putrefaction fungi—are found mainly upon dead organic substances which are undergoing putrefaction. A second group—parasitæ—vegetate upon living organisms, animals or plants.

Morphologically, the fungi consist of cell threads, mycelia free from chlorophyll. These are single or branched, in places divided by septa in the interior, often intimately interwoven with one another. They form the principal part of the so-called vegetative portion of the fungus (thallus or mycelium). In addition to the vegetative portion the fungi exhibit a fructifying part, which varies in shape and is the chief point of distinction between the different varieties.

One group of fungi are known as mould fungi (hyphomycetes). The common mould fungus, penicillium crustaceum Fries (Fig. 61),

may be taken as an illustration of the rest. From the horizontal mycelium rises vertically the fructified hyphæ (*a*), which ramify into basidia (*b*) and sterigma (*c*); from these depend the spores, roundish cells arranged like a row of pearls The entire ascending structure may be regarded as the organ of fructification, and the individual spore as the product, because when it has dropped off it may grow into mycelia and reproduce the fungus together with the organ of fructification. According to the shape of the spore-bearing organ, we distinguish the fungi as penicillium, with a brush-like position of the spores; mucor, with the spores enclosed in a capsule; aspergillus, with the spores collected in spherical groups, etc.

The fungi are also propagated by means of gonidia—*i.e.*, cells which sprout from the mycelia threads in the vegetative part, and are then detached. The latter method is the usual one and the same in all mould fungi. Favorable vegetative conditions alone result in the formation of the fructification organ proper, by means of which the species is differentiated, and the spores, by which the variety is maintained.

The fungi of the dermatomycoses in man contain only mycelial threads and gonidia, never fructification organs of the kind described, so that it has not been possible to determine their systematic position.

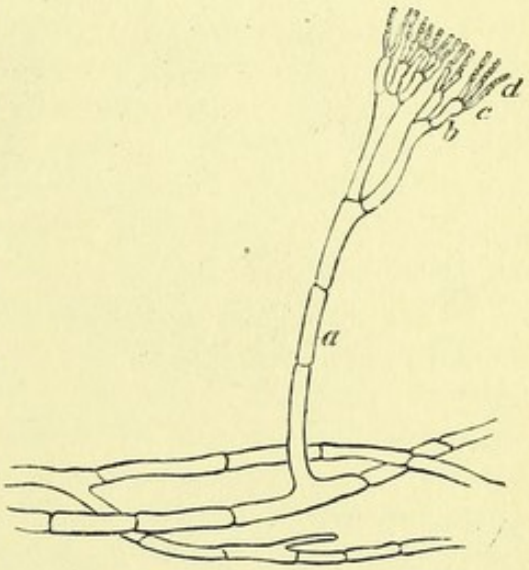

FIG. 61.—PENICILLIUM CRUSTACEUM FRIES.
a, hypha ; *b*, basidia ; *c*, sterigma; *d*, fructifying gonidia.

In the beginning little attention was paid to this fact. In 1839, a fungus was discovered by Schönlein in favus ; in the next few years by Malmsten in herpes tonsurans, by Eichstedt in pityriasis versicolor. It was taken for granted that the fungus found in a special disease must belong to a special variety in nature, and a specific name was therefore employed—for example, achorion Schönleinii, the fungus of favus ; trichophyton tonsurans, the fungus of herpes tonsurans ; microsporon furfur, the fungus of pityriasis versicolor, etc.

The condition of affairs changed when Lowe (1850) declared that the fungus of herpes tonsurans was a spore-producing form of the favus fungus, and that both were derived from the common mould fungus, aspergillus. In 1854 Hebra reported that, under the application of mouldy compresses to the skin of man, circles resembling herpes tonsurans, and, in the midst of them, favus scutula, develop,

and that genuine favus also occurs in combination with herpes tonsurans. It seemed probable, therefore, that the fungus of both diseases was derived from a common mould fungus, and, according to the special conditions of vegetation, gives rise either to favus or to herpes tonsurans, or to both combined. This view received strong support from the "pleomorphia" of the fungi, discovered by Tulasne (1851), and confirmed by prominent botanists. This term was applied to the newly discovered fact that some forms of fungi not alone develop several kinds of fructification organs, but that these appear in a regular succession in such a way that one forms a necessary stepping-stone to the development of the other. In this way many fungi which had hitherto been regarded as special species on account

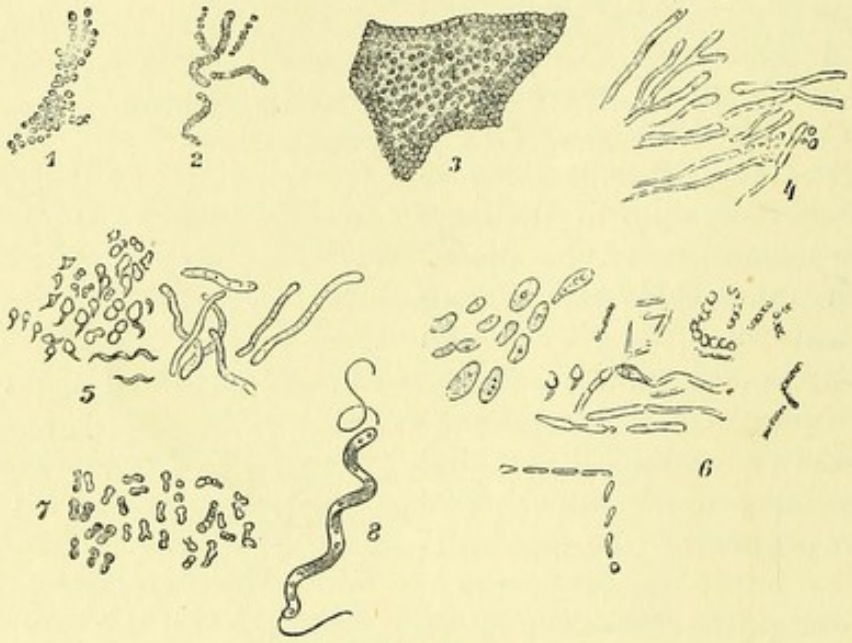

FIG. 62.

1, micrococcus; 2, mycothrix; 3, zoöglea; 4, leptothrix; 5, vibrio; 6, bacterium (coccobacteria, Billroth); 7, bacteridia; 8, spirillum.

of their fructification organs, proved to be mere variations of form belonging to the same fungus.

It was very plausible, therefore, to assume analogous developmental relations between the dermatophytes and the mould fungi vegetating free in nature. This has not been proven. Despite a few apparently successful results, the sowing of the fungi upon the skin has not produced favus or pityriasis versicolor, at the most circles resembling herpes tonsurans or microscopic scutula. Neither could the fungi vegetating on the skin be cultured into fructification in a distinct, constant form. The fructifying fungi (penicillium, aspergillus, etc.) formerly found in such cultures are regarded by the best botanists as the products of contamination of the cultures by foreign fungus germs. Even the brush-shaped spore constrictions

are not sufficient to stamp the fungus as penicillium, since many fungi occasionally constrict gonidia in the brush shape.

During the sixth decade the views concerning the botanical relations of the skin fungi became much more involved, because the classification was apparently made simpler. Hallier then included the lower fungi (yeast fungi and schizomycetes), which had begun to be regarded as the causes of many infectious processes (glanders, diphtheria, cholera, etc.), in the domain of the pleomorphia of the fungi (Fig. 62). Hallier taught that every fungus appears in three forms. Vegetating in the air (aërophyte), it produces fructification organs and forms the well-known mould fungi (pénicillium, aspergillus, etc.). Half-immersed in a nutrient fluid (hemianaërophyte), it forms tree-like cell sprouts—oidium forms, linked mould fungi. Entirely immersed and excluded from the air (anaërophyte), the individual gonidia burst, granules (micrococci) emerge from them. By simple sprouting in a fluid capable of fermentation they form true yeast—in the chain form, leptothrix ; when arranged in a mucoid mass, zoöglea ; and in the rod shape, bacteria. Hallier also maintained that he could develop each form of fungus in this series by means of cultivation. It is sufficient to state that these views are not accepted by scientific botanists or pathologists.

But even the present perfected methods of culture have furnished no result in determining the botanical position of the fungi in question. We have been enabled, however, by means of cultures on agar-agar and bouillon, to recognize a vegetative difference between achorion and trichophyton. The independence of the fungi belonging to the different dermatomycoses thus receives a positive support.

The anatomical site of the dermatophytes is the epidermal tissue (epidermis, hair, and nails) between whose elements they proliferate. They rarely appear to enter an epidermis cell.

The action of the fungi is local and mechanical. They separate the epidermis cells, and these, being detached from the underlying strata, disintegrate and serve for the nutrition of the fungus. The fungi can only flourish in the presence of air, and they absorb nitrogen from the tissues. The question whether they assimilate the elements of the tissues which are undergoing disintegration, or whether, like a ferment, they directly decompose the latter, is still more in dispute than the question of the direct or indirect relation of yeast vegetation to alcoholic fermentation. Their further action consists of the production of hyperæmia (redness), exudation (vesicles, desquamation), and suppuration (pustules), more rarely inflammation and abscess formation. All these effects are to be regarded as mechanical, perhaps partly chemical. The dermatophytes never exer-

cise an injurious influence on the constitution and the functions of the body.

The *extent* and *course* of the dermatomycoses are coincident with the vegetation of the fungus. Most forms run a chronic course. The *prognosis* is favorable.

The general *causes* for the development of dermatomycoses are those external conditions which are favorable to the growth of the mould fungi. The contagious character of these diseases has been proven clinically and experimentally. There appears to be an individual predisposition of the skin, in some cases, to the reception of the dermatophytes.

The *diagnosis* is based on the recognition of the clinical symptoms; in certain stages the microscopical demonstration of the fungus may be necessary. In the hairs and epidermis the fungus only becomes visible under the microscope after the parts are teased or dissolved in a solution of potash (1 : 30) or ammonia.

Disregarding those cases in which the etiological relation between the fungus and dermatosis is not proven, we recognize the following

DERMATOMYCOSES.

1. Favus, with the fungus achorion Schönleinii. 2. Herpes tonsurans, with the fungus trichophyton tonsurans Malmsten, which includes the special forms onychomycosis, sycosis parasitaria, and eczema marginatum. 3. Pityriasis versicolor, with the fungus microsporon furfur Eichstedt. 4. Erythrasma, with the fungus microsporon minutissimum Bärensprung.

FAVUS

(tinea favosa, porrigo lupinosa s. favosa) has always been regarded as an infectious disease which is localized mainly on the scalp. more rarely on non-hairy parts of the body and in the substance of the nails. On the parts first mentioned it is characterized by the formation of sulphur-yellow, umbilicated discs (the so-called favus scutula), perforated by a hair and varying from the size of a lentil to that of a penny.

In favus of the scalp the scutulum begins as a yellow point beneath the epidermis and around a hair. In a few weeks it grows to the size of a lentil and then presents the appearances described above. If the epidermis covering is perforated at the periphery of the disc by a blunt instrument, the favus body can be tipped up as a whole, and, when detached round about, can be removed along the hair which perforates it. It is a hemispherical, sulphur-yellow body, whose upper surface, umbilicated in the middle, is intimately intertwined with the outer epidermis. Its lower, hemispherical sur-

face is smooth, moist, devoid of epidermis, and crumbles readily beneath the finger. At the site of the favus body is a depression, with a red, moist base, which is effaced at the end of a few minutes by swelling of the epidermis layers which are freed from pressure.

The scutular or urceolar form is the primary form of favus. In

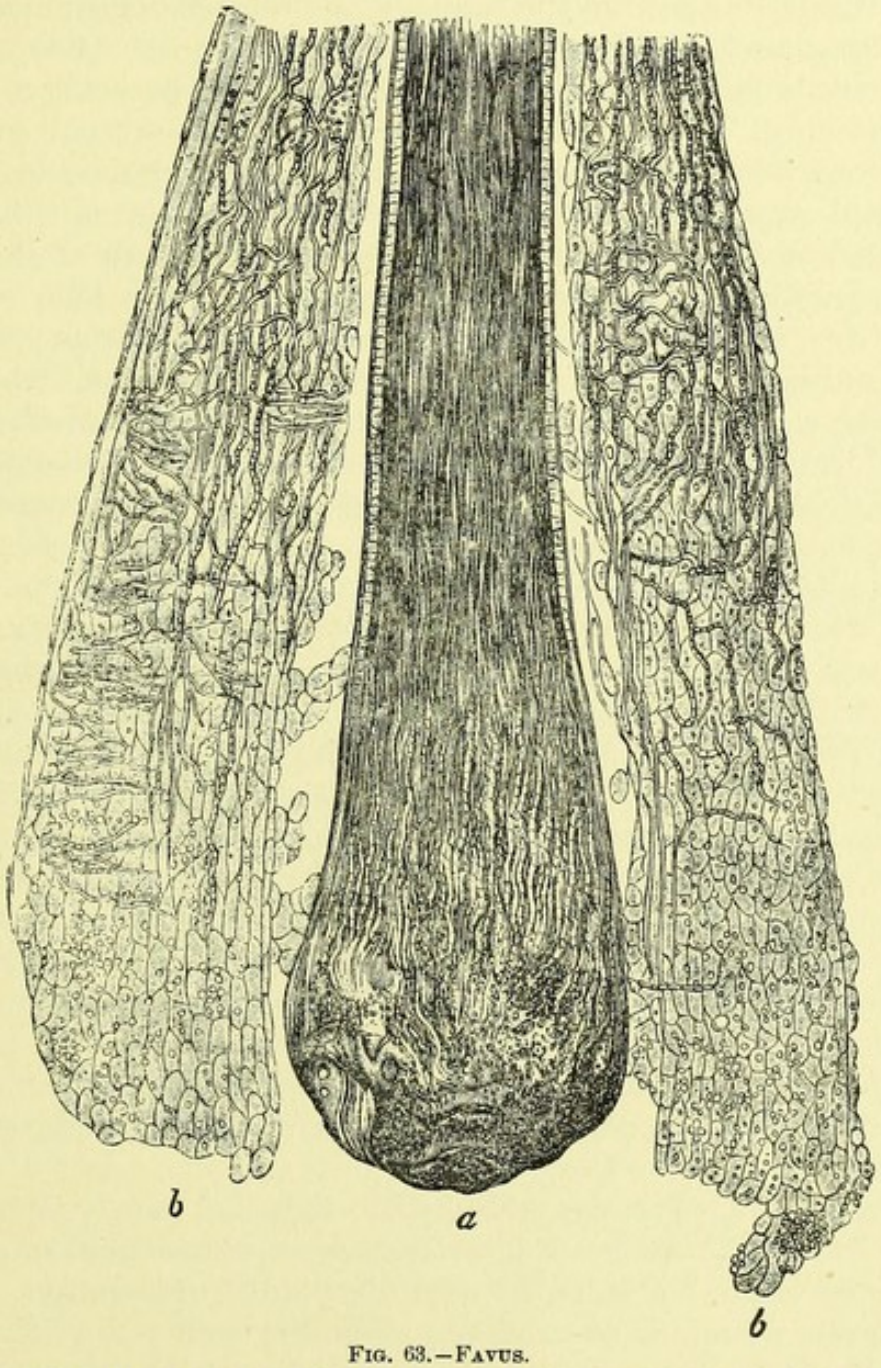

FIG. 63.—FAVUS.

a, hair bulb and shaft ; *b*, root sheath, penetrated throughout by mycelia and spores.

each opening of a hair follicle there is a preformed funnel-shaped space, inasmuch as the uppermost layers of epidermis are applied horizontally to the emerging hair, while the deeper layers are inclined toward the bottom of the follicle. In this space exudations are most apt to collect, and it is here that the fungi, which have

been implanted accidentally or by inoculation or have grown from the depth of the follicle, proliferate into compact bodies. As the upper layer of epidermis is firmly adherent to the cuticula of the hair, it remains flat in this locality or is umbilicated. In the direction of the soft, compressible rete cells the proliferating fungi may spread themselves so that the favus body becomes hemispherical.

Encapsulated in this way between the layers of epidermis, the favus scutula may remain for a long time without being detached by scratching, combing, etc. Several adjacent scutula may approach one another in their continued growth without losing the individual type. After a long time the epidermis coverings are perforated or detached in places, partly by the growth of the favus masses, partly spontaneously. The favus masses are then exposed, become dry, lose the sulphur-yellow color, and appear as yellowish-white, quite hard, dry, irregularly nodular deposits, which are sometimes a centimetre in thickness (favus suberinus, turriformis).

Within the distribution of the favus masses the hairs are devoid of gloss, look as if covered with dust, and are easily removed. The favus spot smells like mould.

At a later period secondary changes develop, because the fungus proliferates between the cells of the root sheaths of the hairs as far as the base of the follicle, then into the hair bulb and hair shaft, and also enter the shaft at the side from the root sheaths (Fig. 63). At first the proliferating fungi cause loosening, later falling out of the hair, and finally atrophy of the papilla, associated with obliteration of the latter and permanent loss of the hair. In addition, the scutula embedded in the epidermis exercise, for months and years, pressure upon the underlying papillæ and thus cause them to undergo atrophy. The skin thus acquires a cicatricial, atrophic, shining, bald appearance, devoid of follicles and hairs. Some writers have even described thinning of the cranial bones as the result of pressure by the fungous masses. There are no true favus ulcers, but complicating inflammations, eczema, enlargement of the glands may undoubtedly develop.

Favus occurs upon the scalp in the shape of one or more islets (favus discretus) ; at times it extends over almost the entire scalp (favus confertus), but is hardly ever distributed uniformly.

It runs a chronic course and may even last twenty to thirty years. Favus of the scalp may also recover spontaneously. It reaches its end on the cicatricial parts of the skin in which the follicles are obliterated, because the fungus cannot adhere without the follicle nest.

Upon non-hairy parts, the trunk, limbs, or face, favus is found more rarely, occasionally in an acute state. I have seen in one case a general favus develop within a few weeks. In this man intense dermatitis developed later in different parts of the skin, and the favus

exfoliated in these parts. Vomiting and intractable diarrhœa were also present, so that I suspected the presence of favus in the intestinal tract. The patient died of exhaustion, and the autopsy showed favus foci on the mucous membrane of the œsophagus and stomach. The intestinal mucous membrane exhibited numerous follicular ulcers and cicatrices, whose shape did not resemble any due to known causes. Hence these were probably also due to favus. Since that time other writers have also seen favus upon the mucous membrane of the stomach. Upon the non-hairy parts of the body favus usually forms discrete scutula or moderate sulphur-yellow deposits which are arranged in heaps, sometimes in a circular form. It usually recovers spontaneously after a few weeks or months. The scutula fall out because the insertion of the downy hairs is shallow, so that the fungus does not reach any considerable depth. In exceptional cases favus of the trunk is said to have lasted twenty years. It very often leaves atrophic depressions

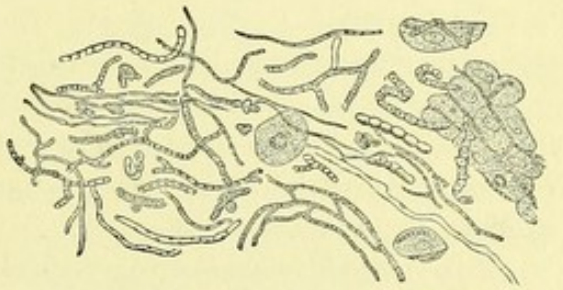

FIG. 64.—FUNGUS GROWTH FROM LOWER PORTION OF FAVUS BODY.

Threads and spores of various sizes and shapes on the right, epidermis cells.

In all localities favus is combined occasionally with red, scaly circles resembling herpes tonsurans. The scutula are either inserted in the rim of the herpes circles or situated in their centre or mingled with them. This circumstance, which has been observed after application of warm compresses to the skin and after artificial inoculation, led Hebra to believe that favus and herpes tonsurans are essentially identical and are due to the well-known mould fungi. Köbner has called these circles the "herpetic prodrome" of favus. Their epidermis scales also contain fungi.

Favus of the nail (onychomycosis favosa) appears in the shape of circumscribed, sulphur-yellow or yellowish-white deposits in the substance of the nail. It also occurs as uniform thickening, cheesy degeneration, and fluffiness of the nails. The affection involves one or more nails of the fingers and often outlasts the favus of the scalp. It is a rare disease.

Microscopical examination of the scutulum shows that it is bounded above by a layer of horny epidermis cells. This is followed by a narrow zone of a finely granular, sticky mass, probably epidermis detritus, which continues between the beginning mycelial threads far into the body of the lesion. The mycelial threads lie concentrically parallel toward the centre of the scutulum, in whose vicinity gonidia are constricted off, so that the centre of the favus consists only of spores and granules.

The individual elements of the favus body can be studied under the microscope by mixing a particle with water and placing it on the slide (Fig 64). They are very manifold, indicating luxuriant vegetation. We find the finest, simple or coarse mycelial threads, showing numerous septa, branches, and ramifications, with nuclei and spores situated on the walls and in other parts. They have the most varied sizes and shapes—round, angular, oval, biscuit-shaped, nucleated and non-nucleated. All these elements form part of the fungus known as achorion Schönleinii. The puccinia discovered later by Arndsten is an accidental admixture.

The same elements, but chiefly mycelia, are found between the epidermis cells of the root sheaths of the hair, the hair bulb and its cortical substance (Fig 63). They do not seem to extend very high in the hair.

The natural history of the fungus has not been learned by cultures, nor has the indirect method—viz., that of placing mould fungi upon the skin—enabled us to decide the question. But the direct demonstration of the transmissibility of the favus fungus has been repeatedly furnished by inoculations upon human beings, from them to animals, and *vice versa*.

Many cultures according to Koch's methods have been repeatedly made in recent years, but the results have varied extremely. Quincke has found three forms, which he calls α, β and γ fungi. The two latter are said to produce favus of the scalp, the first to produce the favus herpeticus of the non-hairy parts. Neebe and Unna (1893) describe nine different forms of the fungus, corresponding to an equal number of varieties of the disease. A satisfactory impression is created by the investigations of Pick and Král. By the inoculation of pure cultures and control examinations of the favus produced thereby, they demonstrated the pure favus fungus and proved that it was identical with the first form of the cultures previously described by Král. They also showed that this fungus alone is the cause of favus scutularis and favus herpeticus and squamosus of the non hairy parts of the body. Unbiassed clinical observers have never doubted the unity of favus as a morbid process.

There appears to be no doubt, accordingly, that the fungus is the *cause* of favus and that other conditions merely play the part of occasional exciting causes. Youth predisposes to the disease, and in persons between the ages of twenty and thirty years the disease has always lasted since childhood. In Austria the disease is quite rare, but is common among the Poles and French. Near Hérault there were said (in 1864) to have been twenty cases of favus among one thousand inhabitants, while among us there are hardly two in a thousand cases of skin disease.

The disease is produced most frequently by direct contagion from

one individual to another. This necessitates the deposit of the fungus upon a layer of macerated epidermis or perhaps in a hair follicle. Animals may infect man, because the disease has been observed in the mouse, rabbit, dog, hen, and cat. It is an astonishing fact that, as a general thing, favus is not very infectious, although the fungi are exposed in enormous masses upon the patient. Thus, the favus may for years be confined in a patient to one small spot, or the patient may live for a considerable time in his family without conveying the disease to others. This may be due to the special vegetative conditions of the fungus.

The *diagnosis* is easy as soon as the characteristic scutula or sulphur-yellow favus masses are present. It is corroborated by the dull look of the hair or by the extensive cicatricial and bald spots.

The diagnosis may be difficult, however, when, as in older favus, the masses have a mortar-like and chalky appearance and are mingled with scales and honey-like crusts. The differential diagnosis must then be made from eczema, seborrhœa, psoriasis, and lupus erythematosus. In doubtful cases it depends upon microscopical examination and the demonstration of the fungi in the deposits. This assures the diagnosis of favus, but if the fungus is only found in the hairs and root sheaths the disease might be a herpes tonsurans. Such a mistake would possess no practical importance

The *prognosis* is favorable. At the most, local changes in the tissues are produced, but no bad effects on the organism In later years the disease may recover spontaneously.

The *treatment* of favus of the scalp is attended with great difficulty. As a matter of course, the favus masses can be easily removed, but they soon return because the fungus sprouts out of the follicles. It was long known that the favus no longer flourished upon parts from which the hairs had been lost spontaneously. This suggested the idea of removing the hairs artificially, and a leather cap, lined with tar, was employed for that purpose. The treatment has now been rendered simpler and more rational.

The favus scutula and masses are first removed by softening with oil, cod-liver oil (with or without the addition of Peruvian balsam, glycerin, carbolic acid, naphthol, etc.), and then lifting them mechanically with the finger or spatula. The rest is washed off completely with soft soap. This procedure is completed in twelve to twenty-four hours.

Next the hairs which are infiltrated with the fungi must be removed, and the fungous elements in the root sheaths of the hairs within the follicles must also be destroyed. This is not an easy matter to accomplish and requires great care and time. It has been proposed that an inflammation of the scalp be produced by the application of croton oil, turpentine, etc., so that the exudation and

suppuration in the follicles would cause exfoliation of the root sheaths and hairs. But this plan would affect diseased and healthy follicles alike. Moreover, not all the diseased hairs would be exfoliated, so that subsequent treatment would again be required.

Hence epilation is the only rational method. This should be done daily, the hairs being pulled between the thumb and a blunt tongue spatula which is held in the hand. This slight traction will remove the diseased, loosened hairs, while the healthy ones will remain *in situ*. The procedure is not painful. We may also epilate with forceps, confining our operations to the favus focus and its vicinity. In addition, we use daily washings with spirit. saponat. kalinus, and, after drying the scalp, apply alcoholic or ethereal mixtures or balsamic oils or tar in order to destroy the fungi. Such substances will also enter the follicles, which are gaping wherever the hairs have been removed. We use: tinct. rusci, acid. carbolic. salicylic., creosote, benzine (1 part to 150 alcohol), naphthol oil (one per cent), petroleum, Peruvian balsam, chloroform, ether, corrosive sublimate (0.5 to alcohol 100.0), oleum caryophyll.; or ointments. oil, ichthyol, lanolin, mixed with white precipitate, tar, carbolic acid, naphthol, salicylic acid, or sulphur-alcohol-tar pastes.

These three methods—washing with soap, epilation, and the application of a parasiticide—are continued every day, the parasiticide being changed occasionally.

The hairs grow very rapidly in the epilated follicles, because the papillæ are destroyed only upon cicatricial places. If we notice, at the end of six weeks' to three months' treatment, that all the hairs are firmly adherent, then the scalp is left entirely to itself (not even washed) in order to convince ourselves that the favus is really cured. If the fungi have been left in some of the follicles, new scutula will appear in two to three weeks. These may then be treated more vigorously. Under the most favorable circumstances the treatment of favus of the scalp requires several months.

Favus upon non-hairy parts may be completely removed by a single softening with oil and washing with soap.

Favus of the nails may either be excised, when it is circumscribed, or, if there is diffuse cloudiness of the nail, it may be removed gradually by the application of emplasturm hydrargyri, corrosive sublimate (1 : 100 alcohol), and by cutting the nails from the edges.

LECTURE LII.

HERPES TONSURANS—FORMS : HERPES TONSURANS CAPILLITII, VESICULOSUS, SQUAMOSUS, MACULOSUS—ONYCHOMYCOSIS—SYCOSIS PARASITARIA —ECZEMA MARGINATUM—PITYRIASIS VERSICOLOR— ERYTHRASMA.

HERPES TONSURANS.

Syn., Common ringworm, tinea trichophytina. According to its location and degree of development, it appears in different forms which have not always been recognized as belonging to the same group. When localized on the scalp Willan applied the term porrigo scutulata, Mahon (1829) the term tinea tondens. When Cazenave (1840) recognized the formation of vesicles in the process and proposed the term herpes tonsurans, it became clear that this was identical with Bateman's herpes circinatus. With the discovery of the trichophyton tonsurans in the hairs by Gruby and Malmsten (1844) the term trichomyces tonsurans appeared justifiable.

To the forms of the disease previously described Hebra (1854) added a new, macular variety, and still later additions are eczema marginatum (Hebra) and sycosis parasitaria (Bazin). All these forms are mere varieties of one and the same morbid process and due to a single fungus. Even the most recent investigations by Sabourand (1893) have not shaken my opinion in this regard.

We should particularly notice the difference in the symptoms of herpes tonsurans of the scalp and of non-hairy parts of the body. This difference is due mainly to the fact that in the former locality the trichophyton may penetrate deeply into the hair follicles.

Herpes tonsurans capillitii forms bald discs from the size of a penny to that of a dollar. They have the appearance of poorly made tonsures, as if the hairs had been cut unskilfully close to their points of exit. The hairs are simply broken off, and any long hairs which are still present will also break off short when an attempt is made to remove them. The scalp in these parts appears moderately swollen, smooth or covered with whitish or dirty-yellow scales. Occasionally the border of the disc is somewhat reddened, very rarely covered with little vesicles, more frequently with gummy crusts. One or more of these discs, of various sizes, are found upon different parts of the head. After a period varying from many months to three years,

during which time new foci develop repeatedly, the process may become extinct locally, inasmuch as firm hairs are constantly growing, and finally the growth of hair again becomes uniform and permanent. Extensive cicatricial baldness is not left over, although here and there a hair follicle is obliterated.

In other cases the process extends over the entire scalp by coalescence of the morbid foci. The scalp is then covered throughout with a dense layer of white, dry epidermal scales. The appearances are not characteristic, and resemble those of eczema squamosum, pityriasis capillitii seborrhoica, psoriasis capillitii. On careful examination, however, we will find here and there a somewhat sharply defined focus, within which the hairs are broken off short, or a red, scaly circle which extends to the forehead or back of the neck. Herpes tonsurans may last several years, either confined to a few spots or generally diffused. No subjective symptoms are produced, with the exception of moderate itching. The termination is always in recovery, although this may not occur for many years. Numerous bald points and small patches may be left behind.

Upon non-hairy parts of the body—the trunk, limbs, and face—herpes tonsurans occurs either in a distinctly vesicular form (herpes tonsurans vesiculosus) or in the shape of red, scaly patches, discs, and circles (herpes tonsurans maculosus et squamosus).

Herpes tonsurans vesiculosus (herpes circinatus of Bateman) forms circles, from the size of a penny to that of a dollar, composed of vesicles. They develop from individual centres, the original central vesicles breaking down into scales, and new vesicles appearing upon a red base at the periphery. This encloses a scaly red or pale area. The eruption is accompanied by moderate burning and itching. One or more circles are found in the face, upon the back of the hand, extending thence to the palm, upon the back of the neck, the trunk, and rarely upon the lower limbs. In rare cases an eruption of vesicular rings is found over the entire trunk and a large part of the limbs, face, and neck. The vesicles then vary from the size of a milium to that of the head of a pin. This form always begins acutely, and is sometimes accompanied by fever, considerable inflammation, swelling, and production of crusts at the site of the drying vesicles. When a few rings are present the process runs its course acutely in three to four weeks; when generally diffused, in six weeks to three months.

Herpes tonsurans maculosus et squamosus may occur in the shape of a few rings from the size of a penny to that of a dollar. They are red, but grow pale under pressure; they develop from the centre toward the periphery; their most frequent site is the border of the hair at the back of the neck, the face, head, and neck.

Herpes tonsurans maculosus may also occur as an acute general

eruption of the trunk and limbs. It appears chiefly on the back, chest, abdomen, the lateral thoracic region, neck, and inner surface of the upper and lower limbs. Red, flat papules or patches, as large as a pinshead, make their appearance and grow, in one or two days, into red, round and oval patches as large as a lentil or penny. Within a few hours the centre of the smallest papules and patches becomes scurfy, and as the redness extends peripherally the epidermis exfoliates from the centre toward the periphery, and the integument of the centre also grows pale. Within a few weeks this process results in the production of oval rings, as large as a dollar, pale and smooth in the centre, finely scaly to the outside and red at the very periphery. Then the redness of the patches fades throughout, and after exfoliation of the loosened epidermis the normal color and smoothness return. In all, a period of three to six months elapses before the return to the normal. This form of the disease is accompanied by moderate, occasionally by quite violent, itching. A large ring often persists in one or more places for a year or two, or a chronic condition results from the extension of the process to the scalp, where it always runs a very slow course. The general eruption develops not infrequently after one or a few patches have been present for many weeks upon several parts of the body.

FIG. 65.

b, hair; *a a*, root sheath in HERPES TONSURANS CAPILLITII penetrated by numerous mycelia and gonidia of the trichophyton tonsurans of Malmsten.

Tinea imbricata is the term applied by McCall Anderson to a form of herpes tonsurans which occurs in Samoa and other South Sea Islands. On account of the rapid growth of the fungus and the more intense inflammation, concentric rings of elevation of the dark-colored and thickened epidermis develop. Many foci may be present at the same time, and, as their diameter sometimes reaches several inches, they rapidly occupy large areas of the body and extremities. This form occurs particularly on non-hairy parts, but may also appear on the scalp. In the latter locality the fungus is said not to penetrate the follicles and hairs.

As an analogue of this variety I have seen a large series of concentric rings of herpes tonsurans vesiculosus extending, in a child, over the thigh and adjacent groin and abdomen.

The direct *cause* of herpes tonsurans is the trichophyton tonsurans Malmsten. In herpes tonsurans capillitii the fungus is found in numerous hairs and their root sheaths (Fig. 65). It has already been shown that no decision has been reached regarding the botanical position of the fungus and its relations to the achorion of favus. For the present it must be regarded as an independent fungus, different from that of favus. There are considerable differences in its vegetation and action when compared with those of the favus fungus. Trichophyton consists chiefly of long, sparsely branching, moderately broad, and uniform mycelia and few gonidia. This fungus evidently attacks the hairs more often than does the achorion and penetrates much higher in the shaft of the hair. It never accumulates, even after the disease has lasted for years, into scutulous heaps at the mouths of the follicles. On the other hand, unlike achorion, it causes brittleness of the hair. Finally, it seems to be much more contagious than the achorion.

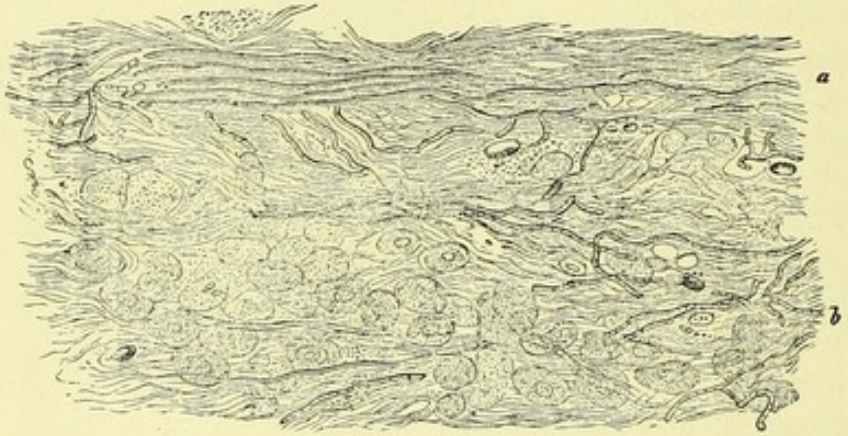

FIG. 66.—SECTION OF EPIDERMIS IN HERPES TONSURANS MACULOSUS.

a, lower horny cell layer; *b*, upper portion of rete with large, nucleated cells. In both are seen the long, thinly scattered mycelia and a few spores.

In herpes tonsurans vesiculosus, squamosus et maculosus the fungus is found between the uppermost layers of the nucleated epidermis, immediately beneath the layers of horny cells (Fig. 66). Here, as in the hairs and root sheaths, it can be made visible under the microscope by macerating the epidermis with ammonia, potash, and other solvents.

In herpes tonsurans maculosus only a few spores can be found during the first days of development, and it is not until the second or third week that the scales of the larger discs contain characteristic, usually short, but occasionally very beautifully developed mycelia. On account of this difficulty in the demonstration of the fungus, many authors are not yet convinced of its mycotic nature and prefer to call this form of the disease "pityriasis rosée" (Gibert). Whether the disease described by Vidal as pityriasis rosacée marginé, with a

fungus which he calls microsporon anomœon, is identical with our herpes tonsurans maculosus, I am unable to say.

That the fungus in question is the essential cause of the disease is proven not alone by the constancy of its presence and the results of experimental and accidental inoculation (Folly has repeatedly produced herpes tonsurans at my clinic by inoculations of pure cultures of the trichophyton), but also by the intimate relation between its vegetation and the duration of the disease. Upon the scalp, where the fungus may proliferate within the follicles and may constantly enter new ones, the herpes tonsurans may last for years. Upon non-hairy parts of the body the fungus is soon cast off by the exudation (formation of vesicles) to which it gives rise, together with the layers of epidermis which have been raised by the exudation. Hence the disease runs an acute or subacute course in such localities and also terminates spontaneously.

Whether one or the other form of the disease, larger or smaller vesicles, crusts or scales will be produced, depends upon the luxuriance of vegetation of the fungus. This is influenced, in turn, by various factors—for example, it is more intense when the fungus is derived from animals (cow, horse) or when the local inflammation and exudation are more pronounced. Transplantation of the fungus from animals to the beard or scalp of man very rapidly produces the most violent inflammation and papillary proliferation in the shape of sycosis parasitaria ("kerion Celsi"?). With the long persistence of the fungus upon one part, thickening of the epidermis follows the constant inflammatory irritation, as in eczema marginatum.

When the skin is macerated by Priessnitz's bandages, which surely become damp and contain mould fungi when used daily, herpes tonsurans maculosus is first produced, and it is only gradually that eczema marginatum, which is so distressing and is cured with such difficulty, is developed.

I present these statements in opposition to the results obtained by Sabourand (1893) in his trichophyton cultures. He described two species of trichophyton, megalospores and microspores, the latter causing herpes tonsurans of the scalp, the former trichophytosis of the non-hairy parts. He also states that these two species are subdivided into nineteen varieties, which are very similar but not identical with one another. I would merely call attention to the illustration (Hebra–Kaposi, Part II., page 639) in which I have shown two hairs taken from the same place on a boy's head; one hair contained thick mycelia and large spores, the other narrow threads and the very smallest spores.

Hence we must admit variations of the fungus in regard to luxuriance of growth and the irritant action on the skin, dependent on

the nutrient medium (human or animal skin), but this does not justify us in regarding them as true varieties. The majority of recent investigators entertain the same opinion.

The exciting causes of herpes tonsurans are the general factors which are favorable to the vegetation of mould fungi. Hence the process is more frequent in the wet seasons, in individuals living in damp apartments, or those who, while taking a cold-water cure, often place poorly dried clothing in contact with the skin. Contagion is next in frequency as an exciting cause. Of all the dermatomycoses, trichophytosis is the most easily conveyed by infection. This may take place from individual to individual. Hence several people are usually affected in the same family, or small endemics occur in crowded dwellings, schools, barracks. The infection may also take place from animals (horse, cow, cat, dog, rabbit).

Although there is a much more general predisposition to herpes tonsurans than to favus, it is much more common in young people than in adults, and upon the scalp it occurs almost exclusively in the former. Herpes tonsurans maculosus universalis is remarkably frequent in Vienna (three to five per cent of all skin diseases), while disease of the scalp is extremely rare (barely 0.1 per cent).

The *diagnosis* of herpes tonsurans capillitii can hardly go astray if the characteristic bald discs are present or there are scaly circles at the border of the hair. The bald spots of alopecia areata are distinguished by the extreme smoothness of the scalp and the absence of stumps of hair ; in lupus erythematosus there is cicatricial depression of the centre of the spots. In general diffusion over the scalp we must exclude eczema squamosum, seborrhœa, and psoriasis. The scientific diagnosis can only be made after microscopical demonstration of the fungus.

Herpes tonsurans vesiculosus of non-hairy parts can hardly be mistaken. When localized on the back of the hand it must be differentiated from herpes circinatus. The latter is always bilateral and is combined with forms of erythema exsudativum polymorphe. Sometimes, however, the demonstration of the fungus will also be necessary in such cases. Djelaheddin-Mukhtar has recently called attention to the difficulty of distinguishing herpes tonsurans vesiculosus of the palm of the hand and sole of the foot from syphilis, eczema pustulosum serpiginosum of these regions. Red, scaly rings are often diagnosed with difficulty from isolated rings of syphilis annularis and psoriasis annularis. Upon the third or fourth day of its development the disease resembles very closely an acute general eruption of eczema papulosum caloricum, psoriasis, or even variola. The diagnosis is clear as soon as the thin central scales of the smallest patches and papules are recognized. It is astonishing to

me that this form is so often mistaken for roseola syphilitica, which is sufficiently differentiated by the absence of scaling.

In the *treatment* the conditions and indications are materially the same as in the treatment of favus, especially as regards the scalp. The plan consists in softening, removal, washing of the scaly masses with oil, soaps, douches, epilation of the diseased hairs, and the application of parasiticides. The epilation must be performed daily with ciliary forceps, as the short hairs cannot be seized in any other way. In addition to the fluids recommended for application in favus, I would especially recommend a mixture of ol. rusci 15.0, spir. sapon. kalin. 25.0, lact. sulph. 10.0, spir. lavand. 0.50, bals. peruv. 1.50, naphtholi 0.50. If the affection is at all extensive, recovery cannot be looked for in less than three to six months. A test of recovery is the disappearance of the redness of the skin and scaling, and the uniform after-growth of thick, firmly growing hair.

The vesicular variety of herpes tonsurans, which is attended with violent inflammation, heals in a short time under applications of starch. The fungus is removed with the layers of epidermis which are detached by the exudation.

Isolated rings of scaly ringworm are surely cured by the application of remedies which destroy the fungus and at the same time cause mortification of the layers of epidermis harboring the fungus. These remedies include : tar, soft soap, cauterization with potash (1 : 2), tincture of iodine, iodized glycerin (iodi pur., kali. iod. āā 5.0, glycerin. 10.0), acetic acid, sulphur in the following combination : lact. sulph. 10.0, spir. sapon. kalin., sp. lavand. āā 25.0, glycerin. 2.0 ; or Wilkinson's ointment. During the last few years the following prescriptions have been found very reliable : pulv. Goa 10.0, acid. acet. 5.0, ung. simpl. 50.0 ; chrysarobin or acid. pyrogallic. 5 : 50 of fat, or acid. pyrogallic. dissolved in alcohol or traumaticin ; naphthol 0.50, spir. sapon. kalin. 0.50, glycerin. 2.0. All these remedies must be applied four to twelve times until the edges of the herpes rings appear pale and sunken, whereupon spontaneous exfoliation of the epidermis crusts takes place.

Not all the remedies mentioned are equally useful in the treatment of herpes tonsurans maculosus universalis, because some of them, when applied to a great part of the body, would produce considerable dermatitis. In these cases I recommend two inunctions daily of sapo. viridis 100.0, naphthol. 2.0, repeated for two or three successive days. The applications are made to the entire body from the neck down. Dusting powder is applied, and a bath is not permitted until the tenth to fifteenth day, after the shrivelled epidermis is completely exfoliated. An equally reliable method is the uniform inunction of sapo viridis with the moistened hand in a series of six to twelve inunctions within six days ; powder is applied.

Herpes tonsurans is related to three other forms of disease. ONYCHOMYCOSIS TONSURANS S. TRICHOPHYTINA is a cheesy degeneration and brittleness of some or all of the nails. Macroscopically the degeneration cannot be distinguished from that occurring in psoriasis, eczema, lichen ruber, and the microscope is necessary for diagnosis (Fig. 67). Onychomycosis is occasionally combined with herpes tonsurans, but the latter may recover in a few weeks or months, while the nails are not renewed in such a short time, and the fungus may also continue to proliferate in the new nail. In this way onychomycosis may be found later as an independent disease. A number of writers have often found fungi in nails which were apparently in a condition of idiopathic degeneration, but have been unable to decide whether these fungi were identical with those of herpes tonsurans. For this reason I think it is preferable to speak

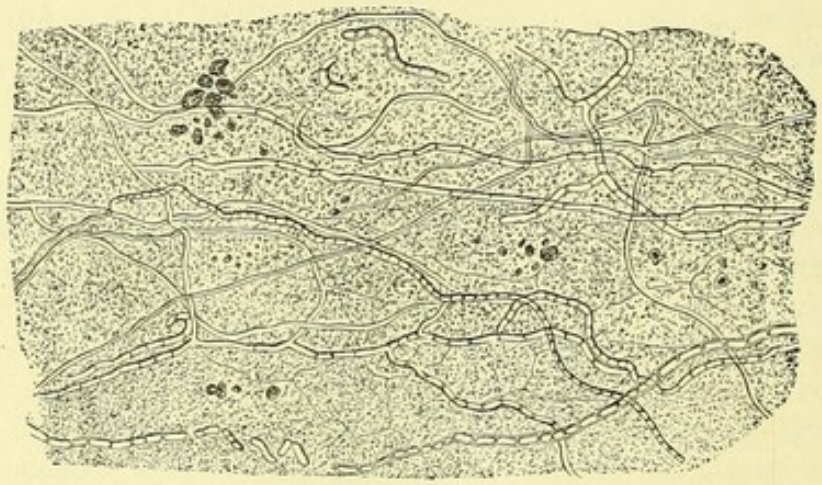

FIG. 67.—ONYCHOMYCOSIS TRICHOPHYTINA.

Rich mycelium formation between nail lamellæ, whose cell contours (after treating with caustic potash) are still recognizable in the preparation.

simply of onychomycosis, unless favus or trichophytosis is also present.

The *treatment* of onychomycosis consists in scraping or excising the degenerated parts, maceration of the nail by a rubber glove finger, and applications of creosote, acetic acid, benzine, corrosive sublimate (1 : 50 alcohol or chloroform).

SYCOSIS PARASITARIA (TRICHOPHYTINA) is an affection of the beard analogous to sycosis ; the affected hairs contain a fungus. In 1842 Gruby demonstrated a fungus in mentagra, and Anderson, Robin, Köbner, and others have confirmed the occurrence of fungi in sycosis. Köbner showed that ordinary sycosis (folliculitis barbæ) is not parasitic, but that herpes tonsurans, when localized in the beard, may produce the symptoms of sycosis (nodular trichomycosis)

by an increase of the local inflammatory processes. Thus, sycosis parasitaria is merely a variety of herpes tonsurans.

As a rule the disease appears in the form of red, scaly rings, even when localized in the beard, but occasionally an acute dermatitis develops under the influence of the rapidly vegetating fungus.

Then we find diffuse infiltration, suppuration, ecchymoses, hæmorrhagic infiltration of the skin, pustular eruptions, and abscesses. After these are opened the skin is perforated like a sieve or looks like a piece of honeycomb. The surface has occasionally the appearance of carcinoma and exhibits nodular projections, with a smooth or granular surface, which secrete a viscid fluid. These appearances have also been observed upon the scalp in combination with herpes tonsurans, and have been described as the analogue of "kerion Celsi." On account of its association with herpes tonsurans and the presence of the fungus, Fox, Auspitz, and Tanturri regard it as identical with that dermatomycosis (Fig. 68).

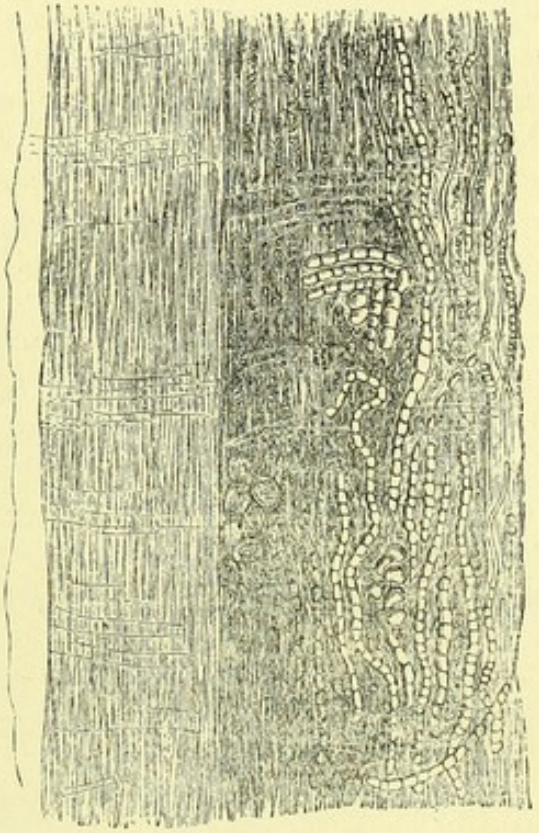

Fig. 68.

Hair from a Nodule of Sycosis Parasitaria (one-half shown) with Mycelia.

The *diagnosis* of the affection is based upon the symptoms just described. In the absence of the herpes rings, and inasmuch as the same papillary proliferations and undermining abscesses also occur in non parasitic sycosis, the assumption of a parasitic origin will be favored by the acute development of the symptoms (three to four weeks).

The exciting *causes* of sycosis parasitaria are the same as those of herpes tonsurans—namely, contagion from the cow and horse. Hence the disease is most frequent among cowherds and hostlers. In Leipzig and Berlin an epidemic of trichophytosis, especially of the beard, was observed in 1882 to 1885 as the result of infection in barber shops.

The *treatment* is the same as that of the form just described. The papillary vegetations are disintegrated and the fungi destroyed most rapidly by applications of sublimate (1 : 100), naphthol-sulphur-

alcohol soap, acid, aceticum, followed immediately by dusting with lac sulphuris, so that epilation is often unnecessary.

Finally, mention must be made of Hebra's ECZEMA MARGINATUM. This affection (page 350) is localized chiefly on the genitalia and their immediate vicinity, where it forms rings and sectors of a circle varying from the size of a quarter of a dollar to that of the palm of the hand or larger. They may extend, for example, from the scrotum across the groin to the inner and posterior surface of the thigh, thence to the sacral region, the other thigh, and around to the mons veneris. Similar circles are also found, singly or in large numbers, upon the trunk, neck, and limbs. The border of the ring appears jagged, covered with small papules, vesicles, or yellowish-brown crusts. The parts within the rings are dark brown in color, scratched, covered with crusts or with newly developing papules and circles. The disease causes very violent itching and scratching.

Köbner and Pick and, later, I have found fungi, similar to those of herpes tonsurans, in the epidermis of the circles of eczema marginatum. These two writers have also furnished other reasons for believing that eczema marginatum and herpes tonsurans are identical. Although Hebra recognized the fungous nature of the disease, he believed that the term which indicated its eczematous character should be preserved. It is a striking fact that the process is intensely itching and lasts a very long time (fifteen to twenty years or more); it resists treatment obstinately and is apt to return. Furthermore, it does not appear to be directly infectious, so that it is never observed in endemics. Finally, the hairs of the diseased region do not break off short and lose their gloss. All these features are absent in herpes tonsurans. Hence Pick regards eczema marginatum as a combination of herpes tonsurans with eczema, and in this opinion I coincide.

The exciting causes include maceration of the epidermis by sweat on the opposing surfaces of skin of the genito-crural folds, pendulous breasts, etc., in obese individuals or those who are sedentary in their habits. In these cases the skin first is affected by eczema intertrigo, later by eczema marginatum. The next most frequent cause is maceration of the epidermis by water in hydrotherapeutics, especially by Priessnitz's compresses. This gives rise usually to simple eczema, often to herpes tonsurans, and quite often to the combination of both in the shape of eczema marginatum.

The *diagnosis* of the disease is easy, inasmuch as the ring form of herpes tonsurans and the characteristics of eczema in the vesicles and scratch effects are distinctly marked. It is difficult to secure permanent recovery, because the continuance of the cause favors relapses, and it is also difficult to destroy all the fungi. The latter are situated at an unusual depth, probably on account of the thick-

ness of the layers of epidermis, and the upper layers must be scratched off in order to find them.

It is erroneous to apply the term eczema marginatum to sharply defined patches of eczema squamosum, such as are often left over in the chronic and remittent eruptions due to chemical agents. It would be preferable to call such forms eczema discoides.

Only a few of the remedies recommended in herpes tonsurans are useful in the *treatment* of this disease. Among these are chrysarobin ointment, ung. Wilkinsoni, naphthol-alcohol (one per cent), naphthol ointment (five per cent), naphthol-sulphur soap and paste, which are used in a series of six to twelve applications. We may also recommend applications of sublimate (1 : 100 alcohol), sulphur-alcohol-tar paste, tar, tincture of iodine. When recovery is tardy and the epidermis is quite thick, it is well to detach the latter by means of potash (1 : 2 of water), soft-soap compresses, or acid. aceti-um, and then to apply the above-mentioned remedies.

PITYRIASIS VERSICOLOR.

The term "chloasma" is wrongly applied to this affection by some pathologists. It is known to the laity as "liver patches." It appears as pale-yellow, yellowish-brown or dark-brown. rarely pale-red specks, and as irregularly shaped patches (which may extend over large areas of the skin), which are sometimes smooth and shining, sometimes dull or scurfy. As the result of central involution the patches sometimes form rings as large as a quarter of a dollar. They are localized chiefly on the trunk, neck, and flexor surface of the upper limbs, more rarely of the lower limbs, never upon the hands, feet, or face. In the shape of sharply defined, yellowish-brown, scaly patches they occur in the axilla and on the surfaces of contact of the scrotum and thigh and the pendulous breast. Scratching with the nails may detach the epidermis of the patches in coherent lamellæ and expose the red, bleeding base. Moderate itching accompanies the affection, which usually lasts fifteen to twenty years, at times in a few patches, at times diffused very extensively. Its development and involution occur imperceptibly, but it always disappears with advancing age.

When the lamellæ of epidermis are placed under the microscope they are found to contain the microsporon furfur (Fig. 69). This consists of uniform, unusually large spores which form heaps of thirty or more, and short. many-branching mycelia which connect the gonidia with one another and in part send out gonidia, in part grow out of them Proliferation of the fungus into the hairs, or even into the epidermis of the mouths of the follicles, has never been seen

Although Köbner and Hublé succeeded in producing experi-

mental inoculation of pityriasis versicolor, nevertheless it is hardly ever conveyed from one individual to another This seems to require an especial predisposition of the skin. In married people not a single undoubted case of transmission has been observed.

A mistake in *diagnosis* is hardly possible. Even the delicate red, discrete patches, which look like syphilitic roseola, are recognized at once, inasmuch as they can be removed with the nails.

The *treatment* is the same as that of herpes tonsurans maculosus (page 643).

Vidal has recently described as a special mycosis, under the term "pityriasis circinata et marginata," reddish or pale brown, moderately scaly patches of the trunk, limbs, and axillæ. Its fungus,

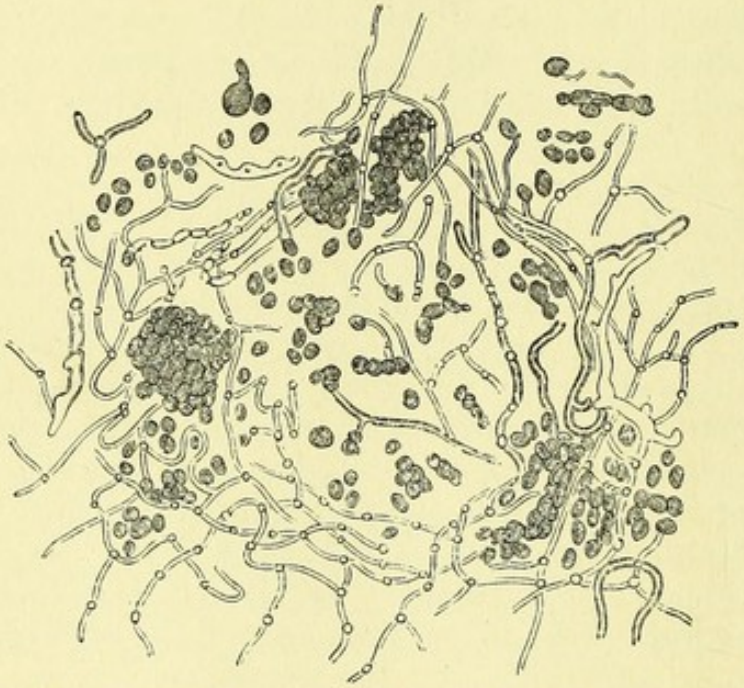

FIG. 69.—MICROSPORON FURFUR, THE FUNGUS OF PITYRIASIS VERSICOLOR
(Magnified seven hundred diameters.)
(In the figure the cells of the epidermis have been omitted.)

which consists of the finest spores without mycelia, is called microsporon anomœon s. dispar (page 641).

ERYTHRASMA.

This disease, which is quite frequent, appears in the shape of sharply defined, pale-red, yellowish- or dark-brown, smooth or slightly scaly discs upon the surfaces of contact of the scrotum and thigh, axillæ, mammary folds, more rarely upon the integument adjoining these regions. It is found chiefly in adult males. The patches itch only under the influence of the perspiration, and are extremely chronic. The epidermic scales of the patches are easily removed and contain the microsporon minutissimum of Bären-

sprung; this consists of extremely fine, long mycelia with corresponding gonidia.

The pityriasis maculata et circinata described by Duhring, and which he regards as identical with pityriasis rosée and circinée of Bazin. Biell, and Gibert, appears to me to correspond in part, perhaps, to herpes tonsurans maculosus, in part to an acute form of erythema. I am unable to arrive at a positive conclusion from the mere description of the cases.

Upon the sternum (usually in hairy males, or when the integument is seborrhœal in this region) and rarely on the back, especially in the interscapular region, we sometimes find extremely obstinate, pale- or brownish-red discs and rings, from the size of a lentil to that of a quarter of a dollar. The border is formed of very fine papules or vesicles, which itch during perspiration and are often scratched off. The enclosed area is moderately scaly or has a fatty, glistening look. This eruption has been described under various names, such as erythema, eczema, pityriasis circiné, furfuracé sebacé. Besnier has properly emphasized the fact that although fungus elements, spores, rarely short mycelia are found in the epidermis, it is difficult to determine which ones are pathological and which are mere accidental admixtures. The affections in question are relieved easily by washing with soap and then applying remedies which macerate and remove the upper layers of epidermis.

ACTINOMYCOSIS CUTIS.

The number of cases of disease of the skin due to actinomycoses is increasing in recent years with our wider knowledge of their clinical history. Actinomycosis cutis is found chiefly on the cheek and angle of the lower jaw, the abdomen and groins in the shape of diffuse, tolerably tense, indolent infiltrations of the skin and subcutaneous cellular tissue. These give rise to suppuration and abscesses with sinuous canals. In addition to tissue detritus and proliferating granulations, grayish, brittle granules, as large as a poppy seed, can be squeezed out of the canals. Under the microscope these granules are found to consist of mycelial masses of actinomyces. The disease usually runs a very slow course. Recovery may be secured by excision, by parenchymatous injection or electrolytic introduction of various antimycotics (solutions of carbolic acid, sublimate, iodine, etc.). In Billroth's case recovery followed the use of Koch's injection.

LECTURE LIII.

SKIN DISEASES DUE TO ANIMAL PARASITES.

ANIMAL PARASITES—PARASITES PROPER AND EPIZOA—THEIR MODE OF ACTION ON THE SKIN—DERMATOZOONOSES—SCABIES—NATURAL HISTORY OF THE ACARUS—CUNICULUS.

DERMATOZOONOSES.

THESE diseases have two sets of symptoms, those due to the parasite as an individual and to its vital conditions (habitat, nutrition, propagation), and pathological changes in the skin which are produced directly or indirectly by the parasites.

The animal organisms in question may be divided into two categories :

1. *True parasites* (dermatozoa), which live exclusively or for a time in the human integument. These include : 1, acarus scabiei ; 2, acarus folliculorum ; 3, pulex penetrans ; 4, filaria medinensis ; 5, leptus autumnalis ; 6, ixodes ricinus ; 7, cysticercus cellulosæ.

2. *Epizoa*, which infest the skin for a time in order to obtain nourishment, and live in its immediate vicinity (the hair, clothes). These include : 1, lice (pediculi capitis, pediculi pubis, pediculi vestimentorum) ; 2, fleas (pulex irritans) ; 3, bedbugs (cimex lectularius) ; also flies and various other insects.

These parasites may produce skin disease directly by injuring and irritating the point of attack and tearing up the epidermis. This causes symptoms of inflammation (hyperæmia, exudation, hæmorrhage, degeneration, hyperplasia of the epidermis and substance of the nails), or they act indirectly by leading to itching, burning, and scratching with its well-known secondary effects (injury of the skin, pustules, inflammation, ulcers, eczema of all grades and kinds).

All these nutritive changes belong essentially to the forms of inflammation, particularly of eczema, but, in combination with the symptoms due directly to the parasite, they form an individual clinical complex. An accurate knowledge of the biology of the parasites is necessary to the comprehension of the corresponding clinical types.

SCABIES—ITCH.

This disease has been recognized for thousands of years, and it has been known for centuries to be due to the burrowing of a little animal in the skin.

The Arabians mention a little animal, called syrones, in itch, and from the twelfth to the seventeenth century numerous statements were made concerning its existence. In the fourteenth century Guy de Chauliac stated that the animal bored a passage into the skin. Mouffet (1634) gave an accurate description of the acarus, and Hauptmann furnished good illustrations. In 1687 Bonomo and Cestoni gave accurate descriptions and drawings of the acarus and its ova; stated that the acari were of both sexes, and that they were the sole cause of itch.

Another century and a half elapsed, however, before this knowledge was generally accepted. Distinguished physicians, such as John Hunter, Lorry, and Willan, ignored the acarus entirely, denied its relations to scabies, or believed that a virus injected by the acarus into the blood gave rise to the itching, or that the acarus was developed from the tainted humors of the patients. Such views were adopted on account of the prevalence of Hahneman's doctrine of the injurious character of repressed itch. But Hebra's classical work (1844), "Ueber Diagnose, Aetiologie und Therapie der Krätze," finally placed the subject on the solid foundation of clinical and experimental facts.

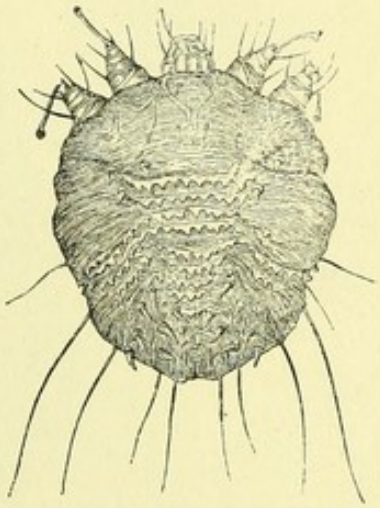

FIG. 70.—ACARUS. SEXUALLY MATURE FEMALE, 0.35 MILLIMETRE LONG, 0.50 MILLIMETRE BROAD.

Back view, magnified three hundred diameters, showing ridges, spines, and spiculæ.

THE ITCH MITE.

The acarus scabiei (Degeer), sarcoptes hominis (Raspail), is classed among the mites or acarinæ. When removed from its canal in the epidermis the female appears to the naked eye as a barely visible, yellowish-white, hemispherical little body. Placed on the finger nail it remains motionless for a time, and then moves rapidly across the nail. Seen under the microscope it is found to be a crab-like animal with a conical proboscis and eight legs. The rounded, elongated body is marked by wavy transverse furrows, which make it possible for the cuirass parts to slide over one another during motion. The back is provided with short and long spines, and rows of spiculæ arranged in annular ridges. The head is provided with six bristles, with four pairs of mandibles, and two adjacent three-jointed palpi. The legs are eight in number, the first and second pairs in both sexes possessing pedunculated sucking discs. The female

(Figs. 70 and 71) has a long bristle upon the third and fourth pairs; upon the posterior rim of the body, between the hindmost (anal) bristles, a slit leading to the vagina, and upon the abdomen an ovipositor. A digestive tube, divided into stomach and intestines, an ovary, and muscles are anatomically demonstrable. A mature ovum is often seen in the interior of the mite. The female may live twenty to sixty days.

The male is smaller (0.20 millimetre in length, 0.35 millimetre in width) than the female, has a sucking disc upon the fourth pair of

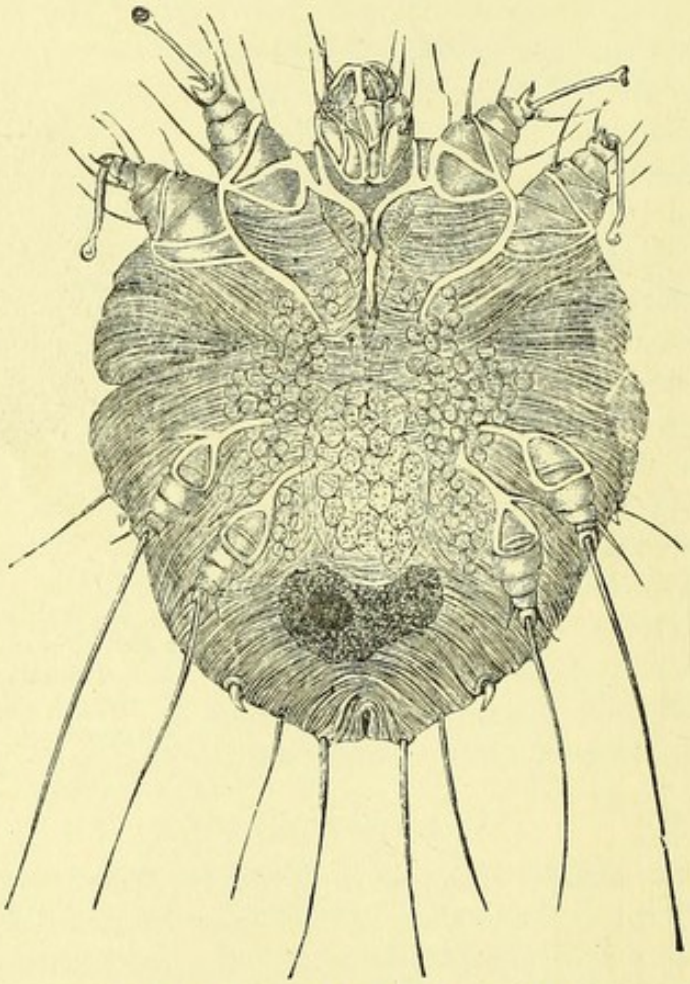

FIG. 71.—PREGNANT FEMALE ITCH MITE.
View of abdomen. In the midst of the mite a mature ovum. The hind legs provided with bristles.

legs (instead of the bristle in the female), and between the hind legs has a median, horseshoe-shaped framework of chitin (Fig. 72), in which is inserted a fork-shaped penis.

The male lives in shallow excavations of the epidermis, in small papules and vesicles, near the cuniculus which contains the female, and appears also to wander about the skin. Hebra once observed, under the microscope, copulation of the male and female. In itch of the human species the males are found in smaller numbers than the

females, and are said to die in six to eight days after impregnation of the female.

The sexually mature, impregnated female alone bores a canal in the epidermis—the mite burrow or cuniculus—in which it deposits the ova, and dies after performing this function (Fig. 73).

In experimental transmission it has been found that the mite divides the epidermis with the sharp mandibles, bores its head into the opening, and disappears beneath the epidermis. Excision of the burrow, in addition to other results of observation, enables us to infer the further course of the mite. It deposits ova behind it, one

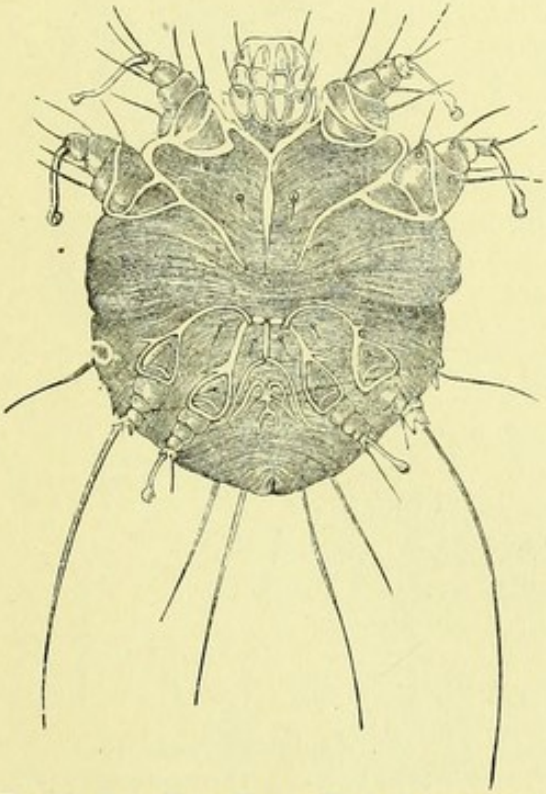

FIG. 72.—ITCH MITE, MALE. VIEW OF ABDOMEN.

FIG. 73.—CUNICULUS, SEEN UNDER LOW POWER.

At one end, the female mite with an ovum in her body. Behind her, twelve eggs and twelve eggshells. It appears that this mite lays two eggs a day. *a*, a mature larva with an anterior pair of legs; between the eggs and eggshells, dark bodies (fæces).

a day, or two at the most, in all twenty to fifty, or perhaps even a larger number.

The ova (Fig. 73) are oval, with the long axis placed at right angles to that of the burrow, about 0.16 millimetre long, 0.11 millimetre wide. Twelve to twenty or more are present in one burrow. The last two or three ova (immediately behind the mite) are filled with the segmented yolk; in the third to the fifth ova the first indications of the embryo are visible; in the sixth to the ninth, the mite larva; in the oldest ova the head and anterior pair of feet are often distinct (Fig. 73, *a*).

The mite larva (Fig. 74) is six-legged, reaches its maturity in

three to six days, breaks through the eggshell, grows until it is 0.15 millimetre long and 0.10 wide, and crawls to the mouth of the burrow. According to some writers it escapes through "air holes" in the burrow, runs about the integument for a time, and bores its way for a short time into a nest where it passes through the moulting process.

The mite moults three (according to Gudden, four) times. The larva, on its escape from the ovum, has only one pair of hind legs, two anal bristles, and ten dorsal spines. After the first moult it has

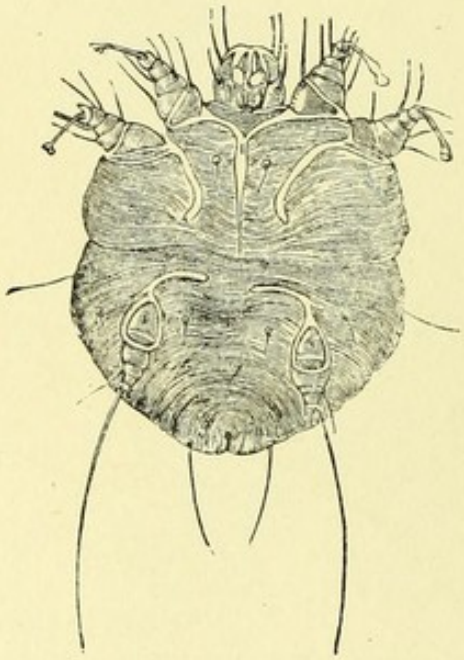

FIG. 74.—LARVA OF ITCH MITE WITH SIX LEGS. VIEW OF ABDOMEN.

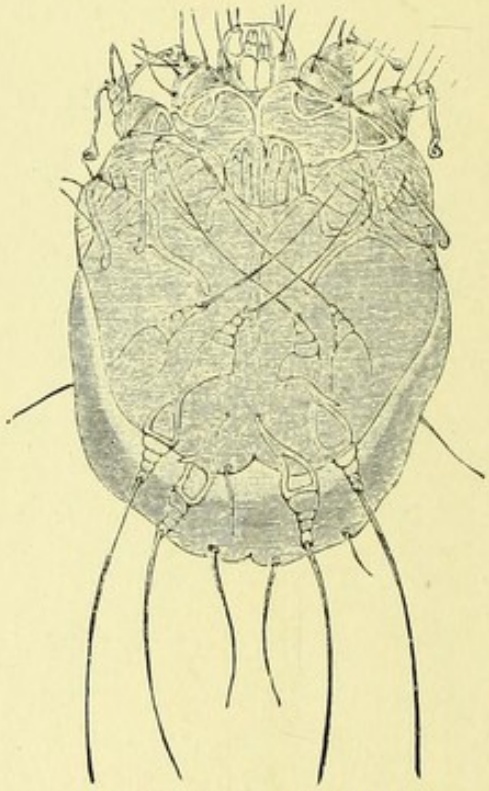

FIG. 75.—SECOND MOULTING. Inside an eight-legged mite is seen another newly formed, also eight-legged animal.

eight legs, with four anal bristles and twelve dorsal spines. In the second moult (Fig. 75) the mite gains two dorsal spines, and, after the third moult, becomes sexually mature.

Outside of the skin the mite may live two to three days, and it may also be kept alive for some time in fluids which exclude the air (water, oil, petroleum). The frequent transmission between man and animals, and the results of comparative investigations, seem to show that the itch mites found in various animals (sheep, cat, rabbit, horse, camel, elephant, etc.) belong to the same class as the acarus hominis.

LECTURE LIV.

SCABIES (*continued*).

SYMPTOMS—PATHOLOGY—ETIOLOGY—TREATMENT.

THE symptoms of itch consist, in the first place, of those changes in the skin which are produced directly by the mites. Among these the most prominent is the itch burrow or cuniculus. This develops in the following manner :

At the point of entrance of the acarus the epidermis is torn up over a round space one to two millimetres in circumference, as shown

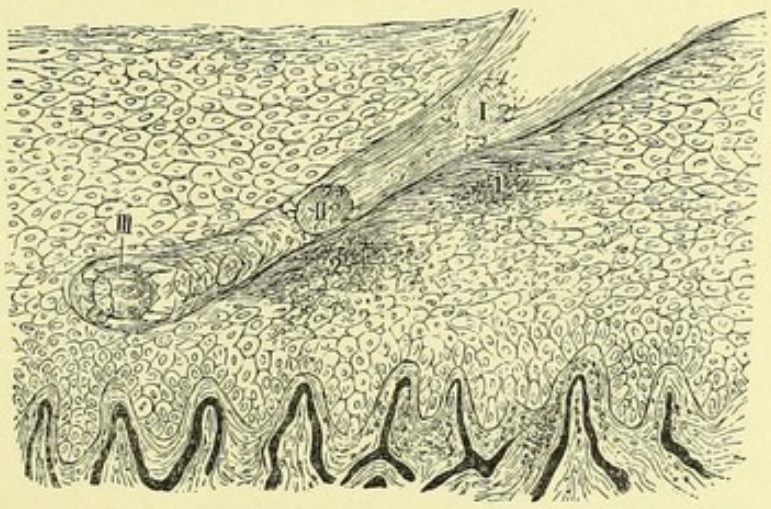

FIG. 76.—SCHEMATIC SECTION OF THE EPIDERMIS, CUNICULUS, AND PAPILLARY LAYER OF CUTIS.

in the schematic drawing (Fig. 76). Otherwise the irritation induced by the bite gives rise to exudation and to a vesicle which possess nothing characteristic of the itch. After desiccation a funnel-shaped exfoliation of the epidermis results. The mite now bores further, in an obliquely descending direction, in order to reach the layers of juicy rete cells, and has now reached, let us say, the point marked I in Fig. 76. As happens in the presence of every foreign body, eliminating hyperplasia and cornification of the epidermis develop at point I. This serves to elevate the part, and at the same time separates the mite from the nutrient layers of the rete. The mite bores deeper in order to find nourishment and room for the

ova, and reaches II. Here the eliminating cornification of the epidermis is repeated, and the mite again bores deeper until it reaches III. The cuniculus and its characteristic appearance are thus explained.

It is a slightly curved, sometimes straight canal, several millimetres, often one to two centimetres, in length. Its contour begins with the exposed, wide oval exfoliation—head end of the burrow (Fig. 76, in front of I)—then passes into a narrow parallel, whose lines separate a little at the end of the canal, and terminate in a rounded extremity (Fig. 76, III). This is marked by the acarus, a

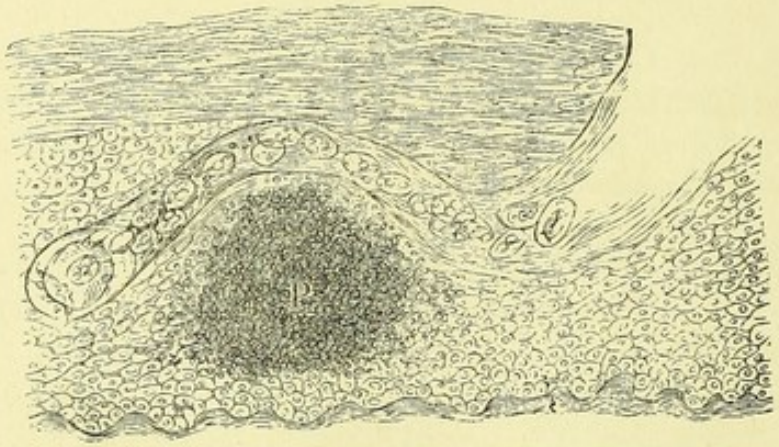

Fig. 77 *a*.—Schematic Section of a Pustule (P) in whose Epidermic Covering an Itch Burrow appears.

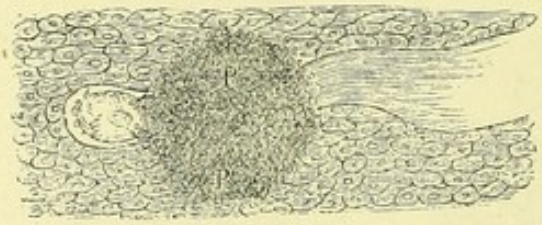

Fig. 77 *b*.—The Same from Above.

The acarus is seen just beyond the pustule, in the rete not yet affected.

yellowish-white, shining point. Above the older parts of the canal the integument is dry, depressed, fissured. The more recent the particular part of the canal the deeper is the situation of the latter, the thicker and more succulent is the overlying epidermal covering. Hence the canal here looks whiter, its contents (ova and fæces) shine through the succulent covering as yellowish and black dots, and the end is marked by a yellowish-white shining speck. The mite is easily removed by piercing the skin, just alongside the yellowish-white terminal point, with the tip of a penknife or a cataract needle, and squeezing out the contents.

The itch burrow may be removed for microscopical examination

by excising it with a pair of scissors. When pressed between two cover glasses and examined with the microscope, the burrow will furnish an instructive picture of the ova, fæces, and the mite, as shown in Fig. 73.

The appearance of the burrow is somewhat different when the presence of the mite has caused more intense irritation of the papillæ, resulting in exudation and the formation of vesicles and pustules (scabies pustulosa). This may occur at any stage of the process. As the exudation always takes place from the papillary vessels, the product will always be found beneath the horny cell layer which forms the base of the cuniculus. The epidermis layer which encloses the burrow also forms the top of the pustule, and in this top covering the punctate line of the burrow is visible (Fig. 77 *a*). The mite bores further in order to pass beyond the pustule and reach normal rete. Hence the contour of the pustule is always hollowed out slightly in the prolongation of the burrow (Fig. 77 *b*).

Cuniculi may be found in any part of the skin, but they are more frequent in certain localities. These are, in the order of frequency, the flexor surface of the carpus, the sides of the fingers and the folds between them, the palm of the hand (in children and individuals with a delicate skin), the extensor aspect of the elbow, the anterior axillary fold, the nipple and its vicinity in females, the umbilicus and its vicinity, the penis, scrotum, buttocks (especially over the trochanters), the inner border of the foot. In addition, the favorite sites of the acarus are those parts which are subject to repeated pressure and whose epidermis is thickened. Hence they are found on the waist in men and women whose skin has been made callous by corsets or suspenders ; in shoemakers, over the tubera ischii, where the skin has been thickened by the constant sitting on a hard stool ; in weavers, over the ribs, which are often pressed by the loom, etc. These localities form central foci from which the mites invade adjacent parts. In cases of moderate severity some only of these regions are affected. In more severe cases of long duration we find not alone that all the parts mentioned are attacked, but also the intervening parts upon the abdomen, arms, nates, thighs, shoulders ; in children the face and scalp, backs of the hands and feet and the soles of the feet, may also be involved.

Apart from the cuniculi an important objective sign of scabies is the eczema, which is partly a direct, partly an indirect effect of the activity of the mites. To the former category belong the papules, pustules, and vesicles which develop at the point of entrance and underneath the burrows. These lesions, however, are not characteristic of scabies unless they are associated with the burrows. Many individuals and localities exhibit a special predisposition in this respect. Thus, numerous large vesicles and pustules are found

very often in scabies of the hands and feet in children and young people, especially females.

Upon the nipple and its areola, the axillary fold, umbilicus, hips and penis, firm red nodules form beneath the burrows and follow the direction of the latter. In addition short canals, which look like fine, jagged slits made with a needle, are found on the penis.

Eczema also develops in part as the result of reflex irritation, and also of local complications, especially the scratching.

The itching of scabies is very intense, but is not constant. It is most severe at night upon undressing and after getting warm in bed, evidently because the mites are then most active. The scratching intensifies the eczema, and, as the itching affects chiefly the location of the mites, the secondary eczema is most severe in these parts. This is an important objective sign. The eczematous eruption consists, in general, of discrete papules and vesicles, which

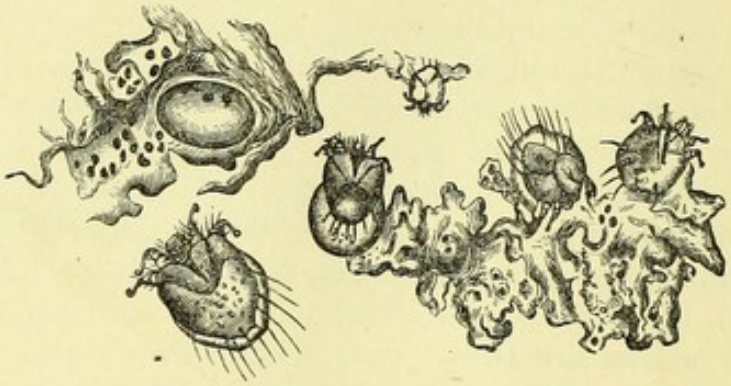

FIG. 78.—SCABIES CRUSTOSA.
Acari in various developmental stages, eggs, and fæces.

are combined with pustules, crusts, and bloody excoriations in proportion as the scratching is more energetic. More rarely we find diffuse, weeping eczema. In a typical case it occupies the integument from the nipples to the knees; also the inner surface of the wrist, the extensor surface of the elbow, the nates, the legs. It is characteristic that the eczema eruption is most dense over the principal foci of the cuniculi and in their immediate vicinity. The appearances are so characteristic that, even at a distance, they may be recognized as the result of scabies.

The eczema is most severe (eczema pustulosum, crustosum, associated with infiltration of the skin) upon the hands, female breast, buttocks of shoemakers, and all parts which are the site of pressure callosities. Upon the hands and feet of infants and young people we often find rupia-like vesicles and bullæ beneath and alongside the cuniculi. Their crusts often conceal the burrows.

Under the title "scabies norwegica" s. Boeckii (s. crustosa) Hebra described a peculiar form of scabies, first observed on lepers

by Boeck and Danielssen, later by other writers. As a result of the long duration of the disease or of individual predisposition, thick epidermic callosities develop upon the palm of the hand, sole of the foot, elbow, and knee. Within them the mites are found in irregularly excavated spaces (Fig. 78), as in scabies pecorina. This was probably due to the enormous number of the mites. A large number in all stages of development were found within the mucous layer, while the layers of horny cells, which could furnish no nourishment, contained only dead remains of the acari. Cheesy thickening of the finger nails and alopecia of the scalp have also been observed as a result of the presence of a large number of mites.

The development of scabies is attended forthwith by itching, and this increases as the disease advances. As the formation of a single cuniculus requires one to two weeks, and as several weeks elapse before the production of new ones, every moderately developed scabies must be regarded as at least six weeks to three months old. If its course is uninterrupted the disease becomes general within a few months, but is always more marked at the sites of predilection. It may even last for life, as was seen by Boeck and Hebra upon Norwegian lepers. The disease has no injurious effect upon the internal organs or functions.

During febrile diseases (pneumonia, variola, typhoid fever, etc.) the symptoms disappear and the burrows become indistinct, but they return during convalescence. This is due to the fact that the acari perish in the hot, dry skin, but the ova survive. When the skin becomes turgescent during convalescence, the ova mature and the symptoms begin afresh.

Infection with scabies occurs only when impregnated mites, or male and female together, gain access to the skin ; they may be derived from human beings or animals. Infection occurs only after prolonged contact, especially when the individuals are sleeping in the same bed. Scabies is rarely acquired from temporary contact during the day, or from handling utensils and clothing of scabies patients. In this city scabies is most frequent in those trades in which the apprentices sleep two in a bed. The shoemakers furnish forty to fifty per cent, the tailors twenty to thirty per cent of all cases. In former years one thousand to twelve hundred itch patients were treated yearly in our dermatological department. Since 1864, when the trades unions employed their own physicians, the number has diminished to three hundred to five hundred cases a year.

The *diagnosis* of scabies merely requires attention to the characteristic symptoms, yet there is hardly a dermatosis which is more frequently overlooked. We have seen many cases in which all sorts of treatment had been employed unsuccessfully for months and years because the diagnosis of scabies had not been made. Not

every physician is sufficiently skilful to remove the mites from their burrows, and it may even be difficult for an expert in complicated forms ; but even when this cannot be done the diagnosis is not difficult. In typical cases the cuniculi are easily recognizable. The examination should not be confined to the hands, where the burrows may be destroyed by the use of soap, acids, and mechanical friction ; attention should also be paid to the penis, to the nipples in women, and to the parts below the ribs which have been compressed by the bands of the underclothing. If the burrow has also been destroyed in these localities by treatment, we can still recognize its characteristic contours (Figs. 73 and 76).

Apart from the character of the eczema (consisting in great part of discrete papules, vesicles, and pustules), its predominant localization upon the parts previously mentioned constitutes a further diagnostic aid. Whenever this localization is observed I would advise that the diagnosis of scabies be made, or, at least, that the case be treated as such.

The *prognosis* is absolutely favorable, as it can be certainly cured in a short time, even after it has lasted for years and has exhibited all kinds of complications.

The first indication of *treatment* is the destruction of the acari and their ova. With the elimination of the latter the itching and scratching will cease, thus furnishing an opportunity for spontaneous involution of the existing eczema ; but, as the eczema itself gives rise to scratching, and in many cases of itch develops into the intensity of an independent disease, the second indication is the cure of the eczema.

Inasmuch as we do not consider the patients cured until the eczema has also been relieved, we do not vaunt our skill in "rapid cures." In the hospital the average duration of treatment in men is three to five days, in women five to seven days. In the former (shoemakers and other workmen) eczema of the nates, in the women eczema of the mamma, often requires three to six weeks' treatment.

The drugs which destroy the mites and their ova are : sulphur ; the infusions, decoctions, and ethereal oils of certain plants (semina staphysagriæ, helleborus, baccæ lauri, ol. caryophyllorum, menthæ, etc.) ; balsamic and empyreumatic oils, bals. peruvianus, balsam of tolu, petroleum, tar, naphthol. To these must be added those remedies, like soap and coarse powders (chalk, pumice), which remove the horny layer and facilitate the entrance of the parasiticides into the cuniculi.

The following combinations have gained more or less renown in the treatment of itch :

Helmerich's ointment (sulph. citrin. 10.0, potass. subcarb. 1.0,

axungiæ 40.0); Alibert's ointment (flor. sulph. 40.0, ammon. muriat. 10.0, axung. 80.0) ; Jadelot's ointment (kalii sulphurat. 20.0, sapon. alb. 80.0, ol. olivar. 14.0, ol. thymi 1.0) ; Hebra's modified Wilkinson's ointment (flor. sulph., ol. fagi āā 40.0, sapon. virid., axung. porci āā 80.0, cretæ alb. pulv. 5.0) ; Bourguignon's ointment (ol. lavand., menthæ, caryophyll., cinnamomi āā 1.50, gumm. tragacanth. 5.0, kalii carbonat. 35.0, flor. sulph. 100.0, glycerin. 200.0).

Sulphur soap, sulphur-sand soap, naphthol sulphur soap, or Peruvian balsam is also useful, while petroleum alone and all aromatic watery decoctions are not very reliable.

In my opinion, unguentum naphtholi compositum acts most promptly in destroying the mites and their burrows, and causes least irritation of the skin. This is made according to the following formula : axung. 100.0, sapon. virid. 50.0, naphthol. 15.0, cretæ alb. pulv. 10.0. Under its use the cuniculi and the eczema eruptions shrivel up and the itching ceases at once. At the same time the skin has an agreeable flexible feel. In moderately severe cases it will be sufficient to use Peruvian balsam or a liniment made according to the following formula : styracis liquid. 5.0, petrol., ol. olivar. āā 15.0, bals. peruv. 10.0. spir. sapon. kalin. 20.0 ; or flor. sulph. 15.0, vaselin., lanolin. āā 25.0, ol. lavand., menthæ, naphæ āā gtt. 5.0.

All fatty remedies, such as balsam of Peru, styrax, etc., possess the disadvantage that, under their administration, the eczema does not dry up, and the cuniculi become pale, succulent, and colorless on account of the swelling of their epidermic covering. Hence it is difficult to convince ourselves that the mites and ova are destroyed ; and on account of the eczema and itching which develop after every form of treatment, especially after the use of sulphur ointments, the physician is easily led to renew the remedial measures. This usually is injurious to the skin and always protracts the treatment.

Preparation of the skin by baths and frictions is unnecessary, and is even harmful if eczema is present. It is sufficient to rub in the itch ointment vigorously with the hand, or an oily remedy with a piece of flannel. Special attention is devoted to the chief sites of the cuniculi. After this is done an application is made to the entire integument. Our ung. naphthol. comp. is used only once, and this suffices to effect a cure. Placing the patient between woollen blankets is unnecessary, or even injurious, as it may result in artificial eczema. After the inunction the patient wears woollen underclothing, in order that the ointment may not soil the clothing. Then the skin is allowed to become dry, the shrivelled epidermis to be exfoliated, and the irritative symptoms (urticaria, erythema, eczema papulosum), which are absent in very few cases, are allowed to disappear. After their disappearance a bath is permitted. This may be done, on the average, on the third to fifth day. For a time we endeavor to avoid

unnecessary irritation of the skin by the use of dusting powder, the prevention of sweating, and the interdiction of baths.

After severe scabies, eczema is usually left over in the shape of nodules, pustules, and weeping on the nates of shoemakers, eczema of the mammæ in women, and pustules and vesicles on the fingers. These are treated according to the well-known rules by ung. diachyli, moist compresses, cauterization with solutions of potash, sublimate hand baths and compresses, tar, zinc paste, etc.

LECTURE LV.

DERMATOZOONOSES (*continued*)—EPIZOQNOSES.

ACARUS FOLLICULORUM—PULEX PENETRANS—FILARIA MEDINENSIS—LEPTUS AUTUMNALIS—IXODES RICINUS--CYSTICERCUS CELLULOSÆ—EPIZOONOSES: PEDICULI CAPITIS, CORPORIS, PUBIS ET PEDICULOSIS S. PHTHIRIASIS—PULEX IRRITANS, CIMEX LECTULARIUS, CULEX PIPIENS—ŒSTRUS.

ACARUS FOLLICULORUM.

Syn., demodex folliculorum, simonea folliculorum. This is found in many individuals, especially those with an abundant fatty secretion and acne of the face. The contents of the sebaceous glands of the forehead, ear, nose, upper lip are removed by pressure or scraping with the back of a knife and placed under the microscope.

The mite is found to be a worm-shaped animal, 0.08 to 0.12 millimetre long and 0.02 millimetre wide (Fig. 79). The head, prolonged like a snout, has two lateral palpi, the masticatory organs consisting of two vertical mandibles; upon the posterior surface are two warty projections. The head is separated from the thorax by a semilunar furrow. The thorax is provided on each side with four stump-shaped, three-jointed feet, which end in three (five) hooks; it exhibits transverse stripes which probably extend around the body and are connected with a median longitudinal stripe. The worm-shaped abdomen, which ends in a rounded tip, is three times as long as the thorax, and provided with lateral constrictions and transverse stripes. A digestive canal, blackish bodies resembling fat drops, and a heart-shaped structure (regarded by Wedl as the embryo) have been seen in the interior. Csokor's investigations on the demodex of the pig have made it almost certain that the mite exists in both sexes and that it moults, like the itch mite. The six-legged animal which is often seen is derived from the ovum; after the first moult it becomes eight-legged, and after the second moult it is mature.

FIG. 79. ACARUS FOLLICULORUM (AFTER KÜCHENMEISTER).

From two to twenty acari are present in the follicles, usually

with the head toward the base of the follicle, but they give rise to no disease in man.

In the dog, pig, and cat a demodex folliculorum is found, which is a variety of the acarus folliculorum hominis. In these animals it produces large numbers of pustules, furuncles, and abscesses, and in the dog it may give rise to alopecia, marasmus, and death. Gruby is the only one who claims to have succeeded in inoculating dogs with the acarus folliculorum hominis.

SAND FLEA

(*syn.* pulex penetrans, rhinochoprion penetrans) is a native of Central and South America. In addition to the skin of man, that of rats, mice, and other animals also harbors the ova of the pulex (chigoe). The male runs about free. The impregnated female (about half as large as the human flea) bores its way into the skin under the toe nails, near the ankle, or on some part of the leg. The part does not swell until the lapse of two to five days, attended by violent pain and inflammatory symptoms. These may terminate in lymphangitis, abscess, gangrene, necrosis of the bones, tetanus, and death (observed in negroes). These symptoms are due to the fact that on account of the maturing of the ova in the ovary the animals grow to five times their original dimensions; the ova are then deposited in the tissues.

The natives extract the animal with a hot needle and cauterize the wound with tobacco. Extraction is easier at the beginning of the inflammatory swelling than immediately after perforation, at which time the mandibles are apt to break off.

Karsten has shown that although the greater part of the animal is situated within the cutis, the end stigma of the respiratory canal remains in connection with the air containing layer of horny cells, and respiration is thus possible.

The "ox worm" ("founza ia ngômbé"), which produces similar symptoms in the integument of the legs and has been observed by Dutrieux in East Africa, is probably the larva of a fly.

FILARIA MEDINENSIS.

Syn., dracunculus, Medina worm. This is a native of the West Coast of Africa (Senegal, Guinea), but also occurs sporadically upon the Indian and Arabian coasts, in Persia and Arabia. In Europe only imported cases are seen. This parasite is located in the subcutaneous cellular tissue of all parts of the body, beneath the conjunctiva or tongue. It is found in numbers varying from one to twenty or more. Its presence excites pain, swelling, resulting in the formation of vesicles and furuncles; when these open a part of the worm becomes visible. Fever and convulsions sometimes form part

of the clinical history, and fistulous ulceration and gangrene are sometimes left over as sequelæ. In certain cases the worm does not give rise to inflammation, and it can then be felt through the integument, or the projection of the rings on its body may even be visible.

It had long been supposed that the worm bores its way into the skin, but this is not true. The investigations of Jakobson and others have shown that the worm, from the cephalic end down, consists of a sarcode-like envelope which encloses millions of young. When the worms are removed they move actively. They are 0.5 millimetre long and 0.02 millimetre wide, have a thick head without organs of mastication, a pointed tail, and are incapable of boring into the skin. It is probable that the young develop to maturity outside of the human body, in the cyclops of streams and swamps, and that they enter the human digestive canal in the drinking water. Thence the animal passes into the tissues and lays its ova beneath the skin. According to well-authenticated cases, the interval between the entrance of the animal into the intestine and its passage into the tissues varies from five to fourteen months.

The best treatment is that practised by the negroes. As soon as the worm becomes visible, upon the opening of the abscess or furuncle, it is carefully wound upon a little stick. The operation will last ten to fourteen hours. It is checked as soon as any resistance is felt, otherwise the worm will be torn, the young brood will enter the surrounding tissues and increase the inflammation. The worm may be one to four metres long.

Cysticercus cellulosæ has been repeatedly observed in the integument, where it forms round, soft, elastic, movable, and painless tumors from the size of a pea to that of a hazelnut. They are scattered throughout the subcutaneous cellular tissue and may remain unchanged for years. When excised they present a connective-tissue envelope whose contents consist of a delicate vesicle with a long-necked head; upon the latter are found four sucking discs and a circlet of hooks. In some cases the cysticercus has also been found in the internal organs (brain, etc.) and has thus given rise to corresponding symptoms.

FIG. 80.—LEPTUS AUTUMNALIS (KÜCHENMEISTER).

HARVEST MITE.

This six-legged mite (leptus autumnalis) is easily seen with the naked eye and has a red or yellowish-red color. According to Schmarda it is the larva of thrombidium autumnale. In the autumn it is found on many vines and grasses, and occasionally bores its way into the skin, where it dies at the end of a few days. It gives rise to the most violent burning and itching,

urticaria wheals and papules. In the centre the worm is visible as a reddish speck and can be removed with a needle. The itching is relieved by cold compresses or alcoholic applications. It is best to rub in a fatty oil mixed with an ethereal oil (bals. peruv. with ol. olivar.), as this kills the mite at the same time.

Other grass and grain mites occasionally bore into the skin and produce a temporary, sometimes very violent, eruption of urticaria and eczema papulosum.

The common *wood-tick*, *ixodes ricinus*, usually found in pine, is oval in shape and yellowish red in color. The female, 1.5 millimetres long, pushes its proboscis into the skin, sucks its fill of blood, and swells into a bag as large as a bean. It often remains hanging for days in this condition. If the animal is torn off, the head is apt to be left behind, and the inflammation to which it has given rise will then last longer. It is preferable to apply an ethereal oil to the animal, whereupon it will let go spontaneously.

Varieties of *œstrus*, one of which was called *œstrus humanus* by Humboldt, occasionally perforate the skin, lay their eggs therein, and thus give rise to a painful abscess, from which the developed larvæ emerge. These and similar insects, such as the larvæ of diptera or of fleas, play only an occasional part as true parasites of the human body.

EPIZOA

AND THE SKIN DISEASES PRODUCED BY THEM.

EPIZOONOSES.

LICE, PEDICULI

CONSTITUTE the first family (pediculida) of the first subclass (parasita) in the first class (rhynchota) of insecta ametabolica. They are wingless insects without metamorphosis, with two simple small eyes, with sucking and masticating buccal parts. They first bite into the skin with the mandibles, and then insert the head into the wound in order to suck. Three varieties infest the human body:

1. The head louse, pediculus capitis.
2. The clothes louse, pediculus humani corporis s. pediculus vestimenti.
3. The crab louse, phthirius inguinalis s. pediculus pubis.

The action of lice upon the skin is the same as that of the other epizoa which infest the skin. First, injury at the point of puncture, local escape of blood and serum, with secondary formation of a crust or a wheal around the bite. Next, itching and scratching, which are followed, not alone at the site of the puncture but also in other parts of the body, by excoriations, signs of eczema in the shape of papules, urticaria, vesicles, pustules, crusts, furuncles, abscesses, and finally pigmentation. The entire symptom-complex, which may be called pediculosis, thus consists essentially of excoriations or eczema e pediculis.

These changes vary somewhat according to the variety and number of the lice and the length of time they have infested the skin. The three varieties remain pretty strictly within their own territory. Head lice rarely ever pass beyond the scalp ; clothes lice never live upon the skin, but in the folds of the clothing immediately adjacent to the skin (back of the neck and small of the back) ; and the pediculi pubis remain chiefly upon the mons veneris.

THE HEAD LOUSE

(*syn.*, pediculus capitis) is gray in color, two millimetres long. The head and limbs are thicker, the thorax broader, than in the clothes

louse; six stigmata on each side for the tracheæ, which are connected by an arch on the abdomen. The thorax is narrow, the abdomen broader, with seven blackish segments dentated at the sides, six feet with a hook upon the last tarsal joint. The males are less numerous than the females; have a prominent posterior abdominal ring; the back is provided with an anus and porus genitalis, a wedge-shaped penis, and two pairs of testicles. The female has a deep excavation at the last abdominal ring, into which empty the anus, two ovaries and their oviduct. The vaginal opening is upon the abdominal surface. In copulation the female sits upon the male.

Fig. 81.—Male Head Louse, with System of Air Tubes and Respiration Stygmen (Küechenmeister).

The ova (nits) are deposited singly, sometimes in a continuous row, and adhere to a chitinous framework which surrounds the hair like a sheath (Fig. 82, *a a*) The louse crawls along this framework from below upward, so that the lowermost ovum develops earliest (Fig. 82, 1). The young leave the ovum in three to eight days and are full grown in eighteen to twenty-one days. A female can lay fifty eggs in six days, and in eight weeks may have five thousand descendants.

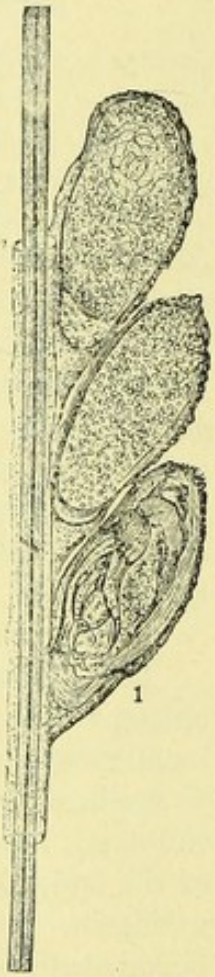

Fig. 82.—Hair with Nits of the Head Louse.

The symptoms (pediculosis capillitii) produced by the presence of head lice are those of artificial eczema with its local sequelæ and complications.

The symptoms develop most completely in females, in whose abundant hair the lice find room for indefinite increase. The pediculosis is shown by the presence of numerous discrete pustules, vesicles, and excoriations upon the back of the neck (from the scalp to the shoulders), and scattered pustules, occasionally pemphigus-like bullæ (impetigo faciei), with corresponding crusts and pigment patches. There may also be diffuse weeping eczema of the face. If the hairs are raised from the neck the lice and nits are easily seen. As the lice always deposit the ova near the scalp, the presence of nits near the ends of the hairs indicates long duration of the pediculosis, inasmuch as the nits have been pushed upward by the growth of the hair.

If the finger, separating the hairs which are matted together by masses of sebum and sticky secretion, is pushed further toward the scalp, we will discover (confined chiefly to detached spots on the occiput) all degrees of eczema. The patches are covered with crusts and pus, or they are moist and bleeding. Spots, varying from the

size of a quarter-dollar to that of a dollar, are sometimes covered with red, granular, moist, and bleeding proliferations (achor granulatus, porrigo, tinea granulata). Enlargement of the post-cervical lymphatic glands, and a pale, sleepy appearance of the patient, complete the morbid picture. The subjective symptoms are intense itching, disturbed sleep, and tenderness of the scalp.

In many cases the development of this condition can be followed from slight beginnings to the severe grades. A child gets a few lice on the head. These give rise to itching and scratching, to local extravasation of blood and serum, formation of crusts, suppuration, and tenderness. In combing, the scurf is avoided on account of the fear of producing pain. The lice thus gain a secure retreat, and they are then enabled to spread. The eczema symptoms which have been described are further results of the direct injury to the scalp by the lice and by the scratching. The inflammatory enlargement of the lymphatic glands is the result of the inflammatory processes in the scalp, while the unhealthy appearance of the patient is due to the insomnia, and perhaps also to a leucocytosis caused by the hyperplastic glands.

Pediculosis capitis is found chiefly in young people and females, particularly in puerperæ. Of course its development is favored by carelessness in combing the hair.

The *diagnosis* presents no difficulty whatever, as the presence of the lice and nits characterizes the condition. Nevertheless it is overlooked by physicians in many cases for months and years, and the patients are treated for ezcema, or, on account of the enlarged glands and pallor of the face, for scrofula.

The indications for *treatment* are the destruction of the lice and nits and the cure of the eczema.

The first indication is met by the use of petroleum. In order to avoid the risk of fire, it is best to use the following combination : petrolei 100.0, ol. oliv. 50.0, bals. peruvian. 20.0, rubbed freely into the hair. If there is slight eczema, we may use naphthol oil (five per cent) and then wrap the head in flannel. At the end of twenty-four hours the lice are dead and the ova incapable of development. The hair is then washed with spiritus saponis kalinus. As the eczema crusts have been softened by this treatment, the scalp appears clean. The hair is now combed, and many hairs are removed in the process. It is unnecessary to cut the hair. The further treatment is the same as in all eczemata of the scalp : daily oiling and washing, or the use of suitable ointments until all the eczematous patches are healed.

The chitinous framework, which adheres to the hairs in the shape of brown, shining nodes after destruction of the eggs, must now be removed. This is best done by loosening it with dilute acetic acid and then using a fine comb.

CLOTHES LICE

(*syn.*, pediculi vestimenti, pediculi humani) are larger than pediculi capitis, and are more active in their movements.

FIG 83. CLOTHES LOUSE, FEMALE (KÜCHENMEISTER).

They live exclusively in the clothing which is in contact with the body, and lay their eggs in its folds in rosary-like chains. They cannot be found on the scalp, and are present on the trunk only while obtaining nourishment. If an individual who harbors numerous clothes lice is suddenly stripped we will find upon the skin perhaps one or two lice which, being engaged in sucking, were unable to escape. These will soon begin to run about in a lively manner, looking for a place of concealment. If an individual who has suffered in this way for months puts on fresh clothing and underclothing, he will be entirely free from the vermin, although his body exhibits all the signs of

PEDICULOSIS CORPORIS.

The symptoms of this disease are very characteristic, and consist mainly of excoriations. As the lice live in the immediate vicinity of the skin, in order that they may quickly visit and leave the body, the changes in the skin occur chiefly in the parts corresponding to closely applied folds of clothing—viz., upon the neck, over the shoulders, the sacrum, waist, nates, and outer parts of the thighs.

The excoriations have a peculiar character, owing to the fact that the louse, with its large organs of mastication, produces directly a superficial injury of the skin, and that a large wheal develops around the wound. The resulting itching gives rise to scratching, and the nail meets with a rete which is loosened by hæmorrhagic and serous infiltration. Into this the nail penetrates more deeply than into the unprepared skin. This results in a broad and deep bloody excoriation which is several centimetres in length. Its centre, which corresponds to the puncture wound, is still deeper and wider. At the end of a few days the stripe-shaped prolongations of the excoriations heal, later the central portion. It is recognizable for two or three weeks by the dark pigment which is left ; after its disappearance the streaks look abnormally white and often even cicatricial in the middle.

If a few lice attack an individual for the first time, we find a few fresh excoriations, chiefly on the back of the neck and in the sacral region. If he also exhibits pigment streaks or white streaks in these localities, he must have harbored the parasites at intervals of a few weeks. In such persons we also find, in not a few cases, a general acute eruption upon the trunk in the shape of miliary papules of eczema (miliaria rubra).

In long-continued pediculosis of individuals who have been infested with lice for years with brief intervals, the symptoms are intensified to a remarkable degree. The excoriations are more numerous and deeper, associated with inflammation, suppuration, and crusts, pustules, lymphangitis, diffuse dermatitis. Large, indolent furuncles, abscess with gangrene of the skin, develop. These lesions are crowded together upon the shoulders, back of the neck, and loins. Undermining communications are formed between them, and finally result in ulcers with overhanging edges and warty granulations. This symptom-complex often lasts for months after the patients have been relieved of the lice-infested clothing.

The symptoms described are supplemented by deep dark-brown, grayish-brown, or bluish-black pigmentation of the skin. In moderate cases this sequela is confined to the back of the neck and the sacral region ; in severe cases it extends over almost the entire body. As these individuals are usually tramps and are browned from exposure to the weather, they present an appearance resembling that of Addison's disease. Indeed, there is no doubt that the literature of this disease contains some cases of severe pediculosis.

The *diagnosis* of pediculosis corporis is not always easy, because the lice cannot be found as soon as the patient removes the infested clothing. The characteristic localization of the excoriations and pigmentations will distinguish it from pruritus and chronic urticaria, in which the marks of scratching are scattered irregularly over the body. In well-to-do people who presented the symptoms of pediculosis, despite the fact that they changed their underclothing every day, I have often found the corpus delicti in the seams of a belly band which was worn constantly.

The *treatment* consists in the removal of the clothing. The infested clothing may be freed from the vermin in hospitals by placing them in an hermetically closed copper vessel with double walls, and heating the enclosed space by steam at a temperature of 60°–65° R. The mild cutaneous lesions heal spontaneously ; the more severe ones are treated according to general principles.

THE CRAB LOUSE

(*syn.*, phthirius inguinalis, pediculus pubis, morpion) has a fiddle-shaped head and broad thorax. It lives upon the hairy parts of the entire body, with the exception of the scalp, but mainly in the genital region. When present in large numbers they are also found on the hairs of the chest, axillæ, limbs, whiskers, and eyelashes, whose hairs are often covered with nits along their entire length. The louse bores deeply and lies motionless, with the head buried in the follicle, the posterior parts elevated, and the forefeet grasping the hair. In order to remove it, it must be grasped with the forceps

from behind and drawn along the hair. When they are present in large numbers, many fall off while the clothes are being removed. They give rise to very annoying itching and eczema in the form of small papules. Upon the trunk and inner surface of the thigh are sometimes found round, bluish patches—*maculæ cærulæ*—of the size of a lentil or a little larger. According to Duguet and Mallet, these are to be regarded as toxic erythema, due to a secretion from a salivary gland situated in the middle thoracic portion of the animal. Recovery is effected by one or two applications of unguentum cinereum or the more elegant preparation, præcip. alba 5.0, ung. emoll. 30.0; or sublimat 1 to 100 aq. dest. As the mercurials often produce severe eczema, it is better to use petroleum, naphthol, balsam of Peru or Tolu, ol. lauri, etc. Powder is dusted on the parts after these applications have been made, and a bath is not to be taken until the irritation of the skin has subsided.

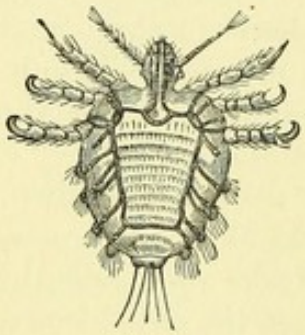

FIG. 84.—THE CRAB LOUSE (SCHMARDA).

Among the epizoa which remain temporarily on the skin, the most frequent are:

The common flea, *pulex irritans*. This insect pricks the skin, producing a punctate hæmorrhage as large as a poppy seed. While the flea is sucking, a red zone of injection, two to five millimetres large, develops around the bite. The latter soon grows pale, but the hæmorrhage does not disappear for several days, passing through the usual changes in color. When the integument is delicate, as in children, the flea also produces urticaria, either by direct contact or by reflex action. When the entire body is covered with flea bites it resembles true purpura.

The bedbug, *cimex lectularius*, acanthia lectularia, provokes intense urticaria and violent itching over the bitten parts, and also over the entire body (reflex). As the raised wheals are scratched by two or three finger nails at the same time, the excoriations occur in the shape of as many parallel, sometimes intersecting, stripes, and are scattered irregularly over the body. The differential *diagnosis* from pruritus cutaneus and urticaria chronica, and from beginning prurigo in very young children, is not easy, and depends mainly on the fact that the eruption is most distinct in the morning, just after leaving the bed, and disappears during the day.

Mention may finally be made of flies, midges (culex pipiens), and mosquitoes, which, together with many allied species, occasionally bite human beings and give rise locally and generally to wheals, œdematous and ecchymotic swelling, itching, and pain. The immediate application of ammonia or sal ammoniac is indicated.

INDEX.

A

Abortive forms of variola, 190
Acarus folliculorum, 650, 663
  scabiei, 650, 661
Acetone, 265
Acidum carbolicum, in lupus, 557
  carbolicum, in psoriasis, 309
  pyrogallicum, in lupus, 557
  pyrogallicum, in psoriasis, 314
Achor, 53
  granulatus, 346
Achorion Schönleinii, 627, 634
Achroma, 51, 481
  vitiligo, 481
Achromatia, 480
Acne, bromic, 375
  cachecticorum, 374
  contagiosa, 377
  contagiosa sèche, 122
  disseminata, 370
  exulcerans serpiginosa nasi, 373
  forms, 370
  frontalis, 372
  hordeolaris, 370
  indurata, 370
  iodic, 375
  mentagra, 370, 381
  necrotisans, 372
  punctata, 370
  pustulosa, 370
  rosacea, 377
  sebacée fluante, 122
  syphilitica, 580
  tar, 374
  telangiectodes, 374
  urticata, 372
  varioliformis, 372
  vulgaris, 370
Acne, anatomy, 371
  diagnosis, 372
  etiology, 371
  treatment, 376
Acne keloid, 383
Acnitis, 374
Acrodynia, 226
Acrothymion, 434
Actinomycosis cutis, 649
Acute exanthemata, 152
Acute, circumscribed œdema, 464
Addison's keloid, 454, 513
Addison's disease, 418
Adenocarcinoma, 605
Adenoma sebaceum fibrosum, 120
Aërophyte, 629
Aetiology, general, 61
Agaricin, agaricus, 116
Ainhum, 462
Akanthosis nigricans, 442
Albinismus partialis, 480
  universalis, 479
Albinos, 479
Aleppo boil, 297
  etiology, 299
  symptoms, 297
Algæ, 626
Algidité progressive, 462
Alibert's ointment, 661
Alopecia, 488
  acquisita, 488
  adnata, 488
  areata, 489
  areata, anatomy, 490
  areata, cause, 490
  areata, treatment, 491
  furfuracea, 123, 493
  furfuracea, anatomy, 495
  furfuracea, cause, 496
  furfuracea, treatment, 496
  neurotica, 491
  pityrodes, 493
  præmatura, 489, 492
  senilis, 488
  syphilitica, 496
Anæmiæ cutaneæ, 100
  cutaneæ, cause, 101
  cutaneæ, diagnosis, 103
Anaërophyte, 629
Anæsthesia cutanea, 564
Anaphalantiasis, 488
Anasarca, 464
Anatomy of skin, 17
Angina scarlatinosa maligna, 163, 166
Angioneuroses, 63, 98, 150, 210, 622
Angio-elephantiasis, 528

Angiokeratoma, 468
Angioma, 526
elephantiaticum, 528
lipomatodes, 528
neuroticum, 528
prominens, 527
simplex, 527
verrucosum, 527
Angioma, anatomy, 528
cause, 528
prognosis, 529
treatment, 529
Anidrosis, 116
Anomalies of the cutaneous glandular secretion, 104
of secretion of fat, 121, 130
of secretion of sweat, 104
Anthrax, true, 294
true, cause, 295
true, prognosis, 295
true, treatment, 295
simplex, 292
bacilli, 295
Argyria, 422
Arrector pili, 35
Arsenic, treatment with, 307
Asiatic pills, 308
Asperitas unguium, 451
Asphyxia localis, 98, 263
Asteatosis, 129
Aster, 428
Atrichia, 488
Atrophic cicatrix, 515
Atrophoderma pigmentosum, 499
Atrophy, 479
of the hair, 488
of the hair proper, 496
of the hair pigment, 483
of the nails, 498
of the pigment, 479
of the skin, 499
of the skin, stationary condition of, 501
Atrophy of skin proper, 499
diffuse, progressive, 503
idiopathic, circumscribed, 509
idiopathic, diffuse, 499
secondary, 504
senile, 501

B

Bacillus lepræ, 576
Bacteridia, 628
Bacterium, 629
Basidia, 627
Bedbug, 672
Biscra button, 298
Blattern, 175
Bleeder's disease, 409
Blood vessels of skin, 26
Boeck's scabies, 658
Boil, 292
Bourguignon's ointment, 661
Bouton d'Alepp, 297
Bromidrosis, 105
Burn, 265
Burow's liquor, 355
Burrow of itch mite, 355

C

Cachexia strumipriva, 475
Cacotrophia folliculorum, 436
Callositas, 430
treatment, 431
Callus, 430
Calvities, 488
Cancer, 604
cachexia, 604
chimney-sweep's, 606
connective tissue, 612
definition, 605
epithelial, 605
Cancroid, 605
corpuscles, 606
Canities, 483
anatomy, 484
præmatura, 48
senilis, 483
treatment, 486
Carbuncle, 292
anatomy, 293
cause, 293
treatment, 249
Carbunculus verus (anthrax), 294
Carcinoma, 604
lenticulare, 612
melanotic, 613
tuberosum, 613
Caustics, 84, 311, 555
Cell fission, 143
Chancre, hard, dure, 619
soft, mou, 619
treatment, 619
Charbon, 294
Chitin, 24, 668
Chloasma, 50, 415
cachecticorum, 417
caloricum, 416
diagnosis, 422
symptomaticum, 417
toxicum, 417
traumaticum, 416
treatment, 424
Chloride of zinc, 556
Chromatophores, 412
Chromidrosis, 117
Chrysarobin, 313
Cicatrix, 56, 515
atrophic, 514
hypertrophic, 514
sequelæ, 519

Cicatrix, treatment, 518
Cimex lectularius, 650, 672
Classification, 84
Clavus, 431
treatment, 432
Cleavage lines, Langer's, 59
Clinical aspect of skin diseases, 66
Cnidosis, 228
Combustio, 265
Comedo, 131
cause, 132
treatment, 134
Congelation, 278
Continuous water bed, 204, 275
Contusions, 405
prognosis, 405
treatment, 405
Corium, 20
Corn, 431
Cornu cutaneum, 432
Corpuscles of Meissner, 30
of Wagner, 30
Crustæ, 56
lamellosæ, 56
Culex pipiens, 672
Cuniculus, 653, 655
Cutis anserina, 621
laxa, 504
Cyanosis, 96
Cysticercus cellulosæ, 650, 655

D

Dal-fil, 464
Darier's disease, 443
Dartre rougéante, 539
Dasytes, 447
Decoctum Zittmanni, 591
Decubitus acutus, 264
Delhi boil, 297
Demodex folliculorum, 663
Derma, 20
Dermatitis, 260
ambustionis, 265
ambustionis bullosa, 266
ambustionis, cause of death in, 271
ambustionis, degrees, 265
ambustionis erythematosa, 265
ambustionis escharotica, 268
ambustionis, prognosis, 273
ambustionis, treatment, 274
anatomical changes in, 260
calorica, 261
congelationis, forms, 278
congelationis, prognosis, 279
congelationis, prophylaxis, 280
congelationis, treatment, 279
contusiformis, 219
diabetica papillomatosa, 265
exfoliatrice généralisée, 401
Dermatitis exfoliativa neonatorum, 250, 400
exfoliativa neonatorum, treatment, 401
herpetiformis, 398
idiopathic, 261
idiopathic a venenatis, toxica, 261
idiopathic dynamica, 261
idiopathic neuropathica, 261, 263
idiopathic traumatica, 261, 263
papillaris capillitii, 382
symptomatic, 281
Dermatology, relation to pathology, 1
history, 2
importance, 2, 14
Dermatomycoses, 630
Dermatophytes, 628
action on skin, 629
cause, 630
diagnosis, 630
prognosis, 630
relation to mould fungi, 628
site, 629
Dermatoses diabeticæ, 264
Dermatozoa, 650
Dermatozoonoses, 650
Desquamation, 55
Diagnosis, general, 67
Diapedesis, 403
Diaster, 428
Dispirem, 428
Donovan's solution, 307
Dracunculus, 664
Drug eruptions, 223
Dupuytren's powder, 557
Dyschromasia, 411
Dysidrosis, 106

E

Eau de princesse, 425
Ecchymoma, 404
Ecchymosis, 50, 404
Ecthyma, 53, 386
Eczema, 339
acute, 342
chronic, 345
chronic, localization, 345
crustosum, 340
discoides, 647
erythematosum, 340
herpetiforme, 346
idiopathic, 352
impetiginosum, 340
intertrigo, 343
larvale infantum, 346
lichenoides, 343
lotricum, 348
madidans, 340

Eczema manuum, 348
papulosum, 340
pustulosum, 344
squamosum, 340
sycosiforme, 347
symptomatic, 353
tyloticum, 348
vesiculosum, 340
Eczema, anatomy, 342
diagnosis, 350
etiology, 350
prognosis, 353
treatment of acute, 354
treatment of chronic, 357
Eczema marginatum, 350, 646
treatment, 647
Electrolysis, 449, 557
Eleidin, 24
Elephantiasis arabum, 464
congenita, 468
cruris, 465
genitalium, 467
neuromatosa, 468
scroti, 467
Elephantiasis arabum, anatomy,
diagnosis, 472
etiology, 469
symptoms, 465
treatment, 472
Elephantiasis græcorum, 561
Endarteritis obliterans, 262
Endemic furuncle, 297
Ephelis, 415
Epidermin, 80
Epidermis, 19, 21
Epidermolysis bullosa, 229, 259
Epilation, 449
Epithelioma, 605
flat, 606
nodular, 607
papillomatous, 607
Epithelioma, anatomy, 609
diagnosis, 611
etiology, 610
prognosis, 611
treatment, 612
Epizoa, 650, 667
Epizoonoses, 667
Equinia, 296
Erasmus Wilson's disease, 401
Eruption, arrangement of, 57
Erysipelas, 282
grave uterinum, 287
perstans faciei, 284, 287
Erysipelas, anatomy, 287
complications, 284
diagnosis, 288
etiology, 286
prognosis, 288
seat, 284
symptoms, 282
Erysipelas, treatment, 289
Erysipelas coccus, 286
Erysipèle salutaire, 284, 286, 288
Erythema, 50, 210
caloricum, 93
exsudativum annulare, 214
exsudativum bullosum, 214
exsudativum iris, 214
exsudativum multiforme, 213
exsudativum papulatum, 214
exsudativum urticatum, 214
fugax, 91
idiopathic, 213
infantile, 94
nodosum, 219
toxic, 222
traumaticum, 93
variolosum, 94
venenatum, 93
Erythema, cause, 217
diagnosis, 216
forms of, 212
prognosis, 216
treatment, 222
Erythrasma, 648
Erythrodermia exfoliativa, 401
Esthesiomène, 539
Etiology, general, 61
Excoriations, 54
Exfoliatio epidermidis neonatorum, 123
External applications, 59
Exudation, 141

F

Falcadina, 477
False keloid, 513
Farcy, 296
Fat, 80
degeneration, 150
secretion of, 121, 130
Favus, 630
of intestine, 633
of nail, 633
of non-hairy parts, 632
of scalp, 630
scutulum, 630
Favus, cause, 634
diagnosis, 635
prognosis, 635
treatment, 635
Febris morbillosa sine exanthemate, 157
scarlatinosa, 163
urticata, 232
Fibroma molluscum, 520
Fibromata, 524
Filaria medinensis, 650, 664
sanguinis, 469
Flat epithelioma, 606

Flea, 672
Flies, 672
Folliculitides, 370
Folliculitis barbæ, 381
Formication, 62
Fowler's solution, 607
Framboesia, 476, 582
Frost bite, 292
Fructification organ, 627
Fungi, 626
  morphology, 626
  mould, 626
  putrefaction, 626
Furuncle, 292
Furunculus Vespajo, 292
  vulgaris, 292

G

Galactidrosis, 118
Gangræna diabetica bullosa, 265
Gangrene, 149
  hysterical, 264
  marantic, 263
  multiple, cachectic, 263
  senile, 263
  symmetrical, 99, 263
General characteristics of dermatoses, 21
Glanders, 296
  diagnosis, 296
Glanders bacillus, 297
Glossy skin, 244, 263, 470
Gommes scrophuleuses, 542
Gonidia, 627
Grutum, 134
Gumma, 581
Gutta rosa, 377

H

Hair, 35
  bulb, 37
  follicle, 36
  papil a, 36
  pigment, 37
  root sheath, 35
  sac, 37
  shaft, 37, 38
  sheath of Henle, 38
  sheath of Huxley, 38
  shedding, 494
Hæmatidrosis, 118, 404, 409
Hæmophilia, 409
Hæmorrhages, 403
  forms, 404
  idiopathic, 405
  symptomatic, 407
  traumatic, 405
Hæmorrhage, anatomy, 403
  treatment, 406
Hapolonychia, 498
Hard chancre, 619
Harvest mite, 665
Hebra's modified ointment, 661
Helmerich's ointment, 660
Hemianaërophyte, 629
Hemiphalacrosis, 488
Herpes, general character, 236
  definition, 237
  types, 237
Herpes facialis, 243
  gestationis, 396
  iris et circinatus, 214, 253
  iris et circinatus, treatment, 255
  labialis, 251
  preputialis progenitalis, 252
  tonsurans, 637
  tonsurans capillitii, 637
  tonsurans maculosus et squamosus, 638
  tonsurans vesiculosus, 638
  tonsurans, diagnosis, 642
  tonsurans, etiology, 640
  tonsurans, treatment, 643
Herpes zoster, 237
Heteroplasia, 511
Hirsuties, 447
  acquisita, 447
  universalis, 447
Hirsuties, anatomy, 448
  treatment, 448
Homœoplasia, 410, 511
Horny layer of skin, 25
Horse pox, 377
Hydradenitis suppurativa, 119
Hydrosadenitis phlegmonosa, 119
Hydrargyrum, 588
  preparations, 589
Hydrogen peroxide, 487
Hyperæmiæ cutaneæ, 90
Hyperæmia, active, 92
  ex vacuo, 95, 97
  passive, 95
  passive, treatment, 99
Hyperæsthesia, 622
Hyperchromatosis, 411
Hyperidroses, 109
  cause, 113
  prognosis, 114
  treatment, 114
Hypertrichosis, 447
Hypertrophiæ, 410
  division, 410
Hypertrophy, circumscribed, connective tissue, 476
  diffuse, 454
  of epidermis and papillæ, 426
  of hair, 447
  of nails, 451
  of nails, anatomy, 451
  of nails, treatment, 452
  of pigment, 411

Hypha, 627
Hyphomycetes, 626
Hysterical gangrene, 264

I

Ichthyol, 80
Ichthyosis, 436
congenita, 125
consecutive, 441
hystrix, 437
nacrée, nitida, 437
sebacea, 125
serpentina, 437
simplex, 436
Ichthyosis, anatomy, 438
cause, 440
course, 438
treatment, 441
Icterus, 442
Idiopathic inflammations, 261
Idiopathic, multiple sarcoma, 601
Idiopathic skin lesions, 61
Idradenoma, 120
Idrosadenitis, 119
Impetigo, 53
contagiosa, 349
faciei, 668
herpetiformis, 387
herpetiformis, prognosis, 388
herpetiformis, treatment, 389
syphilitica, 580
variolosa, 191
Induration of cellular tissue, 462
Infection granulomata, 511
tumors, 511
Inflammation, 141
circulatory disorder in, 145
exudation in, 145
symptoms, 145
termination, 147
termination in inflammatory hypertrophy, 148
termination in resolution, 147
termination in suppuration, 148
Inflammatory dermatoses, 141
acute, contagious, 152
acute, non-contagious, 210
acute, non-contagious, groups, 210
chronic, 300
chronic, subdivision, 300
Inoculation, 176
tuberculosis, 546
Insecta ametabolica, 667
Intertrigo, 111
Iodine, 591
Iodine-glycerin, 377
Iodisme pétéchial, 407
Ixodes ricinus, 650, 666

J

Jadelot's ointment, 661

K

Karyokinesis, 143, 427
Keloid, 512
cicatricial, 513
forms, 513
syphilitic, 513
true, 513
Keloid, anatomy, 513
prognosis, 515
treatment, 515
Keratodermia symmetrica erythematosa, 442
Keratohyalin, 24
Keratoses, pure, 430
with papillary hypertrophy, 433
Keratosis follicularis, 333, 442, 443
Kerion Celsi, 641, 645

L

Landolf's paste, 556
Lassar's paste, 355
Lentigo, 415
Lepra arabum, 561
neurorum, 568
Lepra bacilli, 569
Leprosy, 561
anæsthetic, 564
contagiousness, 572
forms, 562
history, 561
macular, 564
nodular, 562
Leprosy, anatomy, 568
diagnosis, 567
etiology, 569
prognosis, 566
treatment, 572
Leptothrix, 629
Leptus autumnalis, 650, 665
Leucoderma, 479
acquisitum, 481
congenitale, 479
syphiliticum, 420
Leukæmia, 599
Lice, 667
clothes, 670
crab, 671
head, 667
Lichen, 321
diabeticus, 524
hæmorrhagicus, 404
pilaris, 436
planus, 326
ruber, 325

Lchen ruber acuminatus, 325
ruber moniliformis, 331
ruber planus, 326
scrofulosorum, 321
syphiliticus, 580
urticatus, 214
ruber, 325
Lichen ruber, anatomy, 330
ruber, cause, 328
ruber, diagnosis, 333
ruber, prognosis, 328
ruber, treatment, 334
Lightning stroke, 277
Liodermia essentialis, 263
Lipoma, 524
Liquor Burowii, 355
Lister's liniment, 204
Livedo calorica, 98
mechanica, 96
neuroparalytica, 98
Lombardy leprosy, 224
Lues, 575
Lupus erythematosus, 124, 505, 548
discoides, 505
disseminatus, 506
erythematosus, anatomy, 507
erythematosus, diagnosis, 508
erythematosus, etiology, 509
erythematosus, treatment, 509
pernio, 510
vulgaris, 539
vulgaris, cancer in, 553
vulgaris, localization, 540
vulgaris of mucous membranes, 540, 553
vulgaris of skin, 539
vulgaris disseminatus, 541
vulgaris exfoliativus, 539
vulgaris exulcerans, 539
vulgaris papillaris, 541
vulgaris scléreux, 552
vulgaris sclerosus, 541
vulgaris serpiginosus, 541
vulgaris tumidus, 539
vulgaris verrucosus, 541, 552
vulgaris ulcers, 540
vulgaris, anatomy, 549
vulgaris, cause, 543
vulgaris, diagnosis, 547
vulgaris, prognosis, 543
vulgaris, treatment, 553
Lustgarten's bacillus, 585
Lymphadenie cutanée, 596
Lymphagoga, 144
Lymphangiectases, 529
Lymphangioma cutis, 529
tuberosum multiplex, 530
tuberosum, prognosis, 532
tuberosum, treatment, 532
Lymphatic system of skin, 27
warts, 531
Lymphodermia perniciosa, 598

M

Macrochylia, 530
Macula, 50
Maculæ atrophicæ, 502
cœrulæ, 672
Mal del sole, 224
rosso, 224
Maliasmus, 296
Malignant œdema, 295
pustule, 294.
Malpighian layer, 22
Mast cells, 412
Measles, 154
atypical forms, 157
cause, 159
complications, 158
diagnosis, 160
periods, 154
prognosis, 161
prophylaxis, 162
treatment, 162
Medicinal exanthemata, 64, 95, 211, 223
Melanosis, 51
Mercury in syphilis, 588
preparations, 589
Micrococcus, 629
Microsporon Audouini, 490
anomœon, 640
furfur, 627
minutissimum, 648
Midges, 672
Miliaria, 256
alba, 256
crystallina, 257
rubra, 256
Milium, 134
cause, 135
treatment, 135
Molluscum contagiosum, 136
contagiosum verrucosum, 136
contagiosum, anatomy, 138
contagiosum, corpuscles, 139
contagiosum, treatment, 140
fibrosum, 520
fibrosum, anatomy, 521
fibrosum, treatment, 521
Morbilli, 154
apyretici, 157
asthenici, 162
synochales, 162
typhosi, 157
Morbus Addisoni, 418
maculosus Werlhofii, 408
Morphœa, 455, 564
Mosquitoes, 672
Multiple, cachectic gangrene, 263
sarcoma, 601
Muscles of skin, 32
Musculi arrectores, 33
Mycelium, 626

Mycosis (Alibert), 476
framboesioides, 593
fungoides, 593
fungoides, anatomy, 595
fungoides, diagnosis, 597
fungoides, stages, 594
fungoides, treatment, 598
Myoma, 525
Myxœdema, 474
treatment, 475

N

Nævus, 50, 413, 527
flammæus, 527
lipomatodes, 413
mollusciformis, 413
pigmentosus, 51, 413
pilosus, 413, 447
spilus, 413
tuberosus, 527
unius lateris (nerve), 413
vascularis, vasculosus, 50, 527
verrucosus, 413, 447
Nævus, etiology, 414
Nail, 40
bed, 41
Naphthol, 315, 441
Necrobiosis, 149
Neoplasmata, 511
Nerve endings, 30
nævi, 413
papillæ, 29
Nerves of skin, 29
varieties, 32
Nettle-rash, 228
Neurofibroma, 468, 520, 524
Neuromata, 524
Neuroses, 621
motor, 621
sensory, 622
trophic, 621
New growths, 511
classification, 512
connective tissue, 512
malignant, 561
Nits, 668
Nuclear division, 427
scheme, 428

O

Objective symptoms, 48
Œdema, acute, circumscribed, 464
Œstrus, 666
Onychatrophia, 498
Onychogryphosis, 451
Onychomycosis favosa, 633
trichophytina, 644
Ophiasis, 488
Opistophalacrosis, 488
Oriental plague, 406, 407
Osmidrosis, 105
Osteoma cutis, 525
Ox worm, 664

P

Pachydermia, 464
Pacinian corpuscle, 31
Pædophlyctis, 259
Paget's disease, 443
Papillæ, 21, 27
Papilloma, 476
inflammatory, of Weil, 477
neuroticum, 433
Papule, 51
Paræsthesia, 622
Parasites, 626
animal, 650
vegetable, 626
Parasitic dermatoses, 626
Paronychia, 451
Pathological processes, 16
Pearson's solution, 307
Pearl bodies, 552
Pediculi capitis, 650, 667
pubis, 650, 671
vestimentorum, 650, 670
Pediculosis capillitii, 668
capillitii, treatment, 669
corporis, 670
corporis, treatment, 671
pubis, 671
pubis, treatment, 672
Peliosis rheumatica, 407
Pellagra, 224
etiology, 225
treatment, 226
Pemphigus, 258, 390
acutus, 258
cachecticus infantum, 392
circinatus, 391
confertus, 391
contagiosus, 349, 401
crouposus, 392
disseminatus, 391
diphtheriticus, 392
diutinus, 391
epidemicus, 359, 401
febrilis, 258
foliaceus, 393
gyratus, 391
leprosus, 397
malignus, 392
pruriginosus, 393
syphiliticus, 398, 583
vegetans, 392
vulgaris, 390
variolosus, 191
Pemphigus, anatomy, 394
causes, 396

Pemphigus, diagnosis, 398
prognosis, 399
treatment, 399
Perniones, 278
Petechiæ, 50, 404
Pétite vérole, 175
Phalacrosis, 488
Phlegmonous inflammations, 201
Phlyctænoses, 210, 236

Phthiriasis, 671
Phthirius inguinalis, 671
Phyma, 52
Physiology, 42
Pian, 476
ruboid, 477
Pick's emplast. saponatum, 358
Pigment, 23
Pigment atrophy, 479
cancer, 613
hypertrophy, 411
hypertrophy, acquired, 415
hypertrophy, congenital, 413
hypertrophy, formation, 411
syphilis, 419
Pigmentation, 56
Pityriasis, 55
capillitii, 123
maculata et circinata, 649
rosée, 640
rubra, 318
rubra, anatomy, 320
rubra, diagnosis, 320
rubra, treatment, 320
rubra pilaris, 337
simplex, 129
versicolor, 627, 647
versicolor, treatment, 648
Plasma cells, 550
Plasters, 81
Plenck's solution, 435
Pleomorphia, 628
Plexiform neuroma, 468, 592
Plica polonica, 450
Poliosis, 480, 483
Polytrichia, 447
Post-mortem pustule, 297
Powders, 83
Prickly heat, 256
Primary lesions, 50
Prognosis, general, 72
Pruriginous dermatoses, 339
Prurigo, 363
agria, 364
hiemalis, 367
mitis, 365
summer, 367
Prurigo, anatomy, 366
diagnosis, 365
etiology, 367
reatment, 6
Pruritus, 622
Pruritus, analis, 624
genitalium, 624
hiemalis, 624
localis, 624
universalis, 622
Pruritus, diagnosis, 624
etiology, 623
prognosis, 624
treatment, 625
Pseudoerysipelas, 291
Pseudoleukæmia, 599
Psoriasis, 301
anatomy, 304
development, 302
diagnosis, 305
localization, 302
prognosis, 306
treatment, 307
Psoriasis palmaris et plantaris, 303, 576, 579
Psorospermiæ, 446, 611
Psorospermosis, 442
anatomy, 445
Psydracium, 53
Pulex irritans, 650, 664
penetrans, 672
Pulvis Cosmi, 556
Purpura, 50, 221, 407
hæmorrhagica, 408
hæmorrhagica, treatment, 408
neonatorum, 406
papulosa, 408
pulicosa, 406
rheumatica, 221, 407
rheumatica, treatment, 222
senilis, 406
simplex, 408
variolosa, 184
Pustula, 54
Pustular eruptions, 386

Q

Quincke's œdema, 464
Quinine eruption, 224

R

Radesyge, 477, 582
Raynaud's disease, 98, 263
Rete, 22
Rhagades, 55
Rheumatokelis, 407
Rhexis, 403
Rhinophyma, 378
Rhinoscleroma, 533
anatomy, 535
bacilli, 537
cells, 536
prognosis, 538
treatment, 538

Rimæ cutis, 55
Ringed hairs, 497
Ringworm, 637
Risipola lombarda, 224
Rodent ulcer, 605, 606
Rosalia, 163
Rose, 282
Roseola, 50, 223
  calorica, 94
  infantilis, 94
  syphilitica, 223, 578
  typhosa, 94
  vaccinia, 94
  variolosa, 94, 178
Rötheln, 161
Rougeole, 154
Rubeola, 154
Rupia, 56
  syphilitica, 577
  variolosa, 191

S

Sand flea, 664
Saprophytes, 626
Sarcoid tumors, 593
Sarcoma, 599
  idiopathic, multiple, 601
  idiopathic, multiple, treatment, 602
  melanotic, 600
  third type, 602
  third type, diagnosis, 603
  third type, treatment, 603
Sarcomatosis cutis, 599
Scabies, 655
  norwegica, 658
  pecorina, 659
  pustulosa, 657
  development, 659
  diagnosis, 659
  prognosis, 660
  symptoms, 655
  treatment, 660
Scabrities unguium, 451
Scarlatina, 163
  hæmorrhagica, 168
  lævigata, 166
  miliaris, 166
  papulosa, 166
  puerperalis, 173
  septica, 168
  variegata, 166
Scarlatina, 163
  anomalous forms, 165
  complications, 166
  diagnosis, 172
  etiology, 171
  periods, 163
  prognosis, 169
  sequelæ, 169
Scarlatina, symptoms, 163
  treatment, 173
Schizomycetes, 629
Sclerema adultorum, 455
  neonatorum, 462
  course, 463
  treatment, 463
Sclérémie, 454
Sclerodactylie, 454, 457
Scleroderma, 454
  anatomy, 459
  cause, 459
  course, 458
  diagnosis, 461
  localization, 457
  prognosis, 461
  symptoms, 455
  treatment, 461
Sclérodermie, 455
Sclerosis telæ cellulosæ, 454
Sclerostenosis cutanea, 454
Scorbutus, 409
Scrofuloderma, 512, 558
Scrofulosis, 558
Scutulum, 630
Sebaceous glands, anatomy, 39
Seborrhœa, 122
  capillitii, 122
  localis, 122
  nigricans, 123
  oleosa, 122
  sicca, 122
  syphilitica, 123
  universalis, 125
Seborrhœa, diagnosis, 126
  prognosis, 126
  treatment, 127
Sebum warts, 131
Secondary lesions, 54
Senile atrophy, 501
  warts, 501
Shedding of hair, 494
Shingles, 237
Sifilide pigmentaria areolata, 420
Simonea folliculorum, 663
Sivvens, 477
Siwwens, 582
Skin as protective covering, 42
  as organ of secretion, 44
  as sense organ, 45
Small-pox, 175
  history, 175
Soaps, 81
Spirem, 428
Spiritus saponis kalinus, 127
Spontaneous gangrene of hysteria, 264
Sporisorium maidis, 226
Squamæ, 55
Squamous dermatoses, 300, 301
St. Anthony's fire, 282
Staphylococcus albus, 139

Staphylococcus pyogenes, 139
Sterigma, 627
Streptococcus erysipelatis, 286
Striæ atrophicæ, 503
Subcutaneous connective tissue, 20
Subjective symptoms, 48
Sudamina, 256
Suppuration, 148
Sweat, 104
  abnormalities of secretion of, 106
  secretion of, 105
Sweat glands, 34
  functional disorders of, 104
Sycosis, 381
  cause, 384
  diagnosis, 383
  prognosis, 384
  treatment, 384
Sycosis parasitaria, 383, 644
  cause, 645
  diagnosis, 645
  treatment, 645
Symmetrical gangrene, 263
Symptomatic skin lesions, 61
Symptomatic inflammations, 281
  cause, nature, 281
Symptomatology, general, 48
Syphilides, 575
  general character, 575
Syphilis, 575
  corymbosa, 581
  cutanea vegetans, 582
  framboesiaformis, 582
  gummatosa, 581
  hereditary, 582
  lenticularis, 579
  maculosa, 578
  nodular, 581
  orbicularis, 579
  papulosa, 578
  pustulosa, 580
  ulcerative, 581, 620
Syphilis, treatment, 584
  by drugs, 588
  of initial lesion, 584
  methods of, 588
Syphilitic rupia, 577
  ulcers, 577
Syphiloderma, 512
  pigmentosum, 419
Syringocystadenoma, 120
System of skin disease, Hebra's, 87

T

Tar, 83, 311
Tattooing, 423
Teinte broncée, 418
Telangiectasis, 50, 526
Therapy, general, 74
Thiol, 81
Threefold function of skin, 42
Tinctura rusci, 311
Tinea favosa, 630
  imbricata, 639
  tondens, 637
  trichophytina, 637
Treatment, 76
  internal, 84
  local, 79
  means and methods, 76
Trichauxis, 447
Trichoptilosis, 497
Trichophyton tonsurans, 627, 640
Trichorrhexis nodosa, 497
Trophoneuroses, 621
Tubercle bacilli, 545
Tubercula, 51
Tuberculin, Koch's, 553
Tuberculosis cutis, 558
  diagnosis, 559
  verrucosus, 560
Tumenol, 81
Tumor cavernosus, 528
Tumors, sarcoid, 593
Tyloma, 430
Tylosis, 430
Typhus fever, eruption, 223

U

Ulcer, 614
  contagious, 619
  idiopathic, non-contagious, 616
  scrofulous, 618
  symptomatic, non-contagious, 618
  syphilitic, 577
Ulcera cutanea, 55, 614
Ulcus cruris, 616
  treatment, 617
Ulerythema acneiforme, 492
  ophryogenes, 492
Unguentum diachyli, 115
  naphtholi compositum, 661
  vaselini plumbicum Kaposi 355
  Wilsoni, 355
Unna's ointment, 336
Uridrosis, 118
Urtica, 52
Urticaria, 228
  annularis, 228
  bullosa, 228
  chronica, 230
  evanida, 229
  factitia, 228
  giant, 464
  gyrata, 228
  pigmentosa, 233
  porcellanea, 228
  recidiva, 230
  tuberosa, 239

Urticaria, anatomy, 229
etiology, 230
prognosis, 233
treatment, 234

V

Vaccination, 176, 205
Vaccine eruption, 207
Varicella, 177
Variola, 175
anomalies, 182
apyretica, 183
confluens, 190
hæmorrhagica, 184
hæmorrhagica, forms of, 184, 187
sine variolis, 190
vera, 178
vera, stages, 178
Variola, anatomy, 198
complications, 191
diagnosis, 198
etiology, 200
prognosis, 199
prophylaxis, 205
treatment, 202
Variolation, 176
Varioloid, 177
Vaso-constrictors, 32
Vaso-dilators, 32
Vaso-motor nerves, 3
nervous system, 63
Vater's corpuscles, 31
Vernix caseosa, 123
Verruca, 433
acquisita, 433
caduca, 433
congenita, 433
filiformis, 435
glabra, 434
necrogenica, 560
perstans, 433
plana, 433
senilis, 434
vulgaris, 433
Veruga, 478
Vesicula, 52
Vesicular eruptions, 236, 390
idiopathic, 236, 256
neuritic, 236
Vibices, 50, 404
Vibrio, 628
Vienna paste, 557
school, 11
Vitiligo, 479, 481
anatomy, 482
cause, 482
diagnosis, 481
treatment, 483
Vitiligoidea, 51, 521
Vitreous membrane, 38

W

Wagner's corpuscles, 30
Wart, 433
senile, 434, 501
anatomy, 435
treatment, 435
Water bed, 204, 275
Weil's disease, 223
Wheal, 52, 229
Wilkinson's ointment, 312
Wilson's ointment, 355
Wood tick, 666

X

Xanthelasma, 521
Xanthodermia, 524
Xanthoma, 51, 521
diabeticum, 524
en tumeur, 522
mollusciforme, 523
planum, 522
tuberosum, 522
Xanthoma, anatomy, 523
diagnosis, 523, 524
treatment, 524
Xanthoma cells, 523
Xeroderma, 499
pigmentosum, 499
pigmentosum, anatomy, 500
pigmentosum, cause, 500
pigmentosum, diagnosis, 501
pigmentosum, treatment, 501
second form, 501

Y

Yaws, 476

Z

Zittmann's decoction, 591
Zona, 237
Zooglea, 629
Zoonoses, 650
Zoster, 237
bilateralis, 243
gangrænosus, 241
gangrænosus hystericus, 242, 264
neuriticus, 241
Zoster, anatomy, 239, 248
diagnosis, 250
disease of ganglia in, 248
etiology, 243
localization, 244
sequelæ, 242
symptomatology, 240
treatment, 250