DERMATOLOGY

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DERMATOLOGY

The Essentials of Cutaneous Medicine

BY

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TO

HELOISE DAVISON HIGHMAN

PREFACE

In spite of otherwise great excellence, works on dermatology usually ignore the teaching needs of beginners. Cutaneous medicine is so confusing that, unless its study is gradually led up to, it appears chaotic. Indeed, frankness compels the admission that it is never quite simple, even to an accredited expert. Another reference book would have no justification. But this volume aims to present essentials succinctly, consecutively, completely and simply, without sacrificing important detail. Thus, in spirit, it is dedicated to the novice and to the general practitioner, too busy to grapple with technicalities and abstractions. This group of readers should not be burdened with literary citations that cannot possibly interest them; nor with histological descriptions which, unless exhaustive and technical, are valueless, and in any case incomprehensible except to pathologists; nor with elaborate historical sketches that are mainly exhibitions of pedantry. Accordingly, matter of this sort, unless of peculiar practical importance has been omitted since it would add to bulk rather than strength.

Nothing germane has been sacrificed. What remains represents the author's conception of the minimum requisite for an adequate outline of the subject. Less important diseases appear in finer type, not to create an impression that they may be slurred, but to convey graphically their relative place among dermatoses. And to the practitioner also this arrangement will be welcome, for he will find the kernel without himself having to do the husking. Numerous tables have been compiled classifying diseases, their etiology and relationship, always with the combined idea of constructiveness and analysis. There is no excess of illustrations, but the pictures have been selected purely for their didactic value, and the points they purpose to clarify are categorically enumerated. Thus, although in number they do not compare with what is found in many other books, if properly used, they will prove adequate to the requirements of readers seeking a clear-cut impression of the ground work of dermatology. There are about four hundred more or less distinctly established skin conditions, and the beginner or general practitioner is probably not intimately concerned with more than one-quarter of

them. It is the smaller fraction that is accompanied by pictures, although the rest too are fully discussed in the text.

The subject has been handled, so far as practicable, from the standpoint of internal medicine. The etiology of skin diseases, as a whole, is not yet well understood. Nevertheless, it is obvious that the integument is not autonomous, and that it is greatly influenced by, and greatly influences, the rest of the body. Premature and tentative as must be beliefs connected with this interrelation, it is high time that they were stated, either to be sustained or overthrown in the future. They are set forth in this book, but our knowledge does not yet permit a classification of dermatoses solely on etiological grounds.

Nomenclature in dermatology is discouraging, involved and often unsound. Some terms, however fanciful, have become permanent. Lichen is an example of this. Since its ancient symbolism connotates something definite, there is no reason to assail it. Not so the word eczema! The disease thus styled is one with vesicular dermatitis in aspect, causation, histology and course. Therefore, the sounder term should replace it. The word is nowhere independently employed in this book, but is used parenthetically after the expression dermatitis, wherever the latter appears. This compromise seemed expedient, for the undesirable term, not yet having been abandoned, must be continued until opinion becomes unanimous as to its further restriction. Scientifically, there is nothing to be said for the word eczema. We speak of occupational dermatitis and occupational eczema, of seborrhoeal dermatitis and seborrhoeal eczema, of fungus dermatitis and fungus eczema, of dermatitis of unknown origin and eczema of unknown origin, etc., always meaning in each couple the identical thing. This is a handicap, particularly since dermatitis, being philologically and medically an accurate designation, is preferable; and such writers as Norman Walker, and Pusey have accepted the fact. Another complex subject is cutaneous tuberculosis. Here again there has been a plethora of verbiage. So far as possible an effort has been made at simplification. Congenital syphilis, too, is masked by a literary smoke screen, partly through the inaccurate interpretation of the term hereditary, partly through a series of superstitions that still sustain about a comparatively simple thing an artificial cloud of obscurity. With the utmost respect for venerable traditions, it has been attempted to divest a difficult field of medicine of unnecessary encumbrances.

As to therapy, the author has described only what has commended itself to him through personal experience. As knowledge grows

of the cause of, or of the relation of skin to general disturbances, treatment has become both simpler and better. This fact, and the precise accomplishments possible through the use of mechanical agents, such as the X-Rays, carbon dioxide snow and the like, indicate that in extensiveness the dermatological materia medica will shrink as therapy becomes more accurate. Treatment, symptomatology, differential diagnosis, and the significance of skin lesions in their relation to morbid biology are the really important considerations. These constitute the theme of this book. It is hoped that students who seek a rational exposition of the essentials of cutaneous medicine will here find their requirements satisfied. The true dermatologist is an internist who knows the skin.

The photographs appearing in this volume come from the collections of Dr. John A. Fordyce and Dr. George M. Mackee whom it is a great pleasure to thank for them. A similar expression of gratitude is extended to Dr. Jeffrey C. Michael for his valuable aid and suggestions.

W. J. H.

New York, N. Y.

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CHAPTER I

THE ETIOLOGY OF SKIN DISEASES

By far the most important branch of the science of dermatology is the study of the causation of skin diseases. Upon definite knowledge of etiology alone depends rational treatment; and in the care of dermatoses, as in all other diseases, only rational treatment can be successful. The struggle to classify disease, to increase precision in diagnosis, to recognize new diseases, has so engrossed scientists that the cure, the one thing of interest to patients, has by comparison been neglected. This is more emphatically true of dermatology than of most other subdivisions of internal medicine. Since the time of the great Viennese, Hebra, until recently, dermatologists have bent their efforts in the direction of classifying and arranging dermatoses mainly according to their clinical appearance. Thus there has developed among dermatologists a slavish attentiveness to the minutest details of skin manifestations in the hope that by constant and further analysis of the lesions, their grouping and color, etc., a satisfactory classification or understanding of the diseases might be reached. This has engendered among dermatologists unthinkably keen powers of observation, and in dermatology excessive refinement as to the characteristic features upon the basis of which similar diseases are differentiated one from the other.

The index to a work on dermatology thus is long, it would seem, to the point of attenuation, and to the medical student or to any reader who is not a dermatologist, both index and pages must appear almost chaotic. Nor can it be stated that the matter is entirely simple even for the specialist. The number of dermatoses recognized as clinical entities is enormous, and will so remain even after our criteria become more settled. We have already ceased to regard eczema of the axillae, scalp, face, body, genitalia, legs et cetera, and weeping, scaling and lichenified eczema as different diseases, although

there was a day when this disease, and many others, were subdivided in the manner above outlined, and when each subdivision was regarded as a clinical entity. The most casual accident of appearance or localization was in itself considered sufficient for the creation of another skin disease. The disease was frequently designated in the tongue of the original observer, but more frequently still in Latin or Greek, or an unwholesome blend of both, and usually the name of the author was appended with a Latin genitive ending. These matters are mentioned to account for many of the avoidable sources of confusion in dermatology. A name is but a symbol, and just as every physician knows what Bright's disease is, or hip disease, or a cold, although none of these designations is good, so every dermatologist must accommodate himself to the disadvantages of such names as white-spot disease, lichen planus, eczema, pityriasis rosea; for if these names were to be changed, either the entire dermatological literature would have to be re-written, or discarded and re-commenced. Neither of these two possibilities is practicable, so that it remains to revise our ideas on dermatoses partly with regard to their pathology, but more especially with regard to their causation.

Hebra and his pupil, Kaposi, of Vienna, dominated dermatology during the last two decades of the past century, and their philosophy reduced to its lowest terms was one in which the integument was considered an organ largely dissociated from the tissues which it enveloped. They tended to regard all dermatoses as peculiar to the skin. This is all the more astonishing in view of the fact that it was Hebra who discovered the use of arsenic in one form of lichen, thus converting the latter from a serious to a mild disease. How one so astute as Hebra could at once maintain the doctrine of the independence of the skin from the rest of the body and attempt to cure a disease essentially restricted to the skin by internal medication is inexplicable. It is equally difficult to grasp why with so patent an illustration of the internal origin of dermatoses as in lichen, he failed to perceive its significance, and thus failed to try to clear up the origin of such diseases as psoriasis and many of the eczemas. But Hebra lived in a day in which the study of micro-organisms was engaging the world, and in which they were being held accountable for all diseases. Hebra was not a good bacteriologist, and he was an even worse histologist, although he sensed the importance of this subject, and referred for study to his former master, Rokitansky, all dermatological questions within this field. The bearing of biochemistry on dermatology was not even thought of in Hebra's time,

and questions included in serology and studies in anaphylaxis which to-day are engaging us, were then, of course, unknown.

It must be remembered, however, that while the Teutons were endeavoring to restrict all dermatoses to the skin, the French had, for nearly a century, been approaching the problem by another avenue. As early as 1777 Lorry, in his "*Tractatus de Morbis Cutaneis,*" under the Hellenic influence of the humoral causation of disease, sought to explain the etiology of many skin diseases by "arthritism." Alibert and Rayer actually tried to classify dermatoses on a basis of their internal causation. The Frankish tendency to correlate all given effects with definite causes, and to find for each an antidote or specific, led the Parisians and their followers far afield. Thus, while the Germans were cataloging dermatoses without reasoning backwards from them, the French were studying etiology without reasoning forwards. Until within recently the two extremes failed to meet. Then a few deductive thinkers endeavored to co-ordinate cause and effect, and a new era in dermatology began to dawn.

In 1839 Schoenlein discovered the fungus which produces favus. In 1853 Gruby rediscovered the microsporon, which had been found by Audouin, and which causes a certain variety of ring-worm, while Eichstedt found the exciting agent of pityriasis versicolor, the microsporon furfur; and Bärensprung that of erythrasma, the microsporon minutissimum. The significance of these discoveries was not realized until many years had elapsed when Pasteur and Lister revivified the study of bacteriology. During the last quarter of the Nineteenth Century this science was being ardently pursued, while that of histopathology was being quite as ardently developed. As regards bacteriology, enthusiasts sought to explain all diseases by the activity of microscopic agents, and the pendulum swung too far. Thus Darier regarded psorosperms, which were later found to be epithelial cell degenerations, as the organisms which caused the disease named after him; Lustgarten isolated the bacillus which he held responsible for syphilis; Unna described a bacillus which he maintained to be the cause of acne. Unna was, however, the pioneer in cutaneous histopathology, and under his influence this science began to flourish.

It is probable that the history of any branch of internal medicine would resemble that of dermatology. The clinical study of this subject is actually the macroscopic pathology of the skin. Its microscopic pathology bears the same relation to the dermatoses that the

microscopic appearance of any lesion anywhere in the body bears to its gross appearance. To a certain extent the etiology of disease may be illuminated by an understanding of its microscopic composition. To a larger extent, however, the etiology may be explained through the medium of bacteriology, biochemistry and serology in its broadest sense. A diagnosis should not merely be equivalent to labelling a disease, but should explain, subject to the limitations of human knowledge, its cause. Nothing may be neglected within the scope of physical diagnosis, laboratory diagnosis, or a knowledge of the course and character of illnesses, which furthers this end. Thus, a dermatologist must be equally a clinician, and a student of the significance of laboratory procedure, if not actually a trained laboratory worker. Only then may he aspire to be an intelligent and successful therapeutist. In other words, dermatology is not a science *sui generis,* but a branch of internal medicine with all that this concept implies.

Every organ, not excepting the skin, is subject to two large groups of morbid processes, those peculiar to itself, and those which include it in the course of systemic disease. The former are autochthonous; the latter are due to the participation of the given organ in the general pathological reaction of the body. In the second group the local reaction is modified by the anatomic and physiologic peculiarities of the given organ. Thus in syphilis, the skin is differently affected from the meninges, although in the pathological changes in both may be found common features. The same is true for example of leukemia, Hodgkin's Disease and tuberculosis. Furthermore, systemic diseases may predispose to skin diseases of an entirely different nature, as diabetes, in which the skin may remain normal, be slightly inflamed, itch more or less, become infected with pus organisms, or become gangrenous. Further illustrations would be superfluous.

Pure diseases of the skin are numerous. Professional dermatoses, erythrasma and ring-worm will serve as examples. Even here, however, the autonomy of the skin is incomplete, for in deep ringworm, a cutaneous disease pure and simple so far as human eye may discern, there is a systemic response. The serum of individuals afflicted with this malady gives specific and group reactions when the Bordet-Gengou test is applied, and the skin of such patients manifests allergic phenomena in the sense of von Pirquet. Not every charwoman develops a dermatitis of the hands, nor every surgeon a bichloride rash; not every human being exposed gets ring-

worm or ivy poisoning. Thus, even in these dermatoses more is required than the mere operation of an external agent. A predisposition on the part of the patient is needed, and the significance of this is the presence of some anatomic, physiologic or metabolic characteristic which favors his liability to an illness to which his brother is not subject.

Thus the distinction between dermatoses considered local and dermatoses which are not, is more one of convenience than one which will withstand the test of fine philosophic analysis. It is nevertheless a distinction of practical value in arranging and classifying skin affections, although more or less arbitrary.

CHAPTER II

AUTOCHTHONOUS SKIN DISEASES

Autochthonous maladies of the skin are either congenital or acquired. The congenital types are either accidental, that is, peculiar to the newborn individual himself; or hereditary, that is, transmitted from an immediate or remote ancestor to the newborn individual by virtue of some property of the chromosomes of the parental cells. No expressions are more loosely used in medicine than the words hereditary and congenital. Not every condition that is congenital is hereditary, or there would be in nature no such thing as variation, the fundamental requisite for the origin of species. On the other hand, every hereditary characteristic, whether actually present at birth or not, is congenital. If it is not present at birth, but develops after weeks, months, or years, it is an hereditary characteristic evincing itself later in life, the congenital impulse to which has until then been dormant.

Congenital diseases of the skin which are usually not hereditary are nevi. Ichthyosis may be congenital and either hereditary or not hereditary. It may also appear later in life as an hereditary or non-hereditary disease. The determination of this point, not only in ichthyosis but whenever similar questions arise, will depend entirely upon the family history. Keratosis palmaris et plantaris hereditaria is a family disease which is rarely present at birth. The same is true of monilethrix. Acquired dermatoses are caused by physical and chemical agents, vegetable and animal parasites, undiscovered infectious agents, and by the influences of occupation, habit, environment, race, age and sex. Before proceeding with a further discussion of these facts, it may be desirable to present them in tabular form with simple illustrations.

GROUP 1. CONGENITAL AND HEREDITARY DISEASES

Section A.	Congenital Diseases (Not Hereditary)	Nevi — Most Forms. Ichthyosis — Some Forms.	
Section B.	Congenital Diseases (Hereditary)	Nevi Ichthyosis	Some Forms.

Section C. Hereditary or Family Diseases (Delayed in Appearing)
- Ichthyosis — Some Forms.
- Epidermolysis Bullosa Hereditaria.
- Keratosis Palmaris et Plantaris Hereditaria.

Group II. Acquired Diseases

Section A. Physical Agents.

1. Light
   - Sunburn.
   - X-Ray Burns.
   - X-Ray Keratoses and Epitheliomata.
2. Heat
   - Burns whether due to fire, electric sparks, or hot substances.
3. Cold
   - Frost-bite.
4. Exposure
   - Seaman's skin.
5. Pressure
   - Clavus.
   - Callous.

Section B. Chemical Agents.

All cause vesicular, bullous urticarial or ulcerating dermatoses.

1. Metallic and Non-metallic Elements.
2. Acids and Acid Salts.
3. Alkalis and Alkaline Salts.
4. Vegetable Poisons
   - Rhus toxicodendron.
   - Poison Oak.
   - Primrose.
   - Nettles.
5. Vegetable and Mineral Oils and Tars (Tar Acne).
6. Dyes
   - Mineral.
   - Vegetable.
   - Aniline.

Section C. Vegetable Parasites.

1. Bacteria
   - A. Cocci, Streptococci
     - Impetigo contagiosa.
     - Erysipelas.
     - Erysipeloid.
   - Staphylococci
     - Folliculitis.
     - Furunculosis.
   - B. Bacilli
     - Tubercle bacillus — T.B. verrucosa cutis.
     - Klebs-Loeffler bacillus — Impetigines.

2. Fungi { Commonest examples, pityriasis versicolor (microsporon furfur) and various types of ringworm, favus, etc.

Section D. Animal Parasites.

1. Protozoa { Oriental boil.

2. Closely related to protozoa, or protista, Spirochaeta causing verruca Peruana, not to be confused with Oroya fever, which is a systemic disease caused by a bacillus.

3. Insects, etc. { Pediculosis. Scabies. Stings of bees, mosquitoes, wasps, bed-bugs, larva migrans.

4. Jelly fish. { Wheals.

5. Vermes { Leech-bite. Anal eczema from thread worms, etc.

Section E. Undiscovered Infectious Agents

1. Mollscum contagiosum.
2. Verruca plana.

{ Recently Wile has discovered a filtrable virus capable of producing these lesions.

Section F. Occupation.

Surgeons, nurses, orderlies, charwomen, employees in slaughter houses, factories, etc., are prone to develop a great number of dermatoses, mostly on the hands. The immediate causes of these diseases are such physical or chemical agents as are peculiar to their particular work. See Group II., Section A., 1–5; and Section B., 1, 2, 3, 5 and 6.

Section G. Habit.

Habit and occupation are closely related. The manner of holding tools and utensils may determine the location of callouses or injuries on the hands. Inadvertent mannerisms, such as scratching, nail biting, biting of the cuticle will determine corresponding injuries. Hysterical and designing individuals inflict upon themselves injuries by divers methods and means.

Section H. Environment.

Inmates of institutions, children at schools, people living crowdedly in filth, are exposed to infections caused both by animal and vegetable parasites. Geographical considerations, too, should be included under this heading. Thus, piedra is a fungus disease of the South (in the region of Panama); verruca Peruana is found only in a certain district of the Andes; Oriental boil is peculiar to countries on the Northeastern and Southern shores of the Mediterranean and Southern Asia.

Section I. Race.

Negroes are peculiarly predisposed to keloids. Madura foot, caused by an organism closely related to the ray-fungus, is more frequent among natives of India and Africa than among aliens.

Section J. Age.

Certain types of ring-worm, molluscum contagiosum and verruca plana are rare, except in childhood. A characteristic type of wart known as senile hyperkeratosis is restricted to the period of life its name suggests.

Section K. Sex.

The two sexes present about the same series of dermatoses. Such variations as the lesions may show are due rather to anatomical peculiarities than to any inherent sex differences.

Race, age and sex are minor considerations in their etiological relation to pure dermatoses, as compared with the rest of the foregoing table. Study of this table will also show a high degree of interdependence among the various factors. For instance, diseases of occupation (Section F) depend directly upon their causation for some chemical agents (Section B) and some physical agents (Section A) and infectious agents (Section D). Diseases are due to environment (Section H) only in a passive and subsidiary sense, for it is due to the environment that the infectious or other active agents (Sections C, D and E) gain access to their host. It is important, too, to bear in mind the fact that not every worker exposed to given chemical, physical, or other pathogenic influences, nor every child exposed to ring-worm or pediculi, becomes diseased. As already stated (Chapter I), the patient must be receptive to the disease which the pathogenic agent is capable of producing. Whether this predisposition is due to race, age, sex, occupation, environment, individual peculiarity, or a combination of these makes no difference, and in so far as this is true there are very few pure skin diseases. For practical purposes, however, it is simpler to accept the existence of such a group, although by no means in the restricted and isolated sense it has heretofore possessed.

CHAPTER III

SKIN DISEASES WHICH ARE NOT AUTOCHTHONOUS. RELATION OF DERMATOLOGY TO INTERNAL MEDICINE

Although, as has been indicated, the line of demarcation is no sharp one between skin diseases which may for convenience be termed local, and those which in no sense may be so termed, the relationship of the second group to internal medicine is not entirely clean-cut. Psoriasis, for example, lichen planus, prurigo, pemphigus and dermatitis herpetiformis are more emphatically characterized by their cutaneous manifestations than by any general symptoms they cause, or with which they are associated. It is nevertheless impossible to be familiar with these diseases and many others without becoming convinced that the part the skin plays in them, though conspicuous, is incidental, and that their causation must be sought elsewhere than in the integument. It is possible that any or all of these diseases may be due either to specific skin parasites, operating locally, to a specific internal infection which causes definite cutaneous changes, or to some metabolic or biochemic disturbance giving rise to characteristic alterations in the skin. Even admitting the likelihood of a specific local infection in these diseases, their course is such as to suggest that some internal disturbance is required to fertilize the soil for the local agent, in which case the local factor after all becomes the incidental one.

There are, indeed, not many ways in which the skin may be pathologically altered, so that many processes which are widely separated as to their origin may be characterized by like or closely allied cutaneous manifestations. Both urticaria and dermatitis aptly illustrate this. The wheals of urticaria are all alike. The causes of urticaria are numerous. The flesh of fish, crustacea, beef, molluscs, egg albumin, strawberries, alien serum, and many other substances, known and unknown, in receptive individuals cause urticaria. The disease is simple to recognize, but its cause in given cases is usually difficult to ascertain. Acute and chronic dermatitis, a vesicular and scaling disease, may be provoked by rhus, primroses, bichloride, dye-stuffs, vanilla, essential oils, wood oils, pollens, many varieties of chemicals, and by a host of internal causes. Nor can dermatitis be clinically or histologically differentiated from eczema. Here again the problem is to discover the ultimate cause of the disease, that is,

to find out what the interrelationship is between the skin and the rest of the body.

Skin lesions primarily or secondarily due to an internal cause are called cutaneous reactions. Thus, the urticarial wheal; the vesicle of acute eczema or dermatitis; the erythematous blotches caused by copaiba, antipyrin or phenacetin, ptomaines, bacteria or their toxins; the scarlatiniform rash of scarlet fever, or belladonna poisoning; the herpetic vesicles of true herpes, or associated with arsenic poisoning or grippe — all respectively are examples of types of cutaneous reactions, similar in appearance but due to a wide variety of causes.

Diseases of the skin which are not local are either those in which the skin participates in a systemic disease, or those in which a disease, beginning in the skin, becomes systemic. The only real difference between the two is one of sequence, for in the second group the skin is the starting point of the malady, instead of being merely a participant. Extensive burns and infectious diseases such as erysipelas, syphilis, glanders and anthrax are illustrations. It must by now be clear that the physiologic and metabolic balance of the human organism is shared to no small degree by the skin, and that the part played by internal medicine in dermatology is large. But this interrelationship is no simple one to trace. A classification of dermatoses from this standpoint is difficult to compose, and roughly parallels that in the preceding chapters. The following list though not complete, suggests by common examples the scope of the field under discussion.

GROUP I. CONGENITAL AND HEREDITARY DISEASES

Section A.	Congenital Diseases (Not Hereditary)	Syphilis. Icterus neonatorum.
Section B.	Cogenital Diseases (Hereditary)	Some types of hemophilia.
Section C.	Hereditary or Family Diseases. (Not Congenital)	Xeroderma pigmentosum. Hemophilia.

GROUP II. ACQUIRED DISEASES

Section A. Chemicals and Drugs.

1. Erythemas	Belladonna. Copaiba. Many of the coal tar products. Arsenic.

2. Vesicles and Bullae	Arsenic. Bromides. Iodides.
3. Pustules	Iodides. Bromides.
4. Wheals	Quinin, the balsams, sera, antitoxins, vaccines.
5. Granulomata	Iodides. Bromides.
6. Pigmentation	Arsenic.
7. Epithelial proliferation	Arsenic.

Section B. Vegetable Parasites.

1. Bacteria	Lepra, Lupus vulgaris and other forms of cutaneous tuberculosis, blennorrhagic granuloma, glanders anthrax, typhoid roseola, typhus roseola, various eruptions associated with erythema nodosum and sepsis.
2. Bacterial Toxins	Toxi-tuberculides, toxins of streptococci and staphylococci in sepsis, accompanying angina of streptococcus origin causing some varieties of multiform erythema, and allied diseases.
3. Fungi.	Actinomycosis, blastomycosis, sporotrichosis.

Section C. Animal Parasites.

1. Protozoa or Organisms closely allied.	Syphilis. Yaws.
2. Vermes	Urticaria accompanying echinococcus and other parasitic infections.

Section D. Undetermined Infections — The Acute Exanthemata:

Scarlet, measles, chicken and small pox, German measles, Duke's disease; probably some forms of erythema multiforme, and possibly pityriasis rosea.

Section E. Toxins — Anaphylatoxins.

1. Anaphylatoxins	Urticaria and probably some forms of dermatitis; prurigo.

2. Toxins expressing faulty metabolism.

a. Disturbed nitrogen metabolism — Some dermatitis, dermatitis herpetiformis, and Ritters' dermatitis; forms of infantile dermatitis; Leiner's erythroderma desquamativa congenita.

b. Disturbed sugar metabolism — Diabetic dermatitis, carbuncle, furunculosis, gangrene. Hyperglycemia, diseases of the pilosebaceous system, sycosis, rosacea, furunculosis, acne, seborrhoea and seborrhoeal dermatitis, some forms of infantile dermatitis.

c. Disturbed fat and cholesterin metabolism — Infantile dermatitis, xanthoma tuberosum.

3. Gastro-intestinal indigestion.

a. Gastric hyperacidity, chronic gastritis, athylia gastrica, etc. — Acne, rosacea, seborrhoea, closely allied to disturbed sugar metabolism.

b. Carbohydrate fermentation. See Section E. 3, a.

c. Disturbed pancreatic and liver function. See Section E. 3, a.

d. Proteid putrifaction associated with disturbed nitrogen metabolism, or changed permeability of gut, allowing of absorption of anaphylatoxins or other toxins. See Section E., 3, a.

4. Renal disturbance, part of disturbed nitrogen or sugar metabolism.

Section F. Disturbances of Internal Secretion.

1. Thyroid. — Skin changes in cretinism, Graves' disease; effect of thyroid on sugar metabolism through pancreas and liver. See Section E., 2, b.

2. Adrenals and Chromaffinic System. — Pigmentation, Addison's disease, bronzed diabetes; possibly vitiligo.

3. Pituitary Body.	Skin disturbances in acromegaly, hirsutes and effect of gland upon sugar metabolism. See Section E., 2, b.

Section G. Disturbances of Primary and Secondary Sexual Organs (possibly of internal secretions).

1. Puberty (both sexes).	Acne; in women, hirsutes.
2. Menstruation.	Acne, vaso-motor dermatoses.
3. Pregnancy.	Acne, eczema, pruritis, urticaria, vesicular and bullous dermatoses, fibromatosis, hyper-pigmentation, hirsutes.
4. Menopause.	Dermatitis, rosacea, vaso-motor dermatoses.

Section H. Nervous System.

1. Central.	Gangrene, herpes, malum perforans pedis, pellagra, neurotrophic disturbances of syringomyelia, multiple sclerosis, tabes; traumatic alopecia, decubitus, scleroderma.
2. Peripheral.	Pruritus and herpes; dermatalgia and possibly symmetrical gangrene.

Section I. Visceral Malignant Neoplasms.

Cutaneous metastases, acanthosis nigricans, pigmentations.

Section J. Cachexias and Prolonged Fevers.

Dry skin, sudamina, alopecia, nail distrophies, pigmentations, exfoliating erythrodermas, etc.

Section K. Diseases of the Blood, Lymph and Blood and Lymph making Systems.

Leukemic and pseudo-leukemic infiltrations of the skin, premycotic dermatoses, erythrodermas.

In other chapters of this book the outline just sketched in will be amplified as each disease is discussed. It is the sole object of this chapter to emphasize that dermatology belongs to internal medicine, and that the dermatologist should be less a reservoir of special designations than an internist regarding internal medicine with an

especial view in mind to which he must train his eye. Conversely too, the internist of the future would do well to be more of a dermatologist than his forefathers have been. The belief has flourished that the dermatologist is no physician, and that an internist need not and cannot understand dermatology. Neither is true, although to judge from the past, the latter belief has a better foundation than the former.

CHAPTER IV

GENERAL CONSIDERATIONS CONCERNING CUTANEOUS LESIONS

A skin disease is characterized by the appearance upon the integument of some visible or palpable variation from the normal. The normal aspect of the skin among the different races of man is too well-known to demand description. Pathological alterations are called cutaneous lesions, and the lesions are either level with the surface of the skin, depressed or raised. They are further characterized by their color, shape, size, consistency, grouping and distribution. Fresh lesions have certain definite attributes which are subsequently modified either in the course of their evolution or involution, or altered by traumata such as scratching, rubbing and the application of medicaments. Fresh lesions are called primary and all the others secondary.[1] It is by a study of the former in most cases that we must learn to recognize dermatoses, for the former tend to be characteristic and specific, while the latter are generic in type. The presence upon the skin of lesions is called an eruption, outbreak or rash. All of these terms date back to a time in the history of medicine when it was thought that poisons within the body, in their effort to escape, attacked the skin before leaving. The primary or elementary lesions of the skin are the macule, papule, tubercle, tumor, vesicle, bleb, pustule and wheal.

Macule means spot, stain or blotch, and the macule is either flush with the skin surface or very slightly depressed, but varies in size from bare perceptibility to any dimensions. Its color may be white, pink, red, buff, brown or bluish, and its border may be sharp or blurred. Its shape may be circular, oval, elliptical or irregular, and finally macules may remain discrete or become confluent. The tiniest macules are found among petechiae in sepsis and the hemorrhagic diseases, the largest among the flat nevi. Examples of the white macules are found in vitiligo, pink, in the rose spots of typhoid and syphilis, red, in syphilis, and the toxic eruptions; buff, in pityriasis versicolor, and in syphilis in seborrhoeal individuals. Brown macules are seen in freckles, chloasma and nevi; bluish, in

[1] These terms must not be confused with the primary and secondary lesions of syphilis.

certain nevi, and the maculae ceruleae in pediculosis corporis. The only examples of depressed macules are found in some of the atrophies.

Papules are small cutaneous elevations varying in magnitude from the minutest visible size to about the dimensions of a coffee bean. They may be either curved, conical or have vertical sides, and in outline they are either circular, elliptical or polygonal. They, too, may vary in shade from pink to deep red, bluish to violaceous, and light yellow to deep brown, or even slate colored or black. They are either single, grouped or confluent, and rest upon a non-inflamed or inflamed base. Examples of conical papules are those of lichen acuminatus; of curved, lenticular syphilitic papules; of steep papules, those of lichen planus. Circular papules are found in syphilis, psoriasis and prurigo; elliptical ones in pityriasis rosea; and polygonal types in lichen planus. The coloring and grouping will be further discussed in the descriptions of the various diseases. Included among the papules should be a larger variety called the tubercle, its maximum diameter being about two centimeters. Small neoplasms such as nevi, epitheliomata and gummata and the lesions of erythema nodosum furnish illustrations of this lesion. Node and nodule are synonyms of tubercle, and it must further be remembered that when used in this sense the word tubercle has no connection with tuberculosis. A further distinction between papules and tubercles is based upon the level of the skin in which they lie. Papules tend to originate in the upper levels of the corium and epidermis, while tubercles originate in the lower levels of the corium and sub-cutaneous tissue, growing thence upwards. Both papules and tubercles vary in consistency as well as in the respects already mentioned. They may be soft as in fibromata, or hard as in gummata, sarcomata and non-ulcerated syphilitic primary lesions.

Skin tumors are solid elevations the dimensions of which vary from those of a tubercle to the size of oranges, and occasionally skin tumors, such as lipomata, reach huge proportions. For the most part tumors are of neoplastic origin, but large ecchymoses, gummata and leprides reach such dimensions as to entitle them to be classed as tumors. The color, consistency, shape and number of tumors is determined by the malady of which they are the feature. A last attribute of these lesions to be mentioned is their mode of connection with the skin. If their base is not constricted tumors are spoken of as sessile. If their base is constricted, they are spoken of as pedunculated.

Vesicles are collections of fluid in the epidermis, or between the epidermis and papillary body, sometimes invading even the latter, and they almost invariably indicate an exudative inflammation. Excepting for vesicles formed by an accumulation of sweat in the mouths of occluded sudorific ducts, the contents consist of serum or serum and blood. In a like manner all vesicles are of inflammatory origin except some sweat vesicles and the lymph vesicles found in a rare disease called lymphangioma circumscriptum, which may be a neoplasm or nevus. For the most part vesicles rise above the surface of the skin, but when the epidermis forming their roof is thick, as on the palms and soles, they remain level. Elevated vesicles are conical, curved or hemispherical and their greatest size is that of a coffee or kidney bean. They are either tense or flaccid and their color is determined by that of their contents. Sweat vesicles are crystalline in appearance resembling dew drops. Vesicles containing clear serum are straw-colored; those filled with serum and blood may be pale pink or deep brown, or any shade between these two, subject to the relative proportions of serum and blood. Vesicles are single or grouped and may remain discrete or coalesce. Dermatitis, dermatitis herpetiformis, herpes, miliaria crystallina are vesicular diseases.

Bullae or blebs are to vesicles what tumors are to papules or nodes. A bulla is an accumulation of fluid varying in size from a lima bean to almost any dimensions. Their color, consistency, grouping and site are determined in a manner identical with those of vesicles. Their contents are invariably either serum or serum mixed with blood.

Pustules are collections of pus in or just below the epidermis. They are caused by a secondary infection of vesicles or bullae, or they arise spontaneously as in impetigo contagiosa. Serum and blood may be admixed with the pus. Pustules are white, yellow or greenish according to the nature of the process and the variety of the exciting organism. These colors are capable of further modification by the presence or absence of serum and blood, and the relative quantities of these fluids. Some pustules are sterile as is usually the case in pustular syphilides. Variola, impetigo contagiosa, sycosis and glanders are some examples of pustular diseases. Pustules mimic both vesicles and bullae in shape, size, consistency and grouping.

Wheals are caused by spasm in the cutaneous vessels effecting a temporary stasis of blood. When the spasm is overcome, the wheal gradually vanishes. Thus, wheals are transitory. In color they

are dead white, pink, or red. Their shape varies and they may be as large as a hand or even greater in dimension, as in giant urticaria. Fresh mosquito bites furnish good examples of the usual type of this lesion. In fading, wheals sometimes leave a pink macule and rarely a hyper-pigmentation.

Secondary or consecutive lesions are the scale, crust, excoriation, rhagade, ulcer, cicatrix and lichenification. In a sense some of the primary lesions may be secondary. A papule, for example, often is the later stage of a macule; a vesicle, the later stage of a papule; a bleb, or pustule, of a vesicle; or all of these types of lesions may be present at the same time, a condition observed in dermatitis and dermatitis herpetiformis. These are not, however, consecutive lesions in the strictest sense of the term as it is usually employed.

Scales are a mass of exfoliated epidermis engendered by a pathological process in the skin. They are made up of cells of the stratum corneum, and surmount macules and papules as in the toxic dermatoses or psoriasis; or, they are formed in the involuting stages of vesicular and bullous diseases, such as dermatitis and pemphigus. Finally, they must be present in cutaneous anomalies such as ichthyosis. They may be adherent to the epidermis below, requiring relatively great force to secure their removal, or they may come off with great ease. They are either very fine and small, or large and thick. Some are veritable casts of parts from which they spring, a condition at times seen in the exfoliation of the hands and feet after scarlatina. They are either dry or greasy, white, yellowish or brownish, and occur in solid flakes or in flakes resembling pie crust. Very fine scaling is described as branny or furfuraceous. Scales may be few in number and sparsely shed, or very numerous falling off in powdery showers, or in great flakes, or even sheets.

Crusts result from the drying of skin exudations. They cover surfaces denuded of epidermis, as in dermatitis, impetigo or burns; they surmount ulcers, fissures, excoriations, scratched papules, vesicles or bullae. They are either adherent or easily removable. Their size, shape and grouping are determined by these characteristics in the underlying lesions. They tend to be slightly convex and consist of a single layer, or of numerous layers suggesting the appearance of an oyster shell. They consist of dried serum or blood, or both, intermingled with scales and at times hairs. Extraneous matter may be included, such as dirt, bacteria and grease. Their color depends upon their main constituent. If serum alone is present they are

as yellow as honey; if blood alone makes up the crust, the color is brown. Should blood and serum both be represented, the color will be determined by the relative proportion of these substances. Dirt and bacteria impart to the crusts a greenish, grayish or muddy appearance. Crusts are hard or soft, brittle, powdery or tough.

Excoriations are solutions in the continuity of the epidermis, produced by trauma. The trauma may be involuntary — the scrape of a pin, the rubbing of a garment, etc., but usually it is voluntary and purposeful, namely, to relieve itching. Insect bites and pruriginous dermatoses are relieved by such trauma in the form of scratching. The character of the excoriation varies. Should papules or vesicles be scratched, the excoriation is usually punctate and corresponds to the apex of the lesion. Should extensive surfaces be scratched, the excoriations are usually linear and often parallel. At times the scratching is so severe and frequent as, of itself, to be a source of irritation, and the excoriations may serve to mask the characteristic lesions of the disease, as in scabies, and pediculosis corporis. In areas of the body inaccessible to the hands, excoriations are often produced with implements, the character of which will determine the type of the excoriations. These may be punctate, striated or irregular in outline and depth, sometimes amounting to actual tears. Deep excoriations leave scars and are often followed by hyperpigmentation.

Rhagades are solutions in the continuity of the epidermis and immediately subjacent skin, occurring in areas of infiltration, and usually at points at which the integument is subjected to increased tension as over extensor surfaces of joints, or subjected to folding as over flexor surfaces, or near body folds. They are also frequently found at muco-cutaneous junctions. They are linear, and their length is determined partly by the size of the infiltrated area in which they occur, and partly by their anatomical site. A rhagade under the breast may be as long as the junction between the breast and adjacent thorax. At the corner of the lips rhagades are as long as the depth of tissue lying beneath the skin without and the mucous membrane within. These lesions are also called fissures. They are found in dermatitis, callous, in connection with mucous patches, and in general with all infiltrations. They predispose to ulcerations and at times become portals of entrance for infecting organisms leading to local infections, cellulitis, or even sepsis.

Ulcers are solutions in the continuity of the skin involving the epidermis and the tissues below to any depth. The papillary body

alone may be involved, or the cellular tissue may be included, as well as fascia and even muscles. An ulcer implies necrosis, and the process may have many causes. Ulcers vary in shape, size, the character of their margins and their floors, and as to the nature of their discharge. They may be caused mechanically by pressure or cutting; chemically, by caustics; thermally, by freezing or burning, and by actinic rays. Diseases of the central nervous system, cachexias and wasting fevers causing trophoneuroses lead to ulceration, whether in the form of gangrene or bed sores or pressure sores. Infections by streptococci, spirochaetes, lepra, tubercle bacilli, Klebs-Loeffler bacilli, etc., lead to ulceration, as do various types of neoplasms.

The shapes which ulcers may assume are circular, elliptical, circinate and irregular. Circular and elliptical ulcers are in the main those of mechanical or chronic infectious origin. Circinate and irregular ulcers are caused by confluence of the first two types, and by infectious and chemical, actinic and thermal agents. The margins of ulcers may be either vertical or at an angle to the base causing the opening of the ulcer to be larger than the floor, or smaller than the floor. If the opening is larger, the ulcer is cup or basin shaped; if smaller, the margins are called undermined. Vertical or basin-shaped ulcers are found in non-infectious granulomata, neoplasms and those of chemical and thermal origin. Syphilis, too, may cause ulcers of this type. Nearly all ulcers with undermined edges are due to pyogenic organisms, the tubercle or Ducrey bacillus, central nervous disorders or gangrenes. The base of an ulcer discharges either serum, blood or pus, according to the etiology of the lesion, and the discharge is often foul. It tends to form crusts upon the removal of which, the presence or absence of granulation tissue in the floor, and the nature of the margins may be ascertained. Gradually progressive ulcers are called phagedenic.

Cicatrices or scars are the result of healing when tissues deeper than the epidermis have been destroyed. They consist of fibrous connective tissue in which the hairs, glands and muscles of the skin do not regenerate. Nerves, however, and elastic tissue do reappear after months or years, and gradually the papillary body is formed anew. Young scars consist of organizing or organized granulation tissue; old scars of fixed connective tissue cells with fully developed connective tissue bundles. Young scars are abnormally vascular since scar tissue is the product of granulation tissue. The shape and size of scars depends upon the lesion of which they are the

result. Scars are either flush with the skin, atrophied or hypertrophied, according to the wealth of connective tissue produced. The last type is spoken of as a keloid or hypertrophied scar, but has nothing to do with true keloid which is a fibroma. The color of the scar depends upon its vascularity and pigmentation. Fresh scars are invariably reddish or bluish, fading in the course of time to white or flesh tones. If excessive pigment is formed, scars may be yellowish or brown. For the most part scars are painless, but total analgesia or hyperalgesia, though rare, are occasionally found. Large scars tend to contract, particularly those caused by burns or caustics, and if they are situated over joints may produce deformities, or if situated elsewhere cosmetic defects.

Lichenification is a thickening of the skin caused by an increase in depth of the epidermis, and an infiltration in the papillary body and sub-papillary stratum. Clinically, it is characterized by an elevation of the skin, which is thick, hard and criss-crossed by exaggerated cutaneous markings. It is the result either of scratching or rubbing in pruriginous diseases, such as scabies, dermatitis, prurigo, essential pruritis and senile itching dermatoses. Lichenified areas vary greatly in shape, size and outline.

The color of skin lesions varies as already stated. The surfaces may be glistening, waxy, or dull, subject to the tension and character of the lesions. These are occasional alterations which will be described in connection with the diseases in which they occur. White lesions are rare excepting in exfoliative diseases, the scales of which are white. They usually result from loss of pigment. White spot disease, morphea, some of the idiopathic atrophies, syphilitic leucoderma, vitiligo, the peculiar whitish blue of argyria, and depigmented scars are examples. Psoriasis scales have a peculiar silvery tinge. Pink and red lesions are usually inflammatory, excepting in nevi, and red tattooing. Yellow and orange lesions are found in xanthoma, and the former in some of the papules in lupus vulgaris, and in other tubercular lesions, vesicles, pustules and in some crusts. Favus scutula are sulphur colored and the crusts of impetigo and other serous exudations are golden. Buff characterizes many of the chronic dermatoses, as seborrhoea, some of the lichens, hyper-pigmentations and pityriasis versicolor. Brown lesions of all shades are due either to melanin or hemoglobin derivatives according to whether the disease is one of hyper-pigmentation, or due to hemorrhages. Nevi, melanomata and black tattooing furnish examples of the blue, slate and black lesions, while cyanoses and

the blue lesions in body lice are examples of the various bluish tones. The later stages of lichen planus are violaceous, as are some nevi, and the latter may also be deep purple.

The shape and configuration of lesions has already been partly touched upon. The simpler forms need no further mention. The following terms, however, circinate, gyrate, geographical and serpiginous must be amplified. Circinate and gyrate lesions are formed usually by the coalescence of ringed and oval types, the peripheries of which, in merging, form a festooned outline. Gyrate lesions are formed by the merging of segments of circles of various sizes, differing from the circinate only in the fact that the latter is made up of coalesced lesions of similar size. Serpiginous is a term often applied to ulcerated lesions tending to increase by the progress of a sinuous convex margin. This imparts to the lesions a fan or kidney shaped outline. Geographical lesions are extensive varieties, the shape and contour of which suggest the outlines seen in maps.

Grouping. The shape and size of lesions have already been described. Their grouping and distribution come under the following general heads — localized, disseminated and generalized, symmetrical and asymmetrical. These terms are self-explanatory, which is not the case however of the special designations, herpetic and corymbose or corymbiform. Herpetic means resembling herpes. Herpes is a disease characterized by a cluster of vesicles in round, oval or slightly irregular patches of a diameter of from one half to two inches. Corymbose is a term applied to groups of lesions of which the central one is large and the surrounding ones small, a condition seen in syphilis when a large papule is surrounded by numerous small ones forming a group in which the central papule is the dominant feature.

A few words as to the consistency of skin lesions will conclude this chapter. They may be soft or hard. Soft lesions may be flabby, boggy or doughy; soft fibromata are examples of these lesions. Half emptied blebs or large pustules, cysts or abscesses are boggy or doughy. Hard lesions are elastic, brawny, stony or cartilaginous. Tense vesicles, pustules, blebs, cysts, etc., are elastic. Oedema may also be elastic or hard, when it fails to pit on pressure. Brawny is a term applied to the hard, inflammatory oedema seen in erysipelas and cellulitis. Stony hardness is observed in some fibromata, rhinoscleroma, keloids and hypertrophic scars. Cartilaginous consistency is found almost solely in the primary lesions of syphilis.

CHAPTER V

METHODS OF EXAMINATION AND DIAGNOSTIC PROCEDURES

The examination of dermatoses should be conducted in evenly distributed, soft, bright daylight. The patient should be stripped and the localization of the eruption, the character of the lesions, their color, distribution and consistency should be studied. Occasionally, there is objection to stripping, but all but very few patients finally acquiesce when they are made to realize that skin diseases frequently present different pictures on different parts of the body, and that intelligent and conscientious conclusions cannot be reached without a comprehensive examination. All patients, particularly the prudish and those of unduly fine sensibilities, must be treated with the utmost tact. In general, less difficulty is encountered in private practice than in public clinics, for unwholesome modesty appears to be an attribute of the ignorant. After carefully looking at the lesions, palpation will determine their consistency, depth, the question of whether they are adherent to deeper parts, the presence of fluctuation, hyperemia, hemorrhages and sensitiveness. By scratching with the finger nail the character and adhesiveness of crusts and scales may be determined. It is more hygienic and infinitely safer to palpate with a finger protected by a finger cot, and to scratch with a curette, for the danger of acquiring syphilis must never be forgotten in dermatological practice. Unna's diascope, a piece of thick clear glass, may often be employed to advantage. It is used to exert pressure over lesions and render them ischaemic. Thereupon the lesion, as seen through the glass, can be studied without the confusing presence of blood in the dilated vessels.

At the conclusion of the examination a detailed history should be taken. The illness for which the patient presents himself should first be taken up, beginning with the time and manner of its onset, the subjective symptoms, their development, and including any general symptoms which may have arisen. Then the clinical life history should be investigated with reference to habits, occupation, mannerisms, appetite, diet, gastro-intestinal, genito-urinary, cardio-vascular, respiratory, nervous, venereal and any other diseases.

Changes in weight should be inquired about, as well as sleep, fatigue, working capacity and the psychic state. In many dermatoses it is of extreme significance to know whether drugs have been employed externally or internally, and whether cosmetics have been used. The family history should next be investigated. When this has all been done, the dermatosis should once more be examined, and a general physical examination should be made as carefully and thoroughly as possible. If necessary, other examinations by qualified specialists should be made. To attempt a diagnosis in dermatology in any other manner is careless and inexcusable. Those who say that a diagnosis in this field should be made by the eye alone are not practicing scientific medicine, but are relying on a species of cleverness which inevitably must often lead them astray.

Besides the clinical methods of study just outlined, there are others often of great worth in clearing up obscure cases, or corroborating hypotheses. These are the laboratory methods. The lesions themselves may be studied histologically, or scrapings or secretions from lesions may be examined under the microscope, or by various bacteriologic procedures. Examinations of the blood, cerebro-spinal fluid, sputum, gastric contents, feces and urine, often aid in diagnosis, as do also some special tests.

The value of the microscopic study of skin lesions is great. It often removes obstacles in diagnosis, and still more often enables the dermatologist properly to classify cases. Comparatively few dermatoses, it is true, possess a characteristic histologic structure, but enough do to make the help of the microscope distinctly valuable. Much may be gained, and certainly nothing lost, by invoking this aid in doubtful cases. The microscopic study of scrapings from lesions, affected nails, scales, fragments of hair, is done to determine the presence of animal parasites, bacteria or fungi. Secretions are studied for the same purpose, particularly to establish the diagnosis of primary syphilis by demonstrating spirochaetes. These facts will receive ample discussion in subsequent chapters.

The detailed bacteriological study of dermatoses embraces all the methods employed in this field. The presence of micro-organisms in the lesions may be ascertained by suitable stains for spirochaetes, lepra bacilli, and other bacteria and fungi, as well as for Leischmann bodies in Delhi boil. The inoculation of guinea pigs in cutaneous tuberculosis is often of great value. The skin lesions of this disease are frequently poor in tubercle bacilli, and animal inoculation with suspected tissue may facilitate a diagnosis when other methods fail.

With increasing precision of technique a similar method will probably prove valuable in syphilis. The testes of rabbits are peculiarly vulnerable to spirochaetes, and suspected tissue or secretions injected into these organs frequently cause the local development of a syphilitic granuloma. Cultivation, on suitable media, of microorganisms from scrapings, secretion and scales of lesions and from suspected hairs and nails, frequently aids in diagnosis. Sabouraud has devised a special medium for the cultivation of ring-worm organisms and allied fungi. The various pus producing organisms luxuriate on ordinary media.

An examination of the blood includes the ordinary hemoglobin and cytological estimations; blood cultures, complement fixation tests and chemical analyses. The blood count may reveal an anemia, leukemia, or some other disease or disturbance, the successful regulation of which will favorably affect dermatoses. In addition, several dermatoses influence the blood picture. Erythema nodosum, for example, is often associated with secondary anemia. Syphilis, too, causes anemia and a leukopenia with relative lymphocytosis. Toxic dermatoses are often accompanied by a mild leukocytosis, with an increase of the polymorphonuclear leukocytes. In erysipelas this is particularly the case. Eosinophilia is common in itching diseases, particularly in scabies, prurigo, pemphigus and dermatitis herpetiformis. Blood cultures have not been found to be of great value in dermatology, excepting in dermatoses due to sepsis. Complement fixation tests have been employed in syphilis, blenorrhagic granuloma and ring-worm. Of these, only the first, the Wassermann test, is of great practical value. The gonococcus complement fixation test of Hans Schwartz may be of use in the recognition of blenorrhagic dermatoses. Work has been done indicating the presence of the Bordet-Gengou reaction in ring-worm, which although of great scientific interest, is superfluous as a diagnostic procedure, for the condition is easily recognized by simpler means. Chemical studies of the blood may be performed with reference to sugar, cholesterin and nitrogen metabolism. Their purpose is to determine the etiology of skin diseases, rather than to simplify diagnosis. An increase of blood sugar has been found in pilosebaceous diseases. A cholesterinemia is present in xanthoma tuberosum. Very little work upon the nitrogen content of the blood has been done in dermatology. Excepting in syphilis, analysis of the cerebro-spinal fluid has no value.

Examination of the sputum for tubercle bacilli is often of great

value in corroborating a diagnosis of cutaneous tuberculosis. The organisms causing Vincent's angina and syphilis may also be found in this secretion, but are more easily demonstrable in scrapings from suspected lesions. Examining secretions from the rhinopharynx for Hansen bacilli is occasionally of diagnostic value in suspected lepra.

Studies of the gastric contents and feces will often show the existence of some type of digestive disturbance, which may be the basis of given dermatoses such as acne, seborrhoea, rosacea, urticaria or dermatitis. Qualitative and quantitative urinalyses may be of similar service, particularly in diabetes which often provokes dermatitis and furunculosis. A disturbed nitrogen metabolism, ascertained by quantitative urinalysis, may be the determining factor in psoriasis, prurigo, dermatitis and dermatitis herpetiformis.

The special tests alluded to are the various tuberculin tests, the luetin reaction and a percutaneous test in the sense of Pirquet for ring-worm and allied diseases. The tuberculin tests are performed by the hypodermatic, percutaneous and cutaneous application of tuberculin. The first method was discovered by von Pirquet and bears his name; the cutaneous test was devised by Moro. The luetin test, based roughly on Pirquet's technique, was devised by Noguchi, and a similar test for trichophytosis was first employed by Bruno Bloch of Basel. All of these tests will be fully discussed in their proper places.

It is apparent from these paragraphs that the analysis of a dermatological case involves all of the principles of internal medical examination. A good history is essential. A careful inspection of the skin, as well as a general physical examination of the patient, must be made. Proper laboratory work must be carried out. The dermatologist should not only be a clinician, but also a histo and clinical pathologist, bacteriologist and chemist, in the sense that he must know how to interpret the findings in these special fields of medicine.

SECTION B. PURE DERMATOSES

GROUP I. CUTANEOUS REACTIONS DUE ESSENTIALLY TO VASCULAR DISTURBANCES

CHAPTER VI

THE ERYTHEMAS AND URTICARIA

The important part played by the cutaneous vessels is the common and striking feature of this group of diseases, nor is it anatomical vascular changes so much as mechanical ones with which we have to deal. Vasodilatation, primarily, or after spastic constriction, is the starting point of these dermatoses. The causes of such vascular instability are numerous and may be arranged under four heads — organic, physical, reflex nervous (so called), and toxic. The organic causes are mainly chronic cardiovascular, respiratory diseases and personal peculiarities causing erythemas and cyanoses, due to changes in the calibre and tone of vessels. Blue fingers and ears, blushing and flushing are examples. These are scarcely cutaneous lesions but are significantly related to such. Physical causes are pressure, heat, light and cold. Reflex nervous causes are exciting psychic disturbances and the neuropathic states associated with dentition, menstruation, gestation and the menopause, while the toxic causes include ingested toxins and those due to metabolic derangements. The former are introduced into the body as food or medicines; the latter are manufactured in the body as the result of indigestion, disturbances of internal secretions controlling digestion or elimination, biochemic changes connected with nephritis, diabetes and the cachexias. Even the reflex nervous causes may be due to the conditions just mentioned influencing the nervous system, rather than to the nerves, per se. Thus, a reflex disturbance during the menopause may not be functional so much as due to a possible endocrine disturbance or a toxin elaborated during this period.

The common symptoms of this group of skin diseases are itching, and the presence of red macules. The macules may remain as such, or develop into papules, nodules, ulcers, vesicles, bullae, or leave hyperpigmentation. The diseases may be acute or chronic, transitory or persistent, but they are all emphasized at the beginning by redness of the skin, excepting urticaria which starts with white lesions quite as often as with red.

The erythemas occur in three important anatomical forms, the

diffuse, macular and nodular, represented respectively by the important conditions known as erythema scarlatiniforme, erythema exudativum multiforme, and erythema nodosum.

ERYTHEMA SCARLATINIFORME

Synonyms. Scarlatinoid Erythema; Desquamative Scarlatinoid Erythema; Scarlatinoïde; Erythema Punctatum; Roseola Scarlatiniforme; Dermatitis Exfoliativa Acuta; Erythema Scarlatiniforme Desquamativa Recidivans; French; Erythème Infectïeux.

Definition. Scarlatiniform Erythema is an acute or subacute disease of the skin characterized by a generalized punctate scarlet eruption, which runs its course in from a few days to three months and which is prone to recur. It is usually accompanied by mild systemic disturbances.

Symptoms. The acute form has a sudden onset accompanied by malaise, prostration and a rise in temperature which occasionally reaches 104° F. The rash appears within a few hours, may remain localized, but usually involves the whole body surface with punctate scarlet macules. The pharynx is uniformly red, but the throat and tongue do not resemble those of scarlatina. Within a few days the temperature is again normal and a fine furfuraceous scaling takes place. There are no complications or sequelae.

The subacute form also begins suddenly in a manner similar to the acute, but it runs a longer course lasting from two to three months. The onset is at times accompanied by gastro-intestinal disturbances. The character of the rash resembles that of the acute form at first, but the redness persists and the desquamation is coarse, the scales having a diameter of from 1½ to 5 cm. The pharynx and conjunctivae are reddened. Török mentions the occasional presence of lymph adenopathies. Recurrences are frequent. In both forms itching, either mild or severe, is present.

Etiology and Pathogenesis. The etiology is not clearly understood. The disease may be idiopathic or secondary to other diseases or medicaments. Of the diseases, sepsis, abscesses, and toxemias of all sorts are the most important; of the medicaments, serum injections, tuberculin, mercury and salvarsan are to be considered. Arsphenamin needs especial emphasis because of its wide use. A few individuals are either born with, or acquire an intolerance to this substance, one of the manifestations of which is the subacute scarlatinoid erythema, at times attended with alarming systemic manifestations such as suppression of urine.

Differential Diagnosis. Scarlatina, the chronic erythrodermias, universal exfoliative dermatitis, and generalized psoriasis are the diseases from which this one must be distinguished. The most important of these is scarlatina. In the latter the early symptoms are severer, the throat and tongue are characteristic, there is a pale zone about the mouth, the fever curve is typical and there are no recurrences.

Treatment. During the exanthematous stage the patient should be in bed on a fluid or semisolid diet. At the start a saline cathartic is to be given and the bowels throughout the disease are to be kept open by means of cathartics or enemata, and above all by a suitably balanced diet. Should the itching be intense, mild sedatives, chiefly bromides, are indicated, but it must be remembered that in some people these themselves provoke itching. Should an underlying cause be determined, the treatment must be directed towards its elimination. Locally, the indications vary according to the stage of the eruption. At the onset soothing lotions must be used, such as calamine or calamine and zinc, or a menthol and camphor spray several times daily. Should the patient be unable to stand the exposure and chilliness the use of fluids involves, powdering with zinc stearate is useful. Under no conditions should an ointment be used as this may irritate the skin. Occasionally, however, a zinc paste may be employed. During the stage of exfoliation a ten percent. zinc salve or simple emolient cream should be gently applied once or twice a day.

Prognosis. The prognosis is good.

ERYTHEMA MULTIFORME EXUDATIVUM OF HEBRA (FIG. 1)

Definition. Erythema Exudativum is an acute disease characterized by the sudden appearance on the backs of the hands, and insteps, and neck behind the angles of the jaw of vivid red or purplish macules or papules.

Symptoms. The onset of the disease is acute with or without systemic symptoms. An eruption of red, bluish red or purplish macules first appears on the dorsal surfaces of the hands, (Fig. 1) feet and neck behind the jaw. Next the extremities and face are involved, and in some cases the entire body. The macules vary from the size of a lentil to a quarter, and may turn into papules raised about ⅛ inch, which are elastic and resistant to the touch. At the onset there may be fever reaching 103° F., malaise and prostration, and at the same time pharyngitis and arthralgias are often encoun-

tered. The conjunctiva was involved in three quarters of the cases observed by Duhring, fever in thirty percent. of the cases, cervical gland and splenic enlargements in a few. Duhring regards the conjunctivitis, fever, malaise and occasional anorexia as prodromata. At times severe itching or burning are present, and occasionally lesions are found upon the buccal mucosa.

Course. The disease usually lasts from a fortnight to a month. At times there are recrudescences and in many individuals recurrences. Complications are exceptional.

Varieties.

Although the typical form is characterized by macules and papules which remain discrete, there are also cases in which a coalescence of the lesions takes place giving rise to erythema annulare or circinatum and figuratum. At times, too, the form of the lesion is modified and we have erythema papulatum or papulosum, tuberculosum, vesiculosum, bullosum, and urticatum. These designations simply indicate the clinical varieties the disease may assume, but are not separate diseases. The term erythema urticatum denotes the severity of the pruritis.

One variety, however, merits special mention. This is **erythema iris,** also called **herpes iris.** Its localization and course correspond to those of the ordinary form from which it differs only in appearance. This difference is due to two factors. The first is the presence of a central vesicle surmounting a hyperemic base and this base tends to extend peripherally pushing the hyperemic zone ahead. With the peripheral extension central involution takes place. At the periphery a new vesicle forms which is ring-shaped. Thus, a target-like figure of several rings is formed, the outermost in the process of evolution, the inner ones in the course of recession. These concentric rings are the second characteristic point.

Differential Diagnosis. Erythema Multiforme should, but cannot always be distinguished from erythema toxicum and urticaria, to both of which it is closely related, and from pellagra; while the vesicular and bullous varieties must be differentiated from dermatitis herpetiformis and pemphigus. The toxic erythema lesions are paler, do not necessarily occur first on the backs of the hands and feet, and usually remain macular assuming the various figures described in the exudative form. The differentiation from urticaria rests on the absence of wheals, in erythema multiforme and the less intense itching. Its difference from pellagra, dermatitis herpetiformis and pemphigus will be considered with those diseases.

Etiology and Pathogenesis. The etiology is obscure. The disease is found more often in men than women, and mostly in young adults. It seems to favor the Spring and Fall and at times it occurs in epidemics as noted by Duehring. It bears a distinct relation to digestive and metabolic disturbances and to rheumatism, but there is possibly a type due to a specific infection. Hebra first noted intestinal disturbances. Freund found evidences of proteid putrefaction in studies of the urine. Mracek corroborated this by determining the beneficial effects of intestinal antiseptics. It has been found associated with nephritis, diabetes and tuberculosis. Thus, it is either secondary to or associated with visceral diseases, in the sense of Osler, or infectious diseases. Drugs, and particularly sera, may cause lesions of this type. Mackenzie noted its relationship to purpura and rheumatism.

Treatment. The treatment is internal and local. The principal indications for internal therapy are dictated by the possible relationship of the disease to intestinal indigestion and rheumatism. As to the former, vigorous catharsis induced by calomel and salts, and intestinal lavage in the form of daily colon irrigations will suffice. Salol, ichthyol, small doses of phenol, internally administered for purposes of antisepsis are useless. By far the most important factor is diet. Analyses of the gastric contents and stools, particularly the stools, in conjunction with the presence of indol and skatol in the urine, indicating a proteid putrefaction, will serve as guides for prescribing a proper diet. In such cases the use of lactic acid bacilli may be of value. When associated with rheumatism, the treatment of this condition is the one of paramount importance. This involves eliminative treatment and the administration of salicylates, an excellent vehicle for which is rhubarb and soda mixture. The local indications are determined by the pathology of the disease. It is a vascular dilatation probably due to a temporary paresis of the vessel walls, or the first stage of a mild acute exudative inflammation. Calamine or calamine and zinc lotion applied several time daily will answer all requirements. Ointments and pastes are useless. For severe itching the menthol spray and the judicious use of sedatives will prove of great value.

Prognosis. The prognosis is good.

ERYTHEMA NODOSUM (FIG. 2)

Synonyms. Erythema Contusiforme; French, Erythème Noueux.

Definition. Erythema Nodosum is an acute disease character-

ized by systemic symptoms and the sudden development on the shins and forearms of tender, painful, red infiltrations or nodules.

Symptoms. Accompanied by mild prostration and a fever of from 100° to 104° F., the eruption appears suddenly on the shins (Fig. 2). It consists of painful, bluish red nodules varying in size from that of a lentil to a robin's egg. The lesions resemble bruises (Fig. 2); hence the name erythema contusiforme. The extensor surfaces of the forearms toward the ulnar side, and finally the extensor aspects of the thighs and arms, even though only rarely, may be involved. Most commonly the disease remains localized to the shins. The lesions are exquisitely tender and never break down. They disappear without trace.

Course. Within from a fortnight to three months the disease disappears, frequently leaving the patient with a secondary anemia. Recurrences are most exceptional, although the malady is often associated with rheumatism. Occasionally erythema nodosum and erythema multiforme appear together.

Differential Diagnosis. Erythema nodosum must be differentiated from erythema indurativum of Bazin, and syphilitic periosteal gummata involving the skin above and known as erythema nodosum syphiliticum. Bazin's disease is tuberculous in origin, and since erythema nodosum is also widely regarded as associated with tuberculosis, it may readily be seen that a differentiation between the two conditions may prove difficult. Erythema nodosum, however, never ulcerates, or leaves any traces after disappearing. Bazin's disease usually does ulcerate, leaves scars and recurs, and finally is nearly always present in connection with other evidences of tuberculosis. In doubtful cases animal inoculation and histological studies will clear up the question. The simple form of erythema nodosum is distinguished from the syphilitic form by the fact that the latter tends to break down, recurs, and is improved by anti-syphilitic treatment. The Wassermann test is positive.

Etiology and Pathogenesis. Some authors consider it rheumatic and a form of erythema multiforme, while others, notably Hebra and Jadassohn, regard it as a distinct clinical entity, a view which to-day is generally accepted. It is known in many instances to follow measles and malaria, and to be associated with sepsis, bacterial endocarditis and tuberculosis. It is generally regarded as rheumatic in origin, but recently an extensive study of the disease by Foerster indicates at least an intimate association with, if not a causative relation to tuberculosis. This seems true to a larger

extent among children than adults. Rosenow isolated a specific diplo-bacillus from the lesions. It is evident that there are etiologically two groups of the disease — one associated with rheumatism and the other with tuberculosis and measles; and finally, if Rosenow's work is correct, there may be a third specific form. Women seem more prone to the affliction than men, and although it may occur at any age, children and young adults seem to be most susceptible. Bass reported a case associated with orchitis in a 2½ year old boy. The majority of cases occur in the Spring and Fall.

Treatment. The treatment is internal and local. The internal treatment is directed toward the associated condition which is either rheumatism or tuberculosis. The local treatment has two objects — first to relieve the pain and tenderness, second to alleviate the inflammation. Wet dressings fulfil these indications. Aluminium acetate, diluted with ten parts of water and changed every half hour, is excellent. It is wise, in order to facilitate evaporation, not to cover the dressings with rubber tissue or oil silk. The evaporation increases the relief of pain and hastens the reduction of the inflammation. Some patients prefer warm or hot wet dressings. As an adjuvant to the fomentations calamine lotion should be used. In cases followed by secondary anemia, this special indication must be met.

Prognosis. The prognosis is in the main excellent, although in some of the cases apparently associated with tuberculosis, the outlook is poor, but in these cases the prognosis becomes that of the tuberculosis and not of the erythema.

TOXIC ERYTHEMAS

Toxic erythemas are due to toxins ingested or resulting from disturbed metabolism or some general disease. The former are due to drugs or tainted foods; the latter are due to indigestion, infectious diseases, particularly sepsis and bacterial endocarditis. They are characterized by the presence of pink, pale or deep red circular or oval macules, which may coalesce into circinate, gyrate or figured lesions. They are evanescent and recurrences are frequent while the etiological factor persists.

PHYSIOLOGICAL ERYTHEMAS

Two types of erythemas must be mentioned, which though not pathological, must be understood. They are the cutis marmorata and the roseola infantilis.

Cutis Marmorata means marbled skin. When patients are exposed to cold air, some of the superficial vessels dilate, giving to the entire skin the striated appearance seen in marble. This must be borne in mind in examining for the syphilitic roseola. In marbled skin there is a tracery of delicate irregular red lines separating the normally colored patches which are round. In the specific roseola, the lesions are red and round or oval, and divided by a delicate lace-work of normally colored skin.

The *Roseola Infantilis* known in German as the *"Schrei Exanthem"* is seen in babies when the effort of crying causes the cutaneous vessels to distend. This develops on the skin a punctate rash resembling that of scarlatina, measles or German measles. It vanishes when the infant is again quiet.

Other erythemas are *Erythema Hyperemicum* or *Simplex,* which is nothing but a blush and is caused by excitement; *Erythema Caloricum,* caused by extremes of temperature; *Erythema Solare,* caused by Finsen, Roentgen or sun's rays; *Erythema Gangrenosum,* a rare disease starting with erythematous spots and ending with gangrenous nodules; *Erythema Laeve,* the shiny red erythema of the legs in infants with cardio-vascular and renal disease; *Erythema Fugax,* an adjective indicating evanescence; *Erythema ab Igne,* a rare gyrate reticulated disease of the anterior surface of the legs, leading to permanent pigmentation and found in senile, weak or alcoholic individuals exposed to intense heat (firemen, stokers); *Erythema Symptomaticum* seen before acute infectious diseases, or following vaccination, or vascular, cardiac or vasomotor diseases; *Erythema Infantum* of Escherich, an epidemic erysipeloid disease of children from four to ten years old; and *Erythema Elevatum Diutinum,* a rare disease of the extensor surfaces seen in children and young adults and allied to granuloma annulare. Finally, there is *Erythema Intertrigo* or intertrigo, a reddening of the proximate surfaces of skin in body folds, predisposed to by heat, moisture and irritation. This probably belongs to diseases of the dermatitis group.

A rare, though important syndrome in this group, is *Erythema perstans.* In this disease, sometimes fading and again developing, always at precisely the same sites are discoid macules. Their size at times equals that of a nickel or quarter. In color they range from red to purple. They may itch. There may be few or a relatively great number of lesions, and there are two groups; one in which the cause may be ascertained, another in which it cannot be. In the former it is usually drugs that are the cause, notably antipyrin and its allies, and phenolphthalein, as in cases reported by Abramowitz and Howard Fox. In rat bite fever this form of

erythema is strongly suggested. The treatment consists in eliminating the known cause, and otherwise in a regimen similar to that considered effective in other forms of erythema. It is usually fruitless to treat the disease unless its etiology is determined. The prognosis is always good as to general health, and never so as to a cure, unless the etiology is discovered. These factors also decide the course. At times lupus erythematosus disseminatus arises apparently as an erythema perstans; the true nature of the disease revealing itself under prolonged observation.

URTICARIA (FIG. 3)

Synonyms. Nettle rash; German, Nesselsucht, Nesselfieber, Nesselausschlag; French, Urticaire, Ortie.

Definition. Urticaria is an acute disease, usually running a short course, and characterized by the sudden appearance, without definite localization, of peculiar and intensely itching raised lesions, which at first are white and subsequently become red.

Urticaria includes angioneurotic edema, is closely related to prurigo and remotely allied to the so-called urticaria pigmentosa. The characteristic feature of urticaria is the wheal, a raised, white, tense, edematous lesion, the best example of which is a fresh mosquito bite. The wheal is due, according to Unna, to vascular spasm and a serous transudate, which accounts for its whiteness. When the spasm is relieved the vessels dilate and the wheal becomes a pink or red raised lesion, difficult to distinguish from that of erythema multiforme. According to Sczamberg of Prag, it is due to a hypersecretion in the cutaneous lymphatics.

Symptoms. The onset of the disease is sudden. Within a few moments or hours the body is covered with wheals of all sizes from that of a lentil to that of a dollar. They may combine into circinate, gyrate and figured groups, or, extending peripherally, may become annular. The freshest lesions are of a distinctive white resembling porcelain; the older lesions are red. As a rule, a mixture of the two is present by the time a physician is consulted. Often systemic disturbances such as nausea, diarrhoea, headache, malaise and fever accompany the outbreak, or it follows a prolonged period of constipation. The itching is usually furious, causing the patient to be agitated and constantly to rub or scratch the lesions. On stroking the skin a wheal develops, the form of which is linear or curved, according to the direction of the artificial trauma, and it is possible even to make figures or form letters on the skin in this

manner. This phenomenon is known as urticaria factitia or dermographism, and is diagnostic of urticaria. At times the pharyngeal and buccal mucosa are the site of lesions. In fading, the wheals leave either no trace, or pinkish spots that last a few days, or hyperpigmentation if the lesions have been persistent, or the transudate has contained blood. When loose skin, such as that of the eyelids, is involved, actual local edemas arise.

Course. Within a few hours to a week the disease is usually well. There are, however, prolonged cases in which the patient is the victim of a series of such attacks, the itching and annoyance of which interfere with his rest, and thus weaken him as much as a graver disease might.

Varieties.

Many of the varieties are due to accidents of arrangement, as *Urticaria Annularis* and *Gyrata,* or to peculiarities of involution as *Urticaria Evanida* (rapid) or *Urticaria Perstans* (slow). A rare variety is *Urticaria Solitaria,* a single recurrent type described by Vörner. More important are *Urticaria Vesiculosa, Bullosa, Papulosa, Tuberosa* and *Hemorrhagica.* The terms are self-explanatory.

Urticaria Papulosa begins in the ordinary way, but some of the wheals are converted into papules forming a link with prurigo and papular dermatitis.

Urticaria Tuberosa or *Giant Urticaria* is a form of the disease entity known as angioneurotic edema, which will be discussed below.

Urticaria Hemorrhagica depends for its development upon the presence of blood in the exudate and connects urticaria with the purpuras.

Urticaria Vesiculosa and *Bullosa* are varieties in which the exudate is excessive and serum accumulates below the epidermis. The bullae may be as large as a robin's egg. These forms connect urticaria with erythema bullosum, dermatitis herpetiformis and pemphigus.

Differential Diagnosis. The three salient points upon which the diagnosis of urticaria rests are the characteristic wheal, the presence of dermographism and the intense itching. Upon these points it must be distinguished from erythema multiforme as already discussed under that disease; and from dermatitis herpetiformis, pemphigus and papular dermatitis, the differential points of which will be taken up subsequently.

Etiology and Pathogenesis. The causes of urticaria are external and internal. The external ones are irritants, as insect and jelly

fish stings and poisonous vegetables, such as the nettle. Changes in environment and climate also cause urticaria, and the bullous type particularly, develops after a sea voyage to a foreign country. The internal causes are toxic and nervous.

I. Toxic Causes.

A. Foods: Eggs, roe, fish and crustacea, bivalves, dried fruits, flesh of poultry and mammals, particularly pork and sausage, cheese, cereals, peas, beans, lentils, corn, mushrooms, nuts, tea, coffee, cocoa, alcohol, strawberries, cucumbers, onions, garlic, pickles, pastries, honey and sweets.

B. Drugs: Sera, antitoxins, vaccines, quinine, copaiba, sandal wood and other balsams; to a lesser degree, opium and its derivatives, hyoscyamus, chloral, the salicylates and arsenic.

C. Diseases due to Animal Parasites: Plasmodia, intestinal amoebae and worms, and echinococcus cysts.

D. General Diseases: Metabolic, gastro-intestinal and eliminatory disturbances.

E. Sequelae or Complications of other Diseases: Asthma, pemphigus, variola, etc.

II. Nervous Causes.

Kreibich and Sobotka in studying the effect of emotions on vasomotor disturbances concluded that these caused some forms of urticaria.

Thus urticaria has been ascribed to a wide and apparently unrelated series of causes. Upon further analysis, however, they are found to hang together relatively coherently, and to operate according to very simple principles. Urticaria is regarded as an anaphylactic phenomenon, clearly due in the majority of instances to a toxic protein. Some substances, though, which cause urticaria are clearly not protein. The problem is to bring these two groups, as well as the influence of the nervous system, into harmony. Before discussing this, let us revise our etiological classification on the basis of two groups of toxins — the protein and the non-protein.

Group I. Protein Toxins.

A. Animal Proteids:

1. Ingested: Egg albumin, milk, cheese, flesh of mammals, birds, fish, crustacea and bivalves, etc.
2. Otherwise Introduced: Sera, including antitoxins.
3. Animal parasites or their metabolic derivatives: vermes and protozoa.

B. Vegetable Proteids:

1. Ingested:
   a. Food: Peas, beans, lentils, cereals, mushrooms, nuts, straw-

berries, cucumbers, pickles, onions, garlic, corn, honey, tea, coffee, etc.

b. Medicaments: The balsams, opium and its derivatives, quinine, and hyoscyamus, etc.

2. Otherwise Introduced: Drugs, sera and vaccines.

Group II. Non-Protein Toxins.

1. Ingested:
   Medicaments: Salicylates, the coal tars and arsenic.
2. Otherwise Introduced: Medicaments, mercury and arsenic, chiefly arsphenamin.

The symptoms of anaphylaxis in man are less severe than in animals used for experimentation. A salient feature is urticaria, another is asthma, a third is eosinophilia. The way in which the reaction is determined is by the union of the toxin by means of amboceptors or without them, to the body cells. This being the case, the action of the non-proteid toxins which provoke urticaria is explicable in one of two ways. They either are similar to the proteid toxic molecules and thus have similar affinities, or they cause chemical changes in the body proteids so altering these as to render them capable of similar behavior as toxic entities, or of altering the cells themselves rendering them hypersusceptible. This explanation, too, would account for the rôle played in the process by metabolic diseases, and disturbances of digestion and elimination, as well as the cases associated with or following asthma, small pox, pemphigus, etc.

In metabolic diseases substances unusual as to composition or quantity are produced, and must be either proteid or non-proteid in character. Their behavior would be subject to the scheme outlined above. The same is true of diseases of elimination which cause the retention of substances promptly excreted in health. In digestive derangements the manner of intoxication may be two-fold, either by the production and absorption of abnormal substances, or by an alteration of intestinal permeability favoring the absorption of substances usually present, but not assimilated in health and which, when absorbed, become pathogenic. Proteid putrefaction is frequently an associated condition, an indicator of which is indicanuria which, in older days, was itself considered a cause of urticaria. Asthma, as already mentioned, is a symptom of anaphylaxis; and pemphigus, variola, etc., would cause urticaria in the manner suggested under disturbances of metabolism and elimination.

The pathogenic rôle of the nervous, preeminently the sympathetic, system has been already touched upon. It may be immediate, and

act simply by means of increasing vascular instability, or mediate, and act by altering metabolism, elimination or digestion, in which case the explanation of this phase of the problem is along the lines laid down in the previous paragraph. The reflex nervous manifestations associated with dentition, menstruation, gestation, lactation and the climacteric would come under this head, when urticaria is a concomitant condition. As a corollary to this the endocrine mechanism would participate in the process.

Among those subject to urticaria the minutest quantities of pathogenic substances, as one strawberry, a trifle of egg albumin, or a drop of quinine solution will suffice to excite an attack. Immunity does not develop in these patients, but may be artificially produced, as Schloss has shown in his ingenious and remarkable study on egg albumin urticaria.

The manner in which the external causes of the disease operate remains to be considered in connection with anaphylaxis. In Schloss's case attacks were provoked when the egg albumin reached the patient's lips, even before he swallowed it. On one occasion, when the child merely played with an egg shell to which some of the white had adhered, he had a seizure. In other words, a high degree of sensitization was present. This is the probable way in which all local irritants operate. Cases following sea voyages and changes in surroundings are probably in the metabolic and digestive groups.

The Mechanics of the Urticaria Lesion: The toxin causes primarily a spasm of the cutaneous vessels, according to Unna, the veins. Whether this is due to a direct effect on the vessel by means of stimulation of the vaso-contractors, is not important. Because of obstructed return circulation, a serous transudate forms locally exerting further pressure on the vessel originally involved, as well as on its neighbors. Thus, a small area is made ischemic. This explains the whiteness of the wheal. As the vessel relaxes and the local circulation is re-established, the wheal fades. Often no trace remains. Frequently, however, a slight local inflammation persists, due to vascular dilatation and congestion after the spasm. When the exudation is extensive a vesicle, bulla or papule forms. The histopathological features need not be mentioned. Other factors in the productive mechanism of the disease are diminished blood coagulability, increased permeability of the vessel walls and increased irritability of the muscles and nerves of the vessels, all of which are phenomena admittedly related to anaphylaxis. Sczamberg, as already mentioned, ascribes the wheal to sudden oversecretion in the cutaneous lymphatics — in other words to an acute local lymphedema.

Treatment. The treatment of urticaria is prophylactic and

actual. When it is definitely known that certain foods or drugs in given individuals cause urticaria, they must be avoided. Thus, a careful history of every case is necessary, in the first place to rule out the simpler etiological factors, and in the next to discover digestive, metabolic or nervous causes which might favor the attack. The actual treatment is internal and external; the object of the former being to eliminate the toxin, of the latter to lessen the itching.

The internal treatment should consist of catharsis, and diuresis. Calomel and salts, or salts alone, followed for a few days by mild vegetable cathartics such as rhubarb or cascara will usually suffice. In obstinate cases, and in those presenting a history of constipation, or in those in which proteid putrefaction is ascertained by examining the stools, suitable diet should be prescribed, and colon irrigations, the administration of intestinal antiseptics, such as salol, ichthyol, small doses of phenol, or cultures of the Bulgarian bacillus. Of these the colon irrigations are the best. Diuresis should be caused by an increased ingestion of fluids and the use of diuretics. Sudorifics and hot packs, to produce cutaneous elimination, are contra-indicated, since the irritable skin will tolerate them poorly. Calcium lactate in massive doses, three grammes before breakfast, is of benefit in many cases. This treatment is based upon Kastle, Healy and Buckner's work. Calcium lactate not only tends to overcome anaphylactic shock, but calcium and its salts are supposed to decrease the permeability of vessels, increase blood coagulability and quiet the nervous system. Alkalies, atropin, the bromides and other sedatives are used to meet special indications, actual or theoretical, but are of little value. In many instances the hypodermatic injection of suprarenal gland extract causes immediate and permanent relief. Recently, in obstinate cases, autoserum administration has been employed, but the results are inconstant and the advocates of the treatment unconvincing. The only rational treatment in obstinate cases would be to determine the etiological factor or factors and artificially immunize the patient along the lines suggested in Schloss's work. This, however, would be difficult in most instances and necessary only when the intolerance exists towards important foods and drugs.

The local treatment includes the use of lotions, powders, washes, baths and ointments. Calamine or calamine and zinc lotion, with one percent. of phenol or menthol, or five percent. of ichthyol, applied several times a day, or mentholated powders made with zinc stearate, zinc oxide, bismuth or talcum often give immediate and

permanent relief. Washes, consisting of one percent. menthol or phenol in equal parts of alcohol and rosewater, either sponged on the itching points or sprayed on with an atomizer, may be employed in addition to the lotions and powders. One of the old household remedies, bicarbonate of soda in water, often does good. Bran baths at the patient's favorite temperature, or soda baths are very useful. The patient, when drying after his bath, should be cautioned not to rub vigorously, but rather to use the towel as a sort of blotter. Ointments of one percent. of phenol or of one to five percent. of menthol are occasionally useful, but on the whole irritate the skin more than they relieve it. Lotions, baths and washes when employed frequently tend to dry out the skin. In this event, either powders or a zinc paste, consisting of from thirty-three to fifty percent. of zinc oxide in lanolin, or in equal parts of lanolin and vaseline, should be used. Frequently following urticaria, as indeed after all itching diseases, the skin may continue to itch after the acute process is over. Whether this is a cutaneous neurosis or not is not known. When all efforts to alleviate these cases by the use of ordinary antipruritics prove fruitless, the Roentgen rays will often succeed.

Schloss's studies in egg urticaria indicated the anaphylactic nature of at least this one type. Many investigators have sought to apply to urticaria of other origin the principles embraced in this concept, with the therapeutic idea as prominently in mind as the diagnostic. Proteid extracts in dry form have been prepared from nearly all foods, some bacteria, and even such substances as horse dander, chicken feathers and the like. With these materials, suitably controlled percutaneous tests are preformed. If wheals larger than the controls develop, or if they are encircled by a hyperemic zone, the particular proteid or proteids producing the reaction are regarded with suspicion. It does not follow, however, that a proteid indicating sensitization is actually the cause of an existing disease; nor does it follow that proteids producing no reaction are not the cause. But it is a matter of experience that the causative factors, if the urticaria actually be due to a food, will be among those giving positive reactions. To prove this it is necessary to feed the patient with the suspected foods. If then, no other food having been eaten, an urticarial attack supervenes, this particular article of diet may be assumed to be pathogenic. In this manner I have found in one patient that buckwheat provoked angioneurotic oedema of the eyelids, in another that veal did, in another that

carrots caused recurrent urticaria, in another a like rôle played by pork. After eliminating these foods further attacks ceased.

It would be impossible to go into the matter in as much detail as both its interest and possible practical value merit. But emphasis should be laid on these facts. After positive percutaneous tests are found, the pathogenic rôle of the particular substance must be established by ingestion. If this causes renewed attacks the food should be removed from the patient's dietary, unless it is so important that active immunization would be warranted, an eventuality rarely conceivable in an adult.

Prognosis. The prognosis in most cases of urticaria is good. There are persistent forms, though, which through their constant itching with its resultant loss of rest, wear patients out. In individuals whose attacks invariably are caused by definite substances, the predisposition is the disease and is incurable save by artificial immunization. The prognosis as to the individual attacks however is good.

In intimate relation with urticaria are three other diseases, angioneurotic edema, urticaria pigmentosa and infantile prurigo which links the urticaria group with prurigo.

ANGIONEUROTIC EDEMA

Synonyms. Giant Urticaria; Urticaria Edematosa; Wandering Edema; Acute Essential Edema; Quincke's Edema; Acute Circumscribed Edema.

Definition. Angioneurotic Edema is an infrequent, acute, recurrent disease of the skin or mucous membranes characterized by the sudden appearance of an edematous swelling varying from the size of a walnut to that of a hand.

Symptoms. The onset of this disease is sudden, at times accompanied by moderate fever, vomiting, diarrhoea, nervousness or psychic depression. It tends to appear in the early morning hours, and frequently patients who have had previous attacks have premonitions of an impending one. The lesions are huge wheals including the skin and subcutaneous tissue. They are usually solitary, occasionally multiple, and at times return at one particular spot. They are white as a rule, but may be red; and they itch, burn, or pain and give rise to a sensation of tenseness. Their consistency is usually elastic and hard, but not invariably. Their shape is circular, oval, or irregular and their favorite sites, in the order of frequency, are the face, genitalia, or anywhere on the body surface or in the buccal orifice. At times when the epiglottis is involved, breathing is interfered with and asphyxiation threatened.

Course. The duration of an attack is a matter of hours, or even days. The disease as a whole, however, lasts for years, the recurrences varying in frequency with free intervals of increasing length until they cease entirely.

Differential Diagnosis. It is almost impossible to confuse this condition

with any other because of the characteristic onset and appearance of the lesion.

Etiology and Pathogenesis. According to Darier, and his views seem reasonable, the etiology of giant urticaria is one with that of ordinary urticaria. This is probably correct. It is true, however, that the disease is more frequent among women than men, that it is often familial, and associated with emotional disturbances, goitre, neuralgia and psychic diseases, chiefly melancholia.

Treatment. The treatment, both local and general, parallels that of urticaria. When the epiglottis is seriously involved, intubation is indicated.

URTICARIA PIGMENTOSA (FIG. 4)

Synonyms. Xanthelasmoidea; Chronic Urticaria leaving Brown Spots.

Definition. Urticaria Pigmentosa is a rare disease usually beginning within the first year of life characterized by the gradual appearance and progressive increase of brown maculo-papular or nodular lesions which, upon irritation, become evanescent wheals.

Symptoms. The disease usually begins before the end of the first year with tan spots or wheals which turn brown and gradually increase in size and number. The back is most frequently involved, chiefly the lumbar region and buttocks (Fig. 4). From these sites the lesions may spread to the rest of the body, rarely including the neck and face, and still more rarely the buccal mucosa. To find the lesions elsewhere than on the back and buttocks is exceptional. At times slight itching is present, but there are no constitutional symptoms.

The lesions vary from the size of a lentil to a lima bean, and are macular, papular, nodular, or a mixture of the three. They are symmetrical as a rule, numerous, although cases with but from two to six spots have been reported. They tend to follow the cutaneous lines of cleavage and their color shades from a yellowish bronze to brown. In typical cases the affected areas suggest the striped markings of a tiger (Fig. 4). When irritated, wheals rise at the sites of the lesions, or the lesions get redder and larger. This is transitory. Dermographism is frequently present, as well as a general glandular enlargement.

Course. The disease is chronic and the pigment persists; but the entire process usually ends at, or at the end of adolescence.

Varieties. There is but one known variety of this disease.

Differential Diagnosis. There are many cutaneous diseases characterized by discolorations, but only two strongly resembling

urticaria pigmentosa. These are urticaria with pigmentation and xanthoma tuberosum (Fig. 41). Urticaria with pigmentation does not begin in infancy and is characterized by the typical picture of urticaria, sudden onset, intense itching, constitutional symptoms and rapid disappearance of the lesions, except the pigment they leave which fades gradually. Urticaria pigmentosa begins gradually in infancy, changes slightly if at all, is devoid of constitutional symptoms and the pigment never disappears during childhood. Xanthoma is almost never seen in infancy and the lesions are, for the most part, orange yellow. It has a definite microscopic structure, as has also urticaria pigmentosa. The latter is characterized by a dense infiltration of mast cells distinguishing it from any similar disease. This point is mentioned because a microscopic study of the lesions may be essential in diagnosis.

Etiology and Pathogenesis. The etiology is absolutely not understood. The disease is obviously due to a congenital disturbance, either anatomic or metabolic. Neisser considers it a nevus; Little a congenital tendency to overproduce mast cells; Knowles thinks it is due to an unknown toxin affecting a congenitally abnormal skin, the most reasonable view of the three.

Treatment. There is no known effective treatment.

Prognosis. The prognosis as to life is good. The pigmentation usually disappears in time.

INFANTILE PRURIGO

Synonyms. Strophulus Infantum; Urticaria Chronica Infantum; Varicella Prurigo; Lichen Urticatus; Prurigo Temporanea.

Definition. This is a not very well defined disease of childhood, the characteristic features of which are the presence of wheals, papular and vesiculo-papular lesions accompanied by severe itching.

Symptoms. The disease begins early in life and its onset is gradual. At first wheals appear, upon which develop small shotty, brownish-red or red umbilicated papules, some of which are surmounted by vesicles. The sites most intensely involved are the extensor surfaces of the extremities, flanks and lower abdomen, but no area of the body surface is necessarily immune. Successive groups of lesions develop so that all stages may co-exist, creating a varied picture. Scratching causes consecutive lesions. At the summits of the vesicles and papules punctate brown crusts form. Diffuse, long excoriations exist by the side of these, and in places the skin becomes thickened. Dermographism is present. To the touch

the skin is roughened and it feels like a grater. Rarely, secondary infections add impetiginous lesions with their types of crusts, to an already diversified picture. Constitutional symptoms, save possible constipation, are entirely wanting. Occasionally, a peculiar ashy pallor is seen.

Course. The disease is chronic, lasting throughout childhood, and disappearing between the eighth and tenth years.

Differential Diagnosis. Two conditions simulate this one — scabies and prurigo. The former favors the skin folds, flexors and interdigital spaces, etc. (See Scabies.) A demonstration of the acarus clinches the diagnosis. Prurigo begins in early life and persists throughout adult years. Since many cases of prurigo begin as lichen urticatus there are no positive criteria for differentiation between the two excepting the course of the disease.

Etiology. The etiology suggests that of urticaria as far as the proteid origin of that disease is concerned.

Treatment. The treatment corresponds in general with that of urticaria, but a few special points must be borne in mind. Internally arsenic and iron are often of great avail. Locally, tar and sulphur salves and pastes, in addition to the medicaments suggested in the treatment of urticaria, are useful.

℞ Ol. Rusci: 4, 0 to 6, 0 or Sulphur ppt. 5, 0
Zinc Oxid: 20, 0
Petrol. Flavi: q.s. ad 50, 0
Sig. Apply twice a day, once after bathing.

℞ Ol. Rusci: 1, 0
Alcohol: q.s. ad 50, 0
Sig. Paint body once a day after bathing.

Prognosis. The general health is not affected in this disease. The attacks are amenable to treatment to the extent that their disagreeable symptoms are controllable, and that frequently the eruption may be caused to disappear. The disease as a whole goes spontaneously at the end of childhood.

PRURIGO (FIG. 5)

Synonyms. Prurigo of Hebra.

Definition. Prurigo is an infrequent, chronic disease beginning in infancy as chronic urticaria, lasting either through adolescence or throughout life, and characterized by the presence of wheals, papules and consecutive lesions due to scratching and pruritis. It is often accompanied by profound constitutional disturbances.

Symptoms. The disease begins in infancy by the gradual increase, in successive crops, of wheals, and papules developing upon wheals, and accompanied by intense itching which is worse at night. As the result of scratching secondary lesions develop. The papules vary in size from that of a millet seed to a pea (Fig. 3). They are red, brown, or yellowish, hard, shotty and convex or obtuse, single or grouped, and situated primarily on the extensor aspects of the limbs (Fig. 5), next on the trunk, and finally anywhere else on the body, sometimes including the face. Scratching causes destruction of the summits of the papules, at which sites brown blood crusts develop. Other types of excoriations, irregular in length and distribution, are found. The skin becomes lichenified or " eczematized " in spots, and thus areas of thickening, vesiculation, redness and scaling are found. In severe forms the entire integument becomes leathery. Secondary infection causes impetiginous lesions and ulcerations which leave depressed pigmented scars, adding marked pitting to a leathery skin which may become diffusely bronzed. Thus a patient, in the severer forms of the disease, is enveloped in a thick, brown integument studded over with papules, ulcers and pigmented scars. The skin feels inelastic when pinched, and sometimes it is impossible to fold it sufficiently to raise it from the lower tissues. The glands, particularly the inguinal groups, become enormously enlarged, and visible as well as palpable.

Course. The general health suffers, sometimes seriously, partly from the underlying cause of the disease, partly from the constant anguish and disturbed rest of the sufferer. The milder forms disappear at the end of adolescence; the severer forms persist to the termination of a life usually shortened by intercurrent infection.

Varieties. There are two types of prurigo; the mild, or prurigo simplex or mitis, and the severe, or prurigo ferox, agria or gravis. The former is the type which disappears at the end of adolescence; the latter, the type which terminates in death. It is believed that these two types are distinct from the onset, prurigo ferox beginning with much intenser symptoms which grow more and more severe.

Differential Diagnosis. The diagnosis of prurigo rests upon the clinical history and appearance of the case. The disease begins in infancy, growing progressively worse. It is characterized by the site and appearance of the lesions, and the constitutional symptoms. Actually, nothing else suggests prurigo, but at times scabies (Fig. 43) and papular dermatitis may be confused with it upon first glance. Scabies is to be differentiated on the history, the milder

course of the disease, and the presence of the mites. Papular dermatitis usually begins later in life, is less severe, and the characteristic vesicles and scaling are present. Only in eczematized prurigo will confusion arise, and the history of the case will eliminate this. Obviously, prurigo must be differentiated from infantile prurigo, an impossibility until the end of childhood.

Etiology and Pathogenesis. The disease is due to disturbed metabolism causing a chronic intoxication, possibly anaphylactic, as shown by Hans Schwartz. The source of the poison is alimentary. Disturbed nitrogen metabolism and the excessive ingestion of proteids are associated conditions.

The disease is not common and is more frequent in Austria than elsewhere. Heredity, poverty, tuberculosis, anemia, filth and the neurotic habitus have all been held etiologically responsible, the last chiefly by the French school; and in the nervous group have been included the reflex phenomena of dentition, puberty and the climacteric. The interrelation between anaphylaxis on the one hand, and neuroses causing vague instability, tryptic indigestion, excessive proteid intake and disturbed nitrogen metabolism, on the other, are facts clearly indicated by Schwartz. The general factors of heredity and lack of hygiene would be contributory. The comprehensive list of causes hangs together consistently when properly reviewed.

Treatment. The treatment is prophylactic, eliminative, tonic and local. The prophylactic treatment consists of cutting down on proteid foods. Eliminative treatment demands free catharsis, intestinal irrigation, diuresis and hot packs. Tonic treatment consists of the use of arsenic either in the form of Fowler's solution, injections or Asiatic pills, and calcium lactate in three gramme doses before breakfast.

Intestinal antiseptics are of little benefit. Obviously, when itching is intense sedatives should be employed.

Local treatment has two objects: to control the itching and to cause the lesions to disappear. The itching may be controlled along the lines laid down in the treatment of urticaria. This, as well as the second object, may be attained by the use of revulsives and keratolytics, followed by sedative and keratoplastic medicants. Revulsives cause an inflammation of the skin and keratolytics a degeneration of the epidermis. In consequence the skin peals and the redundant epidermis and the infiltration are removed. To soothe the skin, after this, sedatives are employed, and to stimulate new formation of epidermis, keratoplastics. In prurigo, the best revul-

sives and keratolytics are tar, sulphur, salicylic acid and resorcin in high concentration. The best way of treating the patients is to paint the affected surfaces with ten percent. Oleum Rusci in alcohol, and immerse them in a hot bath for an hour. After drying, a mild ointment (ten percent. zinc oxide in lanolin or vaseline) is gently applied. Wilkinson's ointment (Ol. Rusci, Flor. Sulph. aa 10,0 Sap virid., Petrolatum aa 20,0) may be substituted for the tar solution; or two to five percent. B. Naphthol solutions or salves are valuable. After the application of these substances, the patient should be loosely wrapped up in single layers of gauze. In the course of several days the skin will become inflamed. Then sedative applications are to be used. One of the best is Lassar's Paste. It is to be applied gently to the entire affected surface twice a day. In addition, wet dressings of two percent. aqueous resorcin solution for half an hour, three times a day, to restore the epidermis, followed by calamine lotions, with or without one percent. of phenol, are indicated. Should the skin be impetiginous ammoniated mercury ointment is to be used until the special need for it disappears. Then the treatment with tars and sulphur is to be begun.

Prognosis. In prurigo mitis the prognosis is good, since the disease vanishes at the end of adolescence. In the other form, recovery never takes place and death usually supervenes.

PRURIGO NODULARIS

Synonyms. Multiple tumors of the skin accompanied by intense pruritis; Urticaria Perstans Verrucosa; Lichen Obtusus Corneus.

Definition. Prurigo Nodularis is an extremely rare, itching, nodular eruption of the backs of the hands, extremities and trunk.

Symptoms. The disease is gradual in onset, intensely pruritic, begins on the backs of the hands, involves the extremities and trunk, and is found chiefly among women. The lesions are papules from the first, or nodules, rising upon papules or vesicles, develop. At first they are smooth and white or pink, but grow rough and, gradually, warty, with age. Scratching fissures or furrows the surfaces. The lesions often coalesce.

Course. The course is chronic extending over prolonged periods of years.

Etiology and Pathogenesis. The etiology is not understood.

Treatment. Extirpation of the lesions and Roentgen therapy have been used without avail.

Prognosis. The disease is incurable.

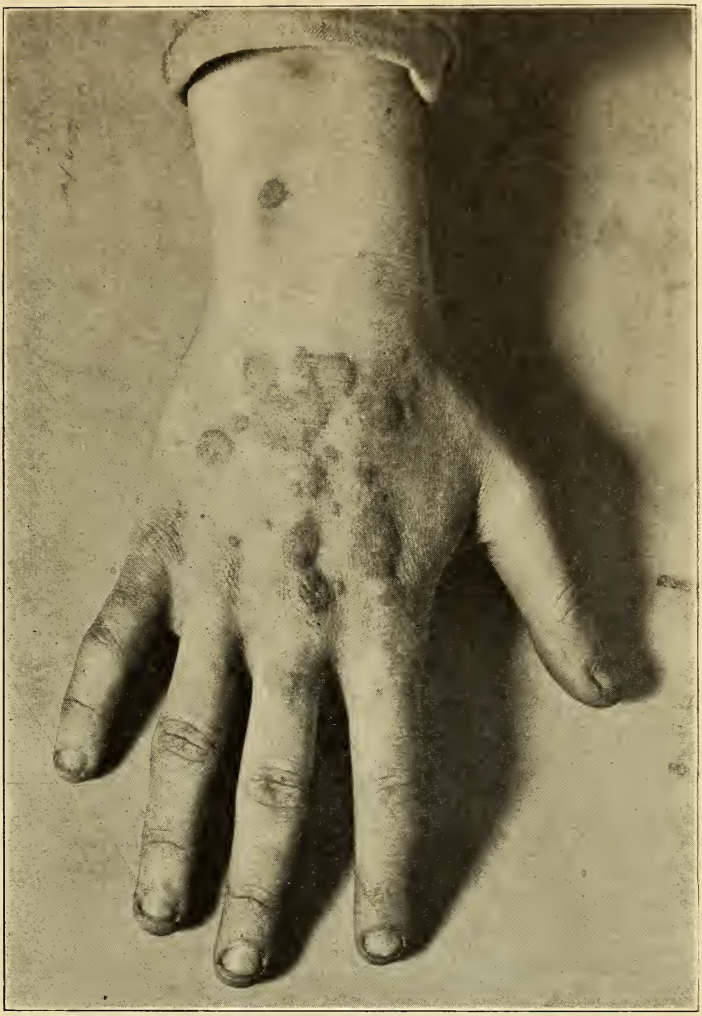

FIG. 1. ERYTHEMA EXUDATIVUM MULTIFORME

In this disease the back of the hands, wrists, neck, vicinity of the ascending ramus of the jaw, and finally any part of the body may be involved. The lesions are pink, red, or even purple, slightly elevated, and sharply circumscribed edematous discs, at times, by coalescence, forming festooned patches. Sometimes bullae arise in the centres; sometimes concentric rings of red and purple form, giving the respective pictures of erythema bullosum and erythema or herpes iris.

FIG. 2. ERYTHEMA NODOSUM

The shins seem covered with bruises of all sizes. The localization is characteristic of the disease. In erythema induratum (Bazin's disease) the lesions are rather on the calf, and many break down. Necrosis is rare in erythema nodosum. At times there is scaling, as the left shin shows. In syphilitic erythema nodosum fewer lesions, occasional necrosis and periosteal thickening are present. Erythema nodosum is often associated with erythema multiforme.

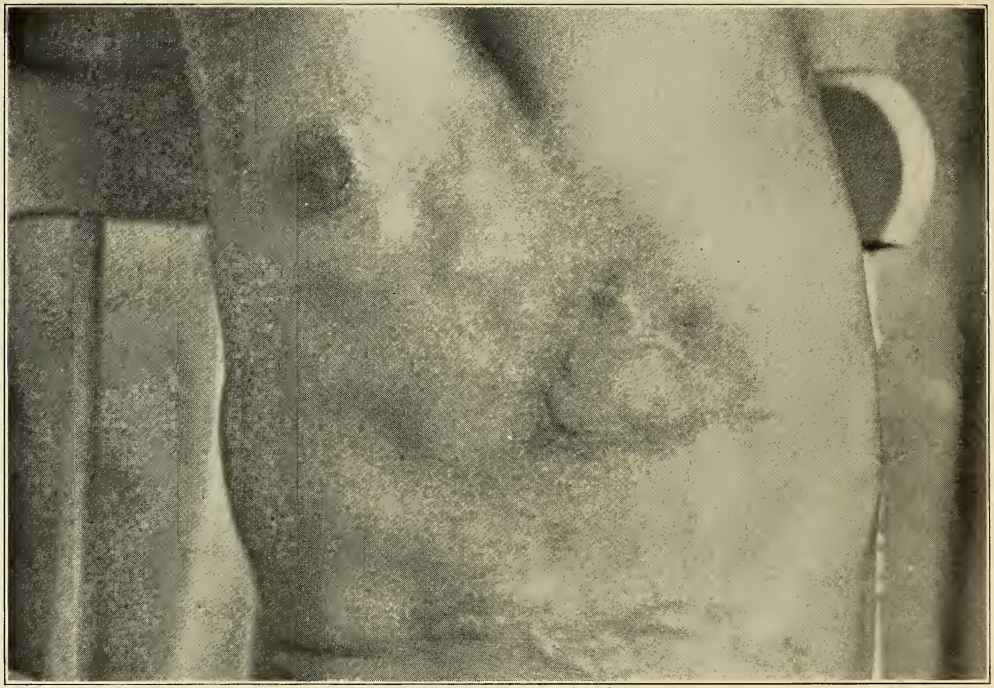

FIG. 3. URTICARIA

The wheals are gyrate and large. Giant urticaria is suggested. Where the high lights show, the whiteness of the lesions is indicated.

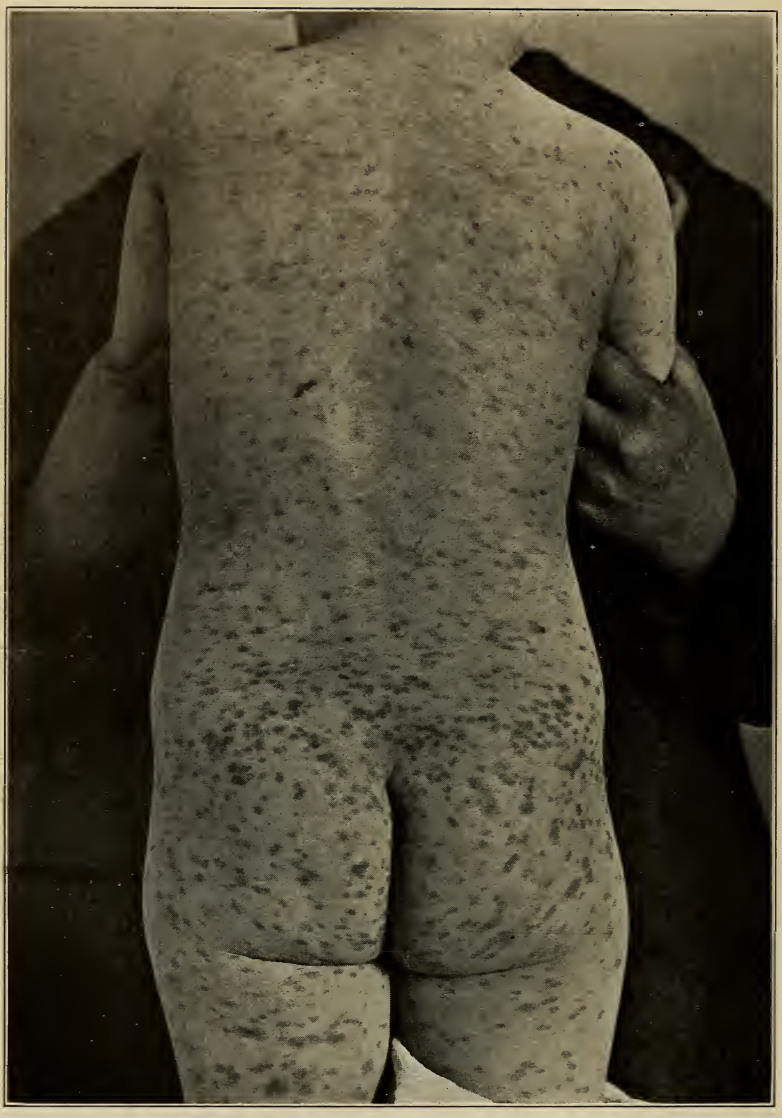

FIG. 4. URTICARIA PIGMENTOSA

A better designation is xanthelasmoidea because of the vague similarity to Xanthoma. The lesions are flat buff or brown papules which, under friction, become slightly elevated and resemble wheals. The lesions group themselves roughly into stripes, a configuration here seen above and on the buttocks. There is a suggestion of spotting as seen in leopards. The disease occurs only in the young, disappearing, as a rule, during adolescence.

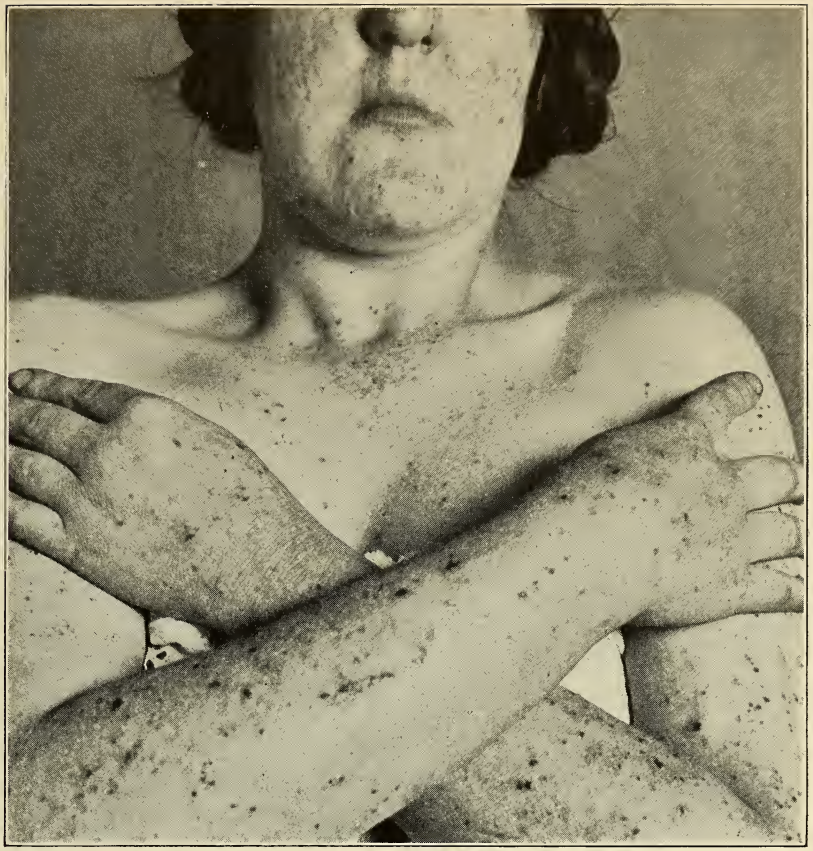

FIG. 5. PRURIGO

Small excoriated papules, mainly on the extensor surfaces of the limbs, and all over the torso, characterize this condition. The face is also involved, and the skin becomes thickened, or lichenified. In scabies the face is free, and the lesions favor the body folds and flexor surfaces. At times, prurigo-like lesions appear in leucemia, and thus it is well to exclude this condition in all cases of prurigo in adults.

CHAPTER VII

DRUG ERUPTIONS, EXANTHEMATA AND PURPURAS

DRUG ERUPTIONS

Because of the great variety of cutaneous reactions medicaments and chemicals are capable of producing, only those will be here described which have a frequent bearing upon ordinary dermatological practice. These dermatoses are closely allied to those of occupational origin caused by chemicals. Whether the pathogenic agent is ingested or locally applied, the brunt of the attack is borne by the cutaneous vessels. Dermatoses due to ingested drugs or chemicals tend to be disseminated and symmetrical, while those due to external irritants tend to be limited to the area exposed. This rule, however, is not inflexible. Arsenic when taken internally sometimes causes herpes of the zoster type; quinine employed locally, as it often is in hair washes, or belladonna locally applied in plasters, may both provoke generalized rashes; chemicals occasionally are the starting point of eruptions which extend far beyond their point of application. (See Chapter on Dermatitis and Eczema.)

The pathological changes underlying these diseases are congestion, exudation and necrosis. The primary objective expression of congestion is redness; of exudation, edema, and the formation of vesicles or bullae containing serum or blood, or both; and finally, of necrosis, or ulceration. The secondary objective expressions of these phenomena are scaling, pustulation, weeping, crusting and scarring. The symptoms consist of itching, tingling, burning or pain, or any subjective sensations whatever. In iodide and bromide poisoning, however, pustules and pustular granulomas form, not necessarily due to a secondary infection. Arsenic may cause palmar and plantar hyperkeratosis. (Chapter XV.)

The following table based upon Crocker's, and abridged in some respects, while amplified in others, indicates in clinical groups the variety of lesions which therapeutic agents administered internally may provoke.

Erythema. Arsenic, antipyrin, belladonna, boric acid, bromides, cantharides, capsicum, chlorate of potash, chloral, copaiba, Dichloramin T and Carell-Daikin solution, digitalis, iodides, iodoform, mercury, morphine

and its derivatives, pyramidon, phenacetin, phenophthalein, quinine, salicylates, sera, strychnine, sulphonal, veronal.

Vesicles. Antipyrin, arsenic, bromides, cantharides, cannabis indica, capsicum, chloral, copaiba, Dichloramin T and Carell-Daikin solution, ergot, iodides, morphine, quinine, salicylates, sera, sulphonal.

Bullae. Antipyrin, arsenic, cannabis indica, copaiba, chloral, iodides, mercury, opium and its derivatives, quinine, salicylates, sera.

Wheals. Antipyrin, arsenic, bromides, copaiba, iodides, iodoform, quinine, salicylates, sera.

Pustules. Arsenic, bromides, chloral, iodides, mercury, salicylates.

Purpura. Antipyrin, arsenic, chloral, ergot, iodides, iodoform, mercury, quinine, salicylates, sulphonal.

Erythroderma. Arsenic, arsphenamin, mercury.

"Eczema." Arsenic, iodoform, mercury.

Necrosis. Arsenic, ergot, iodides.

Hyperkeratoses. Arsenic.

Granulomas. Bromides, iodides.

Herpes. Arsenic, arsphenamin.

Cyanosis. Antipyrin, acetanilid.

Pigmentation. Arsenic, silver, bismuth, lead.

Mucous Lesions. Arsenic, bismuth, lead.

Angioneuroses. Aspirin.

Of this extensive list it is important to bear in mind a few substances and the lesions they may provoke. It must also be remembered that in average doses they are toxic only to the susceptible, in other words, to but few individuals. The iodides are the one exception to this rule, for the majority of people are found intolerant to them in one way or another. These drugs may be classified according to their source, and with their important representatives as follows:

I. Medicaments derived from Animal Sources:
   Sera, small pox virus.

II. Medicaments derived from Vegetable Sources:
   Belladonna, quinine, copaiba, digitalis, opium and its derivatives.

III. Medicaments derived from Mineral Sources:
   1. Metallic elements and their compounds; arsenic, bismuth, lead and silver.
   2. Non-metallic elements and their compounds; iodides, iodoform and bromides.

IV. Synthetic Medicaments:
   Antipyrin, pyramidon, chloral, sulphonal, veronal, salicylates.

SERUM SICKNESS

Definition. This disease is characterized by a polymorphous eruption associated with a systemic reaction following the injection of alien serum.

Symptoms. The symptoms are cutaneous and general. The cutaneous manifestations consist of the appearance of an eruption which, according to von Pirquet and Schick, has the following attributes. It first appears at the site of the injection, spreads symmetrically, the urticarial forms itch, and the exudative and morbilliform varieties favor the extensor aspects of the limbs. Hartung mentions four varieties of eruption, the urticarial, scarlatiniform, morbilliform and polymorphous exudative. As a matter of fact no such distinctions can arbitrarily be made since the enumerated types may coexist and since there are intermediary forms. Ormsby adds to these the rare vesiculo-bullous rashes.

The general manifestations are numerous. Regional glandular swellings take place, subject to the site of injection, and their onset and disappearance precede the arrival and departure of the rash. Edema, usually without albuminuria, is common. It is found on the face, eyelids, penis, scrotum and feet, suggests angioneurotic edema, and has no renal disturbance as a basis. A leukopenia with high relative lymphocytosis is common. Arthralgias, malaise and prostration are all frequent concomitants of the malady and fever is a constant feature. The temperature rises to 101° or even to 105° in the afternoon; it is remittent, disappears by lysis and recrudescences are common. With the height of the fever the glandular enlargement reaches its maximum.

Course. The onset of the attack, preceded by glandular swelling, usually occurs in from one to two weeks after the injection, but may come much earlier or later. At the same time malaise and slight fever appear. The rash begins near the puncture and then, becoming disseminated, assumes the character mentioned above. As it spreads the glands increase in size, while the fever rises more and more. The attack is likely to end within a few days, but may last four or five weeks. Recurrences are not uncommon. The types resembling scarlet and measles desquamate. Re-injection, if at too short an interval, often causes severer attacks.

Diagnosis. The main diagnostic factor is the history of the injection. The next in importance is its beginning near the puncture wound. Since measles and scarlatina often develop in children with

diphtheria, and since serum therapy is almost entirely restricted to diphtheritics, or those exposed to this disease, scarlatina and measles often must be ruled out. This is usually easy. In scarlatina the onset with vomiting, the angina, strawberry tongue and enanthem serve as guides in diagnosis. In measles, the usual coryza, conjunctivitis, bronchitis, and, in the prodromal stage, Koplik's spots are sufficiently distinctive. The urticarial and exudative types of serum disease can be distinguished from hives and the toxic and multiform exudative erythemas, solely by the history.

Etiology and Pathogenesis. The cause of the disease is the foreign proteid contained in the serum and not the antitoxin as was once supposed. The patient is susceptible to the substance, and as has been shown by von Pirquet and Schick, may become hypersusceptible when sensitized by the first injection and exposed upon re-injection at too short an interval. Re-injection may cause the appearance of the attack within a few hours after administration. Thus, the question of etiology becomes one with that of anaphylaxis, along the lines indicated in the discussion of urticaria and multiform erythema.

Treatment. This is largely prophylactic. Von Pirquet and Schick have demonstrated that small doses of serum containing a high concentration of antitoxic units tends to lessen the incidence of the phenomenon. In addition too freshly prepared antitoxins should be avoided. If re-injection becomes necessary, as in recurrent attacks of diphtheria, antitoxin should be employed prepared from an animal [1] other than the serum of which was used in the first treatment. For the rest, the local and general treatment outlined in the chapter on urticaria should be followed.

Prognosis. The disease is never fatal, nor is it followed by any permanent disability. Because of its severity while it lasts, however, it should be guarded against.

Small pox virus. This is a substance capable of producing two noteworthy types of cutaneous response,— vaccinia and a bullous dermatitis.

Vaccinia is either localized or generalized. The localized vaccinia is the pustule developing at the site of a successful inoculation, and its characteristics are sufficiently well known to require no space here. Generalized vaccinia is due to a dissemination of the virus from the point of application over the rest of the body,— an auto-

[1] Horses are usually utilized. For re-injection sheep antitoxin may be used in patients sensitized to horse serum.

inoculation. The transfer is accomplished mechanically and the lesions develop in from four to ten days after the original vaccination. Each lesion undergoes the course of a vaccine pustule, beginning as a papule, becoming a vesicle, then an ulcerated pustule, and finally, crusting and healing with a superficial scar. They appear in successive crops accompanied by systemic symptoms of an intensity directly proportionate to the number and severity of the lesions. At times the lesions are hemorrhagic.

Bullous Dermatitis following Vaccination. This condition is uncommon, but merits attention. It occurs in two varieties; a mild form resembling dermatitis herpetiformis, and a severe form, frequently fatal, resembling acute pemphigus. Bowen (*Journal of Cutaneous Diseases,* 1901) reported six cases of the mild type and Corlett later added another. How, in the same Journal, reported cases of the severe type and Mook has reported eight cases, three of which terminated in death.

In both groups systemic reactions, fever, prostration, albuminuria, are present, varying in intensity. The incubation period is from four days to as many months, and the disease develops irrespective of whether the vaccination was successful or not. It is prone to recur and its course is of variable length up to seven months. It bears no resemblance to vaccinia. Mook was unable in a series of well conceived studies to isolate the causative agent. A personal predisposition must exist since only a small proportion of the patients vaccinated with a given virus were affected.

Belladonna. Belladonna causes an eruption resembling scarlatina. Even the small quantities employed in diseases of the eye may produce eruptions in the susceptible. The rash consists of a diffuse erythema, often intensified by punctate macules of a deeper red; or it is composed of a general confluence of such macules. It fades without desquamation after the drug is no longer administered, and is usually accompanied by dilatation of the pupils and dryness of the pharynx. The history of the use of the drug, the midriasis and suppression of saliva, the absence of fever, angina and vomiting at the onset, all serve to distinguish it from scarlatina, with the earlier stages of which it may be confused.

Quinine provokes erythemas, vesicles, bullae and wheals. Even small quantities of this substance are sufficient to excite such cutaneous reactions in the oversensitive; nor need the drug be internally administered. In a manner analogous to egg albumin urticaria the external application of the drug, in scalp lotions for in-

stance, may produce an attack. The commonest manifestations are the erythematous and urticarial, resembling the toxic and exudative multiform erythemas, or ordinary hives. The latter itch. Vesicles and bullae may develop upon the simpler lesions, but are rare. Polymorphism is often present. Ringing of the ears is frequently associated with the rash, and occasionally there is vomiting.

Copaiba also causes lesions resembling those produced by quinine. The only important variety, however, is an itching erythema closely simulating the macular syphiloderm, or roseola. Since this drug is used in the treatment of gonorrhoea, and since the latter is at times associated with syphilis, the diagnosis between copaiba rash and the roseola may be a difficult one to establish. The history of taking a drug, and the pruritus, coupled with the absence of corroborative evidence of syphilis, constitute a reasonably certain basis of differentiation.

Digitalis may rarely cause an erythema resembling the toxic variety. Because it is indispensable in the treatment of cardiac disease in which toxic erythemas often arise, particularly when the heart lesions are due to rheumatism or sepsis, it is necessary to determine whether the drug or other agents are at fault. Dilatation and overactivity of the heart, as well as gastric irritability, may accompany digitalis eruption. The differential diagnosis depends to a great extent upon persistence of the rash after interrupting the use of the drug. In this event other forms of toxic eruption are to be considered. (See Chapter VI.)

Opium and its derivatives may cause erythemas, vesicles, bullae, pruritus, diaphoresis, pustules, ulcers and scars. The first three conditions are rare except as secondary to the fourth. The last three have a peculiar significance. The most important by far is the pruritus which is severest about the nostrils, but may be generalized and not associated with visible lesions. In the event of free diaphoresis sudaminal vesicles may develop (see miliaria crystallina), and these heal with desquamation. With diaphoresis, too, erythemas and wheals are at times associated. The pustules, ulceration and scarring are all due to one and the same cause, not to the drug, but to its hypodermatic injection under careless asepsis. This is observed only in addicts who inject themselves, and a distinguishing feature of this cutaneous phenomenon is the presence of the lesions only on parts of the body accessible to the hand employed in the injection. Thus, the right upper extremity and other parts without the range of the hand of a right-handed individual will be

free. The pustules resemble small furuncles, the ulcers ecthyma, and the scars those of variola. The face never is scarred. The differentiation from small pox cicatrices can be made only by the curious distribution. Concomitant features of opium eruptions are contracted pupils, moist skin and mental torpor or somnolence.

Arsenic, Fowler's solution, Asiatic pills, arsphenamin and other arsenicals, in the predisposed, all cause a great variety of cutaneous disturbances, namely, erythemas, vesicles, bullae, wheals, pustules, purpura, dermatitis, necrosis, hyperkeratosis, herpes, pigmentation and mucous congestion. Of these, the erythemas, dermatitis, hyperkeratosis, herpes, pigmentation and the arsphenamin dermatoses are important. The rest are rare. Arsenical erythema is scarlatinoid in character. Dermatitis of arsenical origin is not frequent, but must be recognized. Clinically, it has the common features of this disease. (See Chapter IX.) Harding reported cases of this nature in two families, caused by the use of insect powder. Hyperkeratosis excited by this metal is usually limited to the palms and soles. Rare forms of hyperkeratosis have been reported. The palmar and plantar lesions have a curious velvety feel, and are fawn colored. As Udo Wile pointed out epithelioma may develop upon this basis, particularly in psoriatics, a confirmation of the experiences of White, Hutchinson, Hartzell, Darier, Schamberg and others cited in Wile's article. Herpes of the simple variety, about the nose and lips, is not infrequent, and occasionally a form suggesting zoster, particularly in the region supplied by the supra-orbital nerve, is seen. Lustgarten believed that this form occurred only in anemic or undernourished individuals. The pigmentation strongly resembles that of Addison's disease. Another type, however, is observed in psoriasis and lichen planus. In these diseases the lesions, after fading, tend to leave pigmented spots and the discoloration is supposed to be more intense after arsenic therapy.

Arsphenamin sometimes causes either simple herpes, or a variety resembling zoster. Immediately after the intravenous injection of arsphenamin a fugaceous erythema sometimes arises. It starts about the face and neck, rapidly involves the trunk, arms and thighs, and fades within half an hour or so. It is either morbilliform or scarlatinoid, does not itch, and is sometimes associated with hyperemia of the conjunctiva and buccal mucosa, dyspnoea and epigastric pain. At times it is followed by fever and diarrhoea. This syndrome is most frequently observed after the third or fourth injection, and strongly simulates an acute anaphylactic attack, as pointed out by

Homer Swift, even though the toxic agent is not protein. Another type of skin disturbance is one that arises within a week after the administration of salvarsan. This is extremely rare and resembles the scarlatinoid erythrodermas. (See Chapters VI and XII.) It begins either as the form just described, or starts after from three to seven days with a universal, highly itching erythema, strongly resembling scarlet without throat symptoms, and is accompanied by a high remittent fever, prostration, vomiting, diarrhoea, and at times suppression of urine. When renal function is again established albuminuria and hyalo-granular casts are observed. Although death is unusual in these instances, the patient is severely ill. The course of the disease lasts from three weeks to three months, the skin returning to normal after a prolonged period of itching and scaling. Whereas the first type of adverse reaction appears to be due to sensitization, developing as it does after three or four injections, the second variety often comes after the first injection and would thus appear to be due to a fundamental intolerance to arsenic without reference either to its cumulative effect, or to sensitization.

Mercury. When applied locally mercury is capable of producing dermatitis through its irritant action, but when injected it usually has no effect upon the skin, although Jarisch has reported erythemas, eczemas and purpuras. Considering the amount of mercury that is used in medicine and the rarity of such eruptions following its use, it is difficult to hold mercury responsible for them. In the mucous membranes of the buccal orifice, on the other hand, it produces changes, and since stomatology is included in dermatology, these must be understood. The mucosa becomes red, edematous, painful, the gums in particular being affected, and small gray ulcers on an inflamed base develop, some of which resemble syphilitic mucous patches, and others aphthae. Because of the use of mercury in syphilis, the ulcers caused by the former must be differentiated from those due to the latter. (See Section on Syphilis, Mucous Patches.) Diarrhoea sometimes, ptyalism always, is associated with these phenomena, the ptyalism usually being the first sign of mercury poisoning. Formerly loss of teeth and necrosis of the alveolar process were frequent consequences of the unrestricted use of mercury in the treatment of syphilis, for in those days it was thought that the therapeutic value of the drug was insufficient unless the patient had mercurialism.

Bismuth. Bismuth as the cause of changes within the province of the dermatologist has, until recently, received no recognition.

Baehr and Mayer, in connection with the use of Beck's paste in the treatment of obstinate bone sinuses, described buccal ulcers in three patients, chiefly in the lower gum and tongue, and a pigmentation on the tongue and gums. The pigmentation was in the form of a fine line made up of blue-black points, just at the edge of the gums, the tip of the tongue and, in one instance, on the inferior laryngeal surface following the ranine veins. The tongue was swollen and extremely painful. Two cases recovered from the general symptoms of bismuth poisoning, but the pigmentation persisted. The third patient died. She not only had the mouth manifestations enumerated, but a morbilliform universal rash which lasted for two days.

Lead. This causes no cutaneous manifestations, but in the mouth is seen the "lead line," a violet streak of pigmentation on the lower gum, a short distance below the insertion of the teeth.

Silver used protractedly by patients for urethritis, cystitis, or in diseases of the nasopharynx and eye, causes a peculiar ghastly, ivory-blue or slate colored pigmentation. This is intensest on the face down to the shoulder girdle, and usually paler elsewhere. Silver pigmentation is known as argyria.

Iodides (Fig. 6) cause erythema, vesicles, bullae (Fig. 6), wheals, pustules, necrosis, granulomata and purpura. No other single drug, as already mentioned, causes cutaneous reactions in so large a proportion of those to whom it is administered. The erythemas are of toxic, multiform erythematous, morbilliform and scarlatinoid varieties. Frequently these are admixed with papules, or papules arise independently. They are hard and shotty, tend to appear on the face, upper extremities and trunk, and are prone to suppurate, giving rise to pustules resembling acne and furuncles. Ecthymatous and impetiginous lesions too are found, particularly in connection with the bullous and urticarial iododermata. Necroses of the skin may develop in connection with these forms, and all such processes heal with scarring. One of the nodular forms of iododerma occurring on the extremities simulates erythema nodosum. Jarisch mentions a pemphigoid form frequently fatal, and Hallopean has reported a similar result in a case of this type. Purpuras, too, are not uncommon. The most important varieties of iododerma, however, are the papular and pustular. The papular form may cause confusion with the papular syphiloderm (Fig. 85); the pustular with acne, varicella and impetigo. The pustules of iodide acne arise independent of comedones, but this distinction is of no practical value in seborrhoeic patients who may be suffering with both acne vulgaris and iodide acne.

Varicella may be simulated to a nicety because of the admixture of papules with the pustulo-vesicular lesions which are frequently umbilicated. The iodide impetigo is more indurated than the ordinary type.

The history of drug ingestion and iodinuria confirms the diagnosis which can never be made solely on clinical grounds. The onset of the eruption may take place during the time the drug is being taken, or even shortly after it is stopped, and the cause may depend on renal insufficiency, for ordinarily the urinary excretion of iodides is rapid. The treatment consists of stopping the drug, whereupon the eruption gradually disappears. Arsenic is supposed to hasten the healing.

Iodoform causes erythematous eruptions of various types sometimes associated with severe systemic symptoms simulating the typhoid variety of uremia.

The Bromides (Figs. 6 and 7) produce a series of eruptions paralleling those caused by the iodides and in addition two other sorts, both granulomatous, one of which is condylomaform, the other tuberous. The former tends to occur in infants, but is also found in later life. Its favorite site is on the cheek and neck near the angle of the jaw. It consists of one or more lesions about the size of a quarter, elevated like a plateau, or in the form of a walnut, resting on an indurated inflammatory base, and it is composed of numerous minute confluent pustules, forming a picture which simulates the cribriform aspect of a carbuncle. The tuberous form, known as the bromoderma tuberosum, is found chiefly in adults, most frequently on the shins and calves (Figs. 7 and 8), but also on the forearms, and finally, anywhere else on the body. It is either vegetating in appearance and bluish brown to purplish in color, or ulcerous with a vegetating margin, or nodular with central necrosis. It closely resembles erythema induratum, gumma, sporotrichosis or blastomycosis (see respective chapters), from which it must be differentiated. Both of these forms are often associated with other bromodermata such as the acne, the pustular, vesicular, etc. Infants often get the disease through the maternal milk. Epileptics and subjects of hysteria are also often afflicted. In short, the disease is seen in patients who for one reason or another take the drug over long periods. The treatment is precisely like that for iodide eruptions.

Antipyrin produces erythemas, vesicles, bullae, wheals, and petichiae and a rare gummatous lesion of the tongue. *Pyramidon* produces an erythema. *Chloral* causes lesions exactly like those of antipyrin, as do also *Sulphonal*

and the *Salicylates,* while *Aspirin* gives rise to angioneuroses. *Veronal* may cause the numerous varieties of skin reactions observed in antipyrin intolerance, as well as erosions about the mouth and anus, described by Pollitzer. Thus it will be seen that this group of drugs excites cutaneous reactions of the erythemato-urticarial and polymorphous types, with their pathological sequellae of vesicle and bulla formation. It may be added that innumerable other synthetic substances have similar power. One further form of antipyrin rash deserves mention. This is characterized by the appearance of circular violaceous macules, about the size of a quarter, which appear on the trunk, leave pigmentation when they fade, and tend to recur, whenever the drug is resumed, at the same sites. Phenophthalein, employed as a cathartic, causes lesions resembling erythema perstans.

The cutaneous reactions caused by drugs and chemicals applied externally will be dealt with in the chapter on dermatitis. They are entirely due to the local influence of irritants and these may either cause destruction of tissue as in the case of strong acids and alkalies, or a catarrhal reaction in the predisposed, as in the case of substances which are ordinarily not harmful.

THE EXANTHEMATA

The exanthemata are acute infectious diseases of which cutaneous manifestations are an important clinical feature. They may be divided into two groups in the first of which the skin and systemic reactions share equally in prominence; in the second of which the eruption is transitory, subsidiary, but characteristic. The first of these two groups may be subdivided into two classes: erythematous and vesiculo-bullous. The erythematous are measles, German measles, scarlatina and Duke's disease; the vesiculo-bullous are smallpox, varioloid, vaccinia and chicken pox. The second group includes typhoid fever, typhus, Rocky Mountain spotted fever, cerebrospinal meningitis and sepsis. Many of these diseases have an enanthem; that is, mucous lesions of definite character best seen in the buccal orifice. At this point it is well to remember that although whooping cough and mumps produce no enanthem they are often accompanied by mouth lesions. It will be impossible to describe these diseases in such detail as would be required in works on internal medicine or pediatrics, so that only those features will be referred to which are of meaning to the dermatologist. Their therapy will not be discussed, as dermatologists are rarely required to treat them.

MEASLES

Synonyms. Morbilli, Rubeola; German, Masern; French, Rougéole.

Definition. Measles is an acute infectious disease characterized by systemic symptoms, nasopharyngeal catarrh and a characteristic exanthem.

Symptoms. Measles begins gradually with malaise, a catarrh of the nose, throat and larynx, bronchitis and conjunctivitis. Thus, the eyes water, the patient resents light, sneezes and coughs, and shows a general bleariness similar to that imparted by a cold in the head. Chills or chilliness, followed by fever which may reach 104° or more, and a variable degree of prostration and drowsiness are present at the onset. During this period prodromal, urticarial or erythematous rashes appear, and an enanthem is almost constant. The latter is of two types; an erythema of the tonsils, fauces, pharynx and hard palate, in the last named region often taking the form of puncta; and the so-called Koplik's spots. These spots accredited to Koplik who described them in 1896, were first noted in the literature by Filatow a year earlier, but actually Flindt had observed them many years before either. They are bluish white minute dots on the mucosa of the cheek, prevailingly opposite the molars, and may be observed in about ninety percent. of all cases two days before the appearance of the eruption. They may also occur anywhere else in the mouth. They are gone by the time the exanthem sets in.

About four days after the onset the characteristic rash is first noted, and it reaches full bloom in forty-eight hours more. During this period the fever and catarrhal symptoms are most marked. The forehead and temples are first affected, then the face, neck, trunk, upper and finally lower extremities, in the order mentioned; even the hands and feet are covered with a disseminated, symmetrical erythema. Upon close study the exanthem is found to be composed of dusky, red, yellowish-red, or somewhat violaceous macules, grouped either in no particular order, or in segments of circles. In their midst areas of normal skin show through. The lesions are flat, or slightly elevated, one millimeter to a centimeter in diameter, and disappear on pressure. At times, too, there may be veritable papules, some of which feel shotty. The face is slightly edematous. General glandular swellings often occur.

Course. The onset is relatively gradual, the fever rising slowly and reaching its fastigium on the fourth day, beginning its descent on the fifth, and becoming normal by the sixth or seventh, but occasionally not until the tenth or twelfth. On the sixth day the eruption starts to fade, beginning on the face and proceeding in the order of its evolution. Desquamation accompanies this process.

The scales are always fine and branny, and cease to form, as a rule, after about a week, but may remain for ten or twelve days.

Varieties. Morbilli may be mild, severe or hemorrhagic, the first two depending upon the height of the fever and general symptoms; the last being caused by minute cutaneous hemorrhages into the lesions. This form is also called black measles, is rare, severe and often fatal.

Complications and Sequelae. The commonest complications are pneumonia or bronchopneumonia, while otitis media, renal and cardiac diseases rarely develop. Noma, erythema nodosum, lupus vulgaris, impetigo, erysipelas and furunculosis at times follow measles. At times, too, pulmonary tuberculosis is a sequela. Both the complications and sequelae vary in different epidemics.

Diagnosis. In the prodromal stage the coryza, photophobia, general symptoms and cough are suggestive, while the presence of Flindt's (i. e., Koplik's) spots is pathognomonic. These spots must be differentiated from aphthae, painful, small, yellowish ulcers surrounded by a fine red areola, usually seen on the mucous surface of the lower lip and tip of the tongue on both of its surfaces. Aphthae have nothing to do with measles and are probably a form of herpes. Later on scarlatina, German measles, Duke's disease, typhus and the early stages of variola, particularly when papules are present, must be ruled out. (See these diseases below.) Multiform erythema at times resembles measles. The absence of fever and Flindt's spots, and the comparative health of the patient exclude this disease, and these points, plus the history of drug ingestion, aid in the diagnosis of the copaiba rash.

Etiology and Pathogenesis. Measles is a disease of childhood. It may occur in the first year of life, in which respect it differs from scarlatina which is rarely seen before the third year, and almost never in nurslings. The contagion is unknown; exposure must be direct and infectiousness persists for three weeks after the onset. The average incubation period is eleven to fourteen days and the disease confers permanent immunity.

Prognosis. Uncomplicated measles is not a serious disease. Through the pneumonia which may accompany it and the tuberculosis which may follow it, however, the mortality is raised enormously, and, as Osler states, it ranks third in death rate among the eruptive fevers. In institutions for young children Holt mentions that the mortality varies from fifteen to thirty-five percent., while the average mortality is from four to six percent.

GERMAN OR FRENCH MEASLES

Synonyms. Rubella, Rubeola. Epidemic Roseola; German, Rötheln; French, Rubéola.

Definition. Rubella is an acute epidemic exanthem having some of the characteristics of measles, and some of scarlatina, which runs a short, mild course.

Symptoms. The disease appears after a few hours of illness, occasionally with a mild nasal catarrh, or without prodromata, the first symptom being the rash, with the appearance of which the temperature reaches 100° to 101°. The rash starts on the face, spreads to the neck, torso, upper and finally lower extremities. It is either morbilliform or scarlatinoid in character, bright or dusky red, itchy, and occasionally papular, but usually macular. It is symmetrical, disappears on pressure, and at times the papules feel shotty. At the height of the exanthem the post cervical glands are usually markedly enlarged.

Course. After a vague prodromal period, or without any, the rash appears with a low fever. These last a day or two, the rash covering the body within twenty-four hours and fading in the order of its evolution. Slight, fine or coarse desquamation follows; or at times none at all. When present, the scaling is complete within a week.

Varieties. Exceptionally the disease is severe, the temperature reaching 104°.

Complications and Sequalae. There are none.

Diagnosis. Sporadic cases are almost impossible to recognize with certainty. In epidemics the diagnosis is easy. Measles and scarlet fever may be confused with it at the onset; but the severer systemic symptoms of both, combined with the catarrh and Flindt's spots in measles, and the angina in scarlet, in addition to their characteristic subsequent course, should render any doubt of short duration. Forchheimer in 1898 described an enanthem in rubella, consisting of minute, red puncta on the uvula and soft palate, which are supposed to be present in the first twenty-four hours.

Etiology and Pathogenesis. The contagion is not known but is transmitted by direct contact, mostly during the five days following the onset. The incubation period is about three weeks. Immunity is apparently permanent.

Prognosis. Rubella is never fatal or even serious.

DUKE'S DISEASE

This ill defined syndrome, also known as the fourth disease, was originally described by Duke and has not been accepted as a clinical entity. It is mild, accompanied by a fever of about 101°, slight glandular enlargements and an exanthem. The rash appears suddenly, rapidly involves the whole body, resembles that of scarlet fever, but is a more vivid red, lasts a day or so, and fades without marked scaling, if any at all. The period of incubation is given at from nine to twenty-one days.

SCARLATINA

Synonyms. Scarlet Fever, Scarlet Rash; German, Scharlach; French, Scarlatine.

Definition. Scarlatina is an acute infectious disease characterized by an exanthem, fever and angina, and a severe systemic reaction.

Symptoms. A sudden onset with chills or only chilly sensations, in adults, and convulsions in children, sore throat, severe headache, vomiting and a sharp rise in temperature to from 102° to 105° characterize the beginning of scarlatina. Within twenty-four hours a rash breaks out and the skin itches. In a typical case the patient is either apathetic or agitated, but dull, the cheeks are flushed, the ears red and edematous, the eyes bright and the area about the lips pallid, the pallor intensifying the flush of the cheeks. On the neck and chest are seen minute, deep red puncta, some macular, others slightly raised against a lighter, diffuse scarlet background. If the upper extremities be held so that the elbow flexure and backs of the fingers may be seen together, the former is found covered with a rash like that on the neck, the latter with an erythema studded with minute elevations, reddish or whitish in color, and resembling tiny miliaria, but actually being true vesicles, the pathology of which has been excellently studied by Egon Rach of Vienna. A scarlet enanthem covers the pharynx. The entire buccal mucosa is edematous, the tonsils enlarged, at times enormously so, and occasionally covered with dull grey or yellow superficial ulcerations. These findings account for the pains in the throat and occasional dysphagia. The tongue is furred and the swollen red papillae projecting through the coating give this organ an aspect which justifies the term "strawberry" tongue. The fever is high. All of these symptoms and signs are referable to the first day or so of scarlatina. Within an-

other day the body is entirely covered with the eruption which maintains the general features seen in the early stage on the neck. The throat is worse, the general discomfort, dysphagia and prostration more intense, and the fever high with slight morning remissions. The cervical glands are enlarged. During this period the tongue loses its coating, becomes generally red and glistening, but the papillae remain swollen. Thus the tongue roughly resembles a raspberry. The rash starts to fade, desquamation sets in, the scales being furfuraceous, coarse or laminated, and at times actual epidermal casts of the fingers, toes, hands and feet are thrown off. A characteristic of the rash at its height is its disappearance on pressure, the ischemic area thus left being not white, but yellowish-brown, pale, or subicteric in color.

Course. The onset is sudden, the rash fully developed within two to three days, and at this time, or at the most within a week, it commences to fade. Scaling begins and is usually complete within three weeks from the beginning of the disease, the process ending on the palms and soles. At times six weeks are required for desquamation. The fever usually falls with the involution of the rash, and the patient goes on to recovery unless one of the numerous complications arise.

Varieties. There is a milder form of the disease in which the rash remains limited to the neck; a form in which only throat symptoms are present; or a pharyngeal form in which three weeks later nephritis develops. The last two are called scarlatina without exanthem. A variety is known with severe central nervous symptoms, delirium, stupor, restlessness, headache, convulsions and hyperpyrexia. At times the rash is hemorrhagic. A form called surgical scarlatina has its starting point about wounds, but otherwise runs the usual course. Finally, there is a fulminating variety which ends fatally from intoxication, within a day or two of the onset.

Complications. During the eruptive period albuminuria with casts is often present. This is toxic in origin and not serious. After the third week, however, true nephritis, at times severe and causing uremia, may appear. This usually clears up, but frequently becomes chronic. Otitis media, mastoiditis, suppurative cervical adenitis, articular rheumatism, endocarditis and rarely, chorea follow scarlet. Occasionally, diphtheria or varicella are complicating diseases. Immunity is usually conferred by one attack, but recurrences, though rare, have been observed, as have been relapses and re-infections.

Diagnosis. The diagnosis is easy in typical cases. The sudden onset with chills in adults and convulsions in children, the throat symptoms, tongue and rash are all sufficiently distinctive. During the early stage a leukocytosis is present, an eosinophilia, and in the polymorphonuclear leukocytes, Döhle's bodies, originally regarded by him as the cause of scarlatina, are found. These are not pathognomonic, but never occur in toxic or drug rashes, and are thus of corroborative value. Scarlet must be differentiated from measles, Rötheln, toxic rashes, drug rashes and the scarlatinoid erythrodermas. In all of these conditions either the abrupt onset or throat symptoms are wanting. In measles, the catarrah and Flindt's spots are of value; in Rötheln, the onset is mild; in toxic rashes, one of the numerous causes of these may be ascertained; in drug rashes, a history of medication may be elicited and Döhle's bodies are absent. In the scarlatinoid erythrodermas the pharynx is rarely involved, the systemic symptoms are mild, and subsequent attacks are frequent.

In the first ten days of the disease the Wassermann reaction is at times present. This was once thought to be of significance, but did more to bring the Wassermann test into disrepute than to explain or help to diagnosticate scarlet, and after all it possibly only indicated that the case was one of scarlet in a syphilitic. Koessler did some unconvincing work on complement fixation tests in scarlet using the glands of scarlet patients as antigen. Bela Schick of Vienna regarded as a diagnostic point in the early stages the ease with which hemorrhages could be produced by lightly pinching the skin. The author has found it an equally prevalent sign in all exanthemata, whether infectious or toxic. This is in line with Leede's observation that a tourniquet above the elbow produces petechiae below the constriction in scarlet fever patients. Anyone who has administered arsphenamin or taken blood from the veins at the elbow flexure must have observed this purely mechanical phenomenon in all people. In the last analysis the diagnosis of scarlatina rests upon a knowledge of its clinical features.

Etiology and Pathogenesis. About twelve years ago Mallory described the cyclasterion scarlatinale, an alleged protozoon presumed to be the pathogenic agent of the disease. It is a body found in the malpighian cells of the epidermis, and is either an artifact or a cell degeneration. The streptococcus, too, has been regarded as a causative factor. There are no good grounds for this, and the theory, from what we know of streptococci, seems unlikely, since these organisms are not known to confer immunity, an experience to which

scarlet would be an exception. Unquestionably, however, the streptococcus causes many of the purulent complications and sequelae of the disease. Children seem more subject to the malady than adults, but this is due to exposure in schools and institutions, and a large proportion of adults who have escaped the disease in childhood, get it when exposed later in life. Infants under a year old are rarely infected. The contagion must be in the secretion from the nose and throat, as the communicability of the disease is greatest during the anginal stage. The belief in the infectiousness of the scales has been practically abandoned. Even children whose desquamation is not complete are now allowed to associate with their fellows, provided the throat is well. Transmission through a third person is rare, but through infected foods such as milk is common, and this mode of conveyance has accounted for many scourges. The infection of the foods probably takes place through contamination with pharyngeal ejecta of the diseased. Sporadic cases of scarlet are not infrequent, but the disease is prevailingly an epidemic one. The incubation period is from three to five days, the average being four.

Prognosis. Scarlatina is a grave disease, partly because of the numerous severe uncomplicated forms, partly because of the many serious complications. In children under five years of age the mortality rises to thirty percent.; in older children and adults the average mortality is close to ten percent., but this varies with the epidemic, the extreme limits being from three to forty percent.

VARIOLA

Synonyms. Small Pox, the Pocks; German, Blattern, also Pocken; French, Petite Vérole.

Definition. Variola is an acute eruptive fever running a definite course divided into two stages, and characterized by severe general symptoms and a vesiculo-pustular eruption.

Symptoms. The invasion in small pox is sudden. Severe frontal headache, pain in the back and limbs, a chill or rigor, and in children, convulsions, and an abrupt fever of 103° or 104° distinguish the onset. Vomiting and delirium are frequently seen. The skin is dry, the face flushed, eyes bright, expression anxious, and the patient is restless. A morbilliform or scarlatinoid rash, often generalized and occasionally hemorrhagic appears. Its localization may be anywhere on the body, but its favorite sites are the lower abdomen, inner aspect of the thighs, the flanks and axillae. The first

stage lasts three or four days, when the temperature falls, the systemic symptoms vanish, the early rash fades and the true rash appears. On the seventh day the fever begins again to rise, reaching its maximum on the ninth or tenth day with the height of the new eruption's development. The second stage is now in full swing. During this period the eruption is either discrete or confluent. Both forms begin at the hair line of the forehead and on the wrists as small red papules.

The Discrete Variety. Within a day the papules spread to the rest of the face and extremities, particularly the palms and soles, rarely involving the trunk in profusion. They feel shotty, become vesicles on the fifth or sixth day, and present a depression or umbilication. On the eighth day they turn into pustules, the umbilication disappearing. They are yellow or gray and surrounded by a deep red areola of inflammation. It is at this point that the second rise in fever, mentioned above, takes place. The changes in the lesions occur first on the face, and then elsewhere in the order of their evolution. Edema is caused by the inflammation about the pustules, and since the latter are numerous upon the face, it becomes swollen and distorted. By the eleventh day the second fever drops, the pustules rupture and crust, and convalescence sets in. In the discrete form scarring rarely occurs. Vesicles often are seen in the mouth, and even the larynx may be involved.

The Confluent Variety also begins with isolated papules as mentioned, but they are more numerous than in the discrete form and when the pustules develop they run together so that the face is virtually a diffuse pustule. Coalescence is never seen on the body. Otherwise, the course follows that of the discrete type, save that the fever is higher, the mortality greater, the swelling of the face more marked, and the prostration worse. Salivation and cervical gland swellings are frequent. Desiccation begins on the twelfth day, and the process of scarring is complete in three or four weeks. The last sites to return to normal are the palms and soles on which the pustulation is nearly as profuse as on the face, and the healing process delayed because of the depth of the epidermis overlying the lesions. Depressed cicatrices remain. A leukocytosis is present in variola.

Course. The disease begins abruptly. After an incubation period averaging nine days there is a rise in temperature accompanied by an erythema which fades rapidly, to be replaced by the pustular exanthem, at the height of which the fever again rises after a brief normal period. The patient gets well or dies, subject to the

variety and severity of the infection, or the incidence of some of the fortunately infrequent complications. The entire course of a severe infection which leads to recovery is complete within four weeks.

Varieties. There are two main varieties of small pox, the true and the hemorrhagic. The true is divided into the discrete and confluent already described; the hemorrhagic into two varieties, the purpura variolosa, and variola hemorrhagica pustulosa.

Purpura variolosa is the black small pox. It begins as ordinary variola, but in three days a hemorrhagic eruption appears starting with an erythema, particularly in the groins. The face is enormously swollen, the palpebral fissure almost being obliterated by the edema. The conjunctiva and the buccal mucosa are the site of hemorrhages, and bleeding may occur on other mucous surfaces, the blood appearing in the urine, sputum, stools, vomitus, etc. In some cases the skin is actually purple from subcutaneous bleeding. These cases are fatal as a rule.

Variola hemorrhagica pustulosa is ordinary small pox in which hemorrhages occur into the pustules. The purpura variolosa is usually fatal before the pustular stage develops.

Complications. Laryngitis and bronchopneumonia are frequent complications. Formerly, iritis and keratitis were often observed, but these untoward results are rare now since the hygiene of the eyes in small pox has been improved. After confluent variola the skin is pitted.

Diagnosis. In the first stage measles and scarlet must be excluded. Flindt's spots, the coryza and the milder systemic symptoms of measles, the presence of angina and the strawberry tongue of scarlet, the peculiar distribution of the early exanthem of variola, and the severe systemic symptoms are the factors on which must be based the differential diagnosis. In the period of the second rash small pox must be differentiated from varicella, acute pemphigus and the varioliform secondary syphiloderm. Varicella occurs mostly in children, the symptoms are milder, the eruption comes in successive crops, papules and pustules appearing together. Pemphigus often begins with severe systemic symptoms, but the lesions are blebs arising prevailingly upon non-inflamed skin, and are not umbilicated. The syphiloderm in question is of gradual onset, longer duration, accompanied by milder symptoms, general adenopathies, and the Wassermann reaction is present.

Etiology and Pathogenesis. The nature of the infectious agent

is unknown. That it is present in the secretions, in the vesicle contents, pustules, crusts and fomites is undoubted. Lady Mary Whortley Montague's inoculation and the work of Jenner proved its transmissibility, as well as the prophylactic value of milder attacks produced by the human or bovine virus which must be similar if not, indeed, identical. The prevention of the disease through vaccination, the mild course of the disease in the few vaccinated individuals who sustain subsequent infections, the practical elimination of variola in districts in which vaccination laws are strict, arouse astonishment at the fanaticism of those who are opposed to this simple and harmless form of prophylaxis. The contagion has not yet been discovered, although we know exactly where it is situated, but we do know that its virulence may be ameliorated by vaccination. Sex, age, race, even general health have no influence upon the incidence of the disease. Although variola is an epidemic disease, it smoulders in foci throughout the world. The high fever and general symptoms in the pustular stage may, in part, be accounted for by secondary infection with pyogenic bacteria.

Prognosis. The mortality varies from twenty-five to thirty-five percent. in different epidemics. Confluent variola and purpura variolosa are the severest forms, and these as well as hyperpyrexia, and laryngitis, account for the greatest number of deaths. Early death in variola usually occurs on the third day; late death on the eleventh or twelfth.

VARIOLOID

Varioloid is mild small pox modified by previous vaccination. An abrupt onset characterizes the disease, with general symptoms resembling those of small pox. The papules which rapidly become pustules appear on the third day, the temperature which may rise to 103° falls when the rash appears. The entire course of the disease is about ten days. Pitting is rare. Varioloid, when transmitted to the unvaccinated, causes true small pox.

VACCINIA

This is the small pox lesion artificially induced by vaccination. Its characteristics are too well known to need mention in this book. At times auto-inoculation, as mentioned in connection with dermatitis medicamentosa, may cause vaccinia pustules to develop elsewhere in the body.

VARICELLA

Synonyms. Chicken Pox; German, Windpocken, Wasserpocken, Spitz Blattern; French, Variolette.

Definition. Varicella is an acute eruptive disease the cutaneous lesions of which develop at the onset and recur in crops.

Symptoms. The onset is sudden, the fever rising as high as 104° at times, usually, however, to 100° or 101°. Frequently the disease is afebrile. A certain amount of malaise is present. The rash appears at once, starting on the face, involving the extremities and trunk to a variable extent. The conjunctiva and buccal mucosa are sometimes included in the eruption; the palms and soles never. The first lesions are shotty papules which rapidly become umbilicated vesicles, the size of a pea and surrounded by a pink areola. In a day or so after their appearance they become turbid, and in another day begin to desiccate. Successive crops appear, and thus the eruption has a polymorphous character, the elements of which are papules, vesicles, pustules, crusted lesions and pink macules where the crusts have fallen off. Scarring is rare. The temperature lasts a day or so, as a rule, and in a week or ten days the patient is well.

Diagnosis. The two important conditions from which varicella must be differentiated are variola and impetigo contagiosa. Unless the systemic symptoms of varicella are severe, no confusion between this condition and small pox should arise. The absence of the prodromal rash, backache, headache, etc., serve to rule out variola. Impetigo contagiosa is a pustulo-bullous eruption in which the bullae are infrequently seen because of their delicate structure, which causes them to rupture early. At their site a honey-yellow crust develops, unless the lesions are very much irritated and bleed, in which case the crust becomes brown. The bullae when present are never umbilicated. Thus, during the bullous stage no confusion between impetigo and varicella is possible. It is only during the stage of desiccation that there may be any doubt. But the absence of papules and the presence of some associated underlying cause of impetigo, such as pediculosis, scabies, urticaria, as well as the absence of fever and systemic symptoms, should clear up the problem.

Etiology and Pathogenesis. The cause of the disease is unknown, but children are more frequently attacked than adults. The incubation period is ten days or so.

Prognosis. The prognosis is invariably good.

In discussing the group of diseases with exanthems in which the latter are secondary considerations, only the cutaneous lesions and such other points as are of importance etiologically or diagnostically, will be mentionel. Brevity demands the exclusion of general clinical descriptions; nor do these belong in a work on skin diseases.

TYPHOID FEVER

The characteristic rash of this disease is a roseola which appears between the seventh and tenth days. It starts upon the abdomen and back and consists of a few isolated lesions, or they may be very numerous and occasionally occur elsewhere on the body, particularly on the arms and thighs, less frequently on the forearms and legs, and almost never elsewhere. The individual lesion is a minute, hyperemic, slightly raised macule, which disappears upon pressure, and has a faint rose to a dark red shade. When very extensive and dense the rash somewhat resembles measles. The raised spots occur in successive crops, a few in each group until the period of defervescence. New eruptions appear with recrudescences or relapses of the disease. At times the spots are hemorrhagic. Occasionally desquamation follows the exanthem, but as all prolonged fevers are followed by dryness of the skin and hence, scaling, the significance of this process in typhoid cannot be accurately interpreted.

Diagnosis. The general points upon which the diagnosis rests are the history of the case, the general clinical symptoms and signs of typhoid, the positive blood culture in the early stages of the disease, the Widal reaction later on, and the leukopenia and relative lymphocytosis seen in typhoid. Measles, the typhus exanthem, and the syphilitic roseola resemble the typhoid rash. Measles may be ruled out by the absence of coryza, Flindt's spots, and the presence of the positive signs of typhoid. The differentiation from typhus will be considered below. The syphilitic roseola is ruled out by the absence of the Wassermann reaction and the positive signs of syphilis.

Etiology. This rash may be due to cutaneous emboli of typhoid bacilli, for it appears only in the bacteremic stage of the disease, or as a local cutaneous reaction to the bacteria or their toxins, representing an allergic phenomenon.

Other Rashes Seen in Typhoid. During the febrile stage miliaria, later on furuncles, may be observed, and after prolonged typhoid, bed sores are encountered. Post febrile alopecia is frequent, and the nails become transversely ridged, as they do however after all prolonged fevers. The absence of herpes in typhoid is noteworthy. On the fauces Bouveret's ulcers appear.

TYPHUS FEVER

The onset is abrupt with a rigor, chills, chilliness, headache, pains in the back and legs, prostration, bronchitis, and a fever often of

105°. After from three to five days the eruption appears on the abdomen, spreading thence to the chest, face and extremities. It consists of two elements: a diffuse blush and a roseola somewhat darker, which rapidly becomes hemorrhagic. The skin is dry. There are associated delirium, prostration and the general symptoms of typhus. The rash fades after a week and desquamation begins after three weeks. The systemic symptoms last for a fortnight when the temperature falls critically.

Diagnosis. This disease may be confounded with typhoid, measles and black small pox. The positive signs of typhoid, the sudden onset of typhus, the absence of the Widal reaction, and the negative typhoid and positive typhus blood culture and serum reaction, should suffice to prevent error. When the typhus rash is not profuse it may resemble measles. Here the prodromata of the latter disease, the buccal spots, and the severer onset of typhus with its characteristic fever curve and blood findings, are the diagnostic points to be emphasized. Confusion with variola is possible only for the first few days before the appearance of the pustules.

BRILL'S DISEASE

This disease was originally described by Nathan Brill and regarded as due to auto-intoxication until Plotz in 1914 isolated a bacillus which he found also caused typhus, and by a series of excellent investigations established the identity of these two diseases. The syndrome is distinctive. After a brief period of malaise, associated with severe headache and constipation, the fever rises abruptly to from 103° to 105°, and a rash develops which is seen on the belly, back, chest and extremities. It consists either of a few dusky rose spots or a morbilliform eruption on a dusky red background. The fever drops critically in a week to ten days, the rash fades and the patient is well. Body lice are associated with the disease, and the bacillus gains access to the host through the parasite's bite. The disease must be differentiated from typhoid and measles along the lines laid down in connection with typhoid and typhus.

ROCKY MOUNTAIN SPOTTED FEVER

This disease is endemic in the Rockies, in Oregon, Montana, Utah and Idaho. It begins suddenly in the Spring, with severe general symptoms, a sharp rise in fever to 105° or 106°, which remains high for about twelve days and gradually declines. At the onset a rash appears on the wrists, back and ankles. It extends gradually, resembling a toxic purpuric erythema. A diffuse yellowish background sets off the eruption. From four

to ten percent. of the patients die. Recovery in favorable cases is complete in three weeks.

CEREBROSPINAL MENINGITIS

This disease is also known as spotted fever from a punctate rash found in many cases. The eruption is usually roseolar or purpuric, and covers the face, particularly the ears, during the first twenty-four hours of the disease. The diagnosis is made by the concomitant signs of meningitis, and the discovery of the meningococcus in the spinal fluid.

SEPSIS

In sepsis and bacterial endocarditis, usually when caused by one of the numerous strains of streptococci, various eruptions are seen. These are petechial, toxic or erythematous in character, and occasionally purulent. The petechiae may be found anywhere, but favor the wrists, ankles, conjunctiva and buccal orifice. They are minute, bright red spots which persist upon pressure and rapidly turn brown and fade. They appear in successive crops. The erythemas are due to bacterial toxins. The pyodermas are due to emboli which cause local, usually subcutaneous suppurations. Erythema nodosum is sometimes observed. The diagnosis depends upon a positive blood culture, as well as the clinical signs of sepsis, such as the fever curve and cardiac involvement.

FOLLICULAR TONSILITIS

Not infrequently a faint scarlatinoid rash appears on the neck and chest in the first days of follicular tonsillitis, particularly when marked systemic symptoms are present and high fever. To be absolutely certain that the case is not one of scarlatina is impossible. Frequently enough cases of this type get apparently well in a day or so, and three weeks later an acute nephritis develops. Although there are no certain grounds for a differential diagnosis between scarlet and the tonsillar erythema, the fact remains that the majority of these cases are not scarlet. Nevertheless, the greatest circumspection is necessary both as to diagnosis and prognosis.

PERTUSSIS

The effort of coughing causes a rupture of the minute vessels in the mucosa below the togue, on the lips, and in the conjunctiva. The result is the formation of petechiae.

MUMPS

Before the swelling of the parotid glands, thus during the prodromal stage of mumps, a small red circle is seen on the buccal mucosa opposite the upper molars. This corresponds to the opening of Stenson's duct, and is caused by a swelling and inflammation about its aperture. It is of value

in foretelling beginning cases during an epidemic. I have once seen this phenomenon in a child at the onset of glandular fever.

ENCEPHALITIS LETHARGICA

Herpes simplex of the face has been observed.

PURPURAS

Purpura is an eruption due to hemorrhages into the skin. Lesions varying from punctate to coin size are called petechiae; large, irregular ones are ecchymoses; when linear in arrangement, either through linear trauma, or by an accident of distribution, vibices are formed; and when bleeding takes place into vesicles or bullae, these are qualified by the adjective hemorrhagic. At times, blood collects into large masses the size of a nut, or even greater, and such accumulations are termed ecchymoses. Purpuras fall into three classes,—mechanical, symptomatic and primary.

Mechanical Purpura. This form is due to some obstruction in return circulation. Thus, during or after obliterating phlebitis, particularly of the femoral vein, purpuric outbreaks occur below the level of the clot, and are increased by overtaxing the compensatory circulation. In connection with varicose veins, particularly of the lower extremities, a similar condition is seen, partly due to endophlebitis and partly to a fragility of the cutaneous veins. Thus, associated with dermatitis and leg ulcers, petechiae and even ecchymoses are observed.

Symptomatic Purpura. In the course of infectious diseases (see Exanthemata), intoxications (see Drug and Toxic Rashes and Multiform Erythema), and in cachectic diseases such as leukemia, tuberculosis, tabes, Hodgkin's disease, marasmus, etc., purpuric outbreaks occur.

Primary Purpura. There are many varieties of this disease as the following table indicates:

I. Arthritic:
   1. Purpura Simplex.
   2. Purpura or Peliosis Rheumatica.
   3. Henoch's Purpura.

II. Purpura Hemorrhagica.
III. Purpura Neonatorum.
IV. Hemophilia.
V. Scurvy.

Purpura Simplex is more frequent in children than in adults. It is usually, but not always, febrile; arthritis is frequently but not invariably present. The spots appear in crops, are petechial, favor the legs, and are at times seen on the trunk and upper extremities.

Purpura Rheumatica, also known as Peliosis Rheumatica and Schönlein's disease, is sudden in its onset. It starts with pharyngitis or tonsilitis, and is usually seen in males in the third decade of life. Multiple arthritis, fever, and a certain degree of prostration accompany the disease. The lesion is not purely purpuric, but admixed with urticarial, multiform erythematous and nodular erythematous elements, as well as hemorrhagic bullae and vesicles. The eruption tends to localize itself about the affected joints but may occur anywhere, and the angioneurotic lesions favor the eyelids and face.

Henoch's Purpura. This disease has several marked characteristics besides the cutaneous lesions which are either purpuric or those of erythema multiforme. They have no definite localization. Relapses are frequent. The other features are arthritis, mucous hemorrhages and gastro-intestinal crises, attended by colic, vomiting and diarrhoea. The visceral crises are of great diagnostic importance. Cholecystitis, and particularly appendicitis, are simulated, and many an appendix has been removed from people suffering from Henoch's purpura. Hemorrhagic nephritis occasionally follows this disease.

Purpura Hemorrhagica is also known as morbus maculosus of Werlhof and is generally seen in young and delicate girls. After a few days of malaise the purpura breaks out in the form of petechiae and ecchymoses. Nose bleeds, hematuria and hematemesis develop. At times so severe a secondary anemia is brought about as to be fatal, and occasionally cerebral hemorrhages occur which almost invariably cause death.

Purpuras in the Newborn are due to syphilis, or to an unknown cause. In the former type, the child may seem healthy at birth. In any case, shortly after this event cutaneous and mucous hemorrhages and bleeding from the stump of the cord appear, and the infant becomes jaundiced. A great number of these patients die. The second type is known as morbus maculosus neonatorum, and is always fatal. It begins during the first few weeks of life with cutaneous hemorrhages, and bleeding from the nose, mouth, cord, intestines, kidneys and lungs.

Hemophilia. This is an hereditary disease, transmitted to males through females, and the Mendelian law, as shown by Ottenberg, governs its distribution in the family. Hemophiliac males, if they marry sound women, do not transmit the disease to their sons. Females born of hemophiliac males may transmit the bleeding tendency to their sons, but do not invariably do so. Bleeders are found almost wholly among Anglo-Saxons and Teutons, but no race is absolutely immune. Although the commonest symptoms are profuse bleeding from the mucous membranes, or from trifling injuries,

arthritis of the knees and elbows has been frequently noted, but even the smaller articulations may be involved.

Scurvy. This disease, also called Scorbutus, occurs in two forms, the infantile and adult. In infantile scurvy, or Barlow's disease, tenderness and swelling of the bones, chiefly the femurs, ecchymoses about the joints and hemorrhages in the pharynx are observed. The children seem to be paralyzed and although the involved bones do not hurt spontaneously, they are exquisitely tender. The swelling and tenderness are due to subperiosteal and epiphyseal hemorrhages.

Adult scurvy begins insidiously with weakness, swollen and bleeding gums, hemorrhages into the buccal mucosa, ecchymoses on the arms and trunk, largely near the hair follicles. The lesions may also be petechial. Subperiosteal hemorrhages with swelling and tenderness are common. Bleeding from various mucous surfaces may occur.

Etiology. The causes of purpura are numerous. The arthritic forms are associated with rheumatism. The etiology of Purpura Hemorrhagica is not known. One form of purpura neonatorum is due to syphilis; the origin of morbus maculosus neonatorum has not been ascertained. Hemophilia is hereditary in the scientific sense of the word. Scurvy is caused by faulty diet, specifically the lack of vitamins in fruits, fresh vegetables and potatoes.

Therapy. The arthritic forms should be treated as rheumatism. The syphilitic variety of the newborn must be treated as syphilis. Hemophilia is best controlled by the transfusion of blood, or at least the intravenous or subcutaneous injection of suitable human sera. Emsheimer has successfully treated Werlhoff's disease by intramuscular injection of whole blood. Scurvy yields rapidly to fruits, potatoes and legumes, followed by tonics. Local treatment is useless.

Prognosis. With the exception of purpura hemorrhagica, purpura of the newborn and hemophilia, the prognosis as to longevity is good, and as to cure is fair. Hemophilia is not necessarily, in fact not frequently fatal, but unless the new methods of treatment prove as promising as is hoped, the outlook for a cure will be as fruitless as in the past. Scurvy is always curable if treatment is timely.

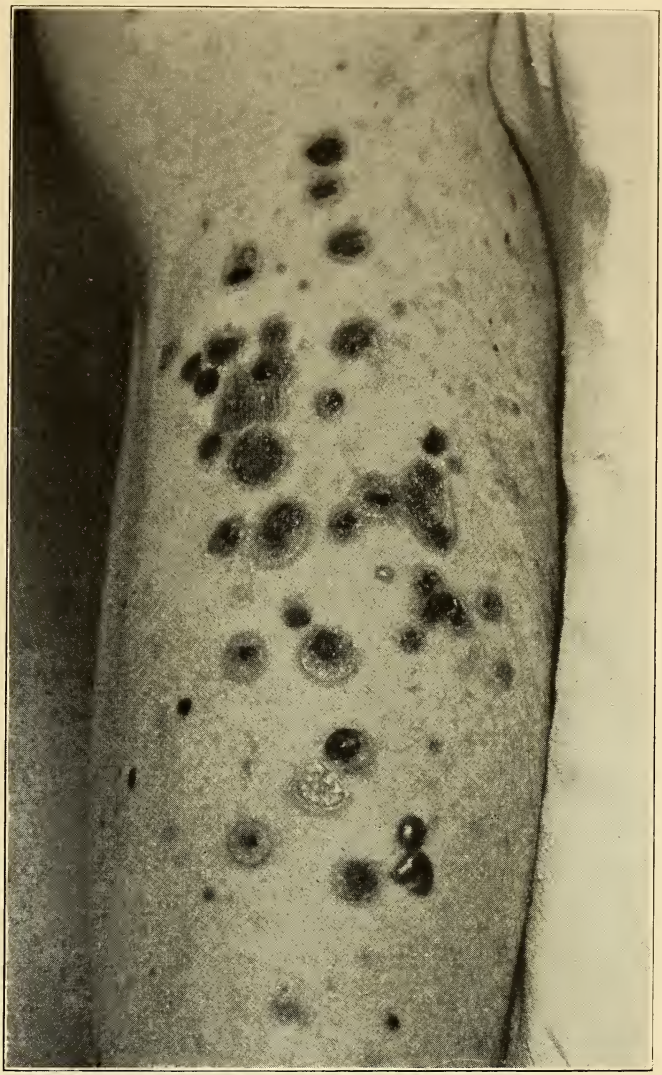

FIG. 6. IODODERMA

This is the bullous type of iodine rash, resembling variola, varicella and pemphigus (Fig. 18). It differs from all of these in the fact that the bullae turn into granulomata, and in the clinical course of the disease. Iodine is found in the urine.

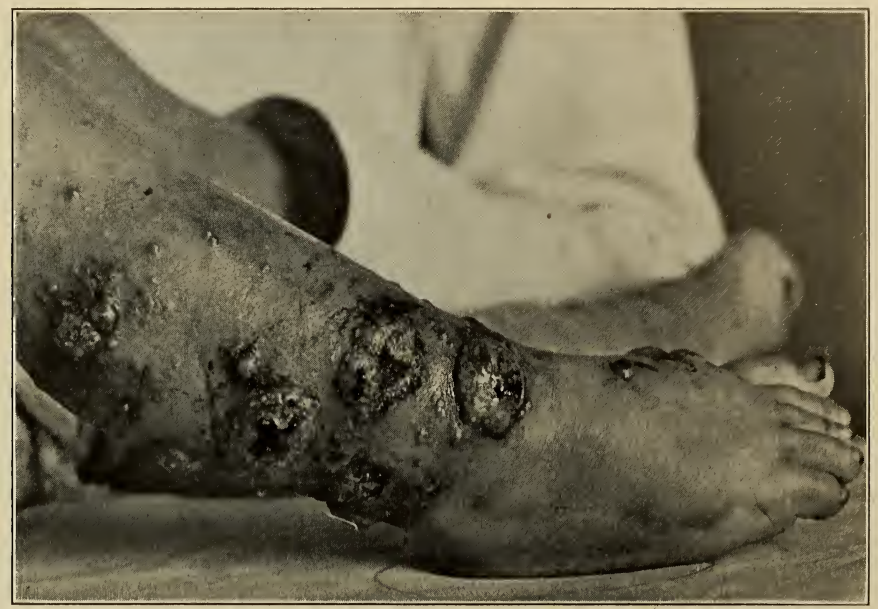

FIG. 7. BROMODERMA (CF. IODODERMA FIG. 9)

Both the bullous and granulomatous lesions are shown; mycosis, blastomycosis, tuberculosis, syphilis and erythema induratum are suggested. The first four are excluded by bacteriological and serological examinations and tuberculin tests. Bromine is found in the urine.

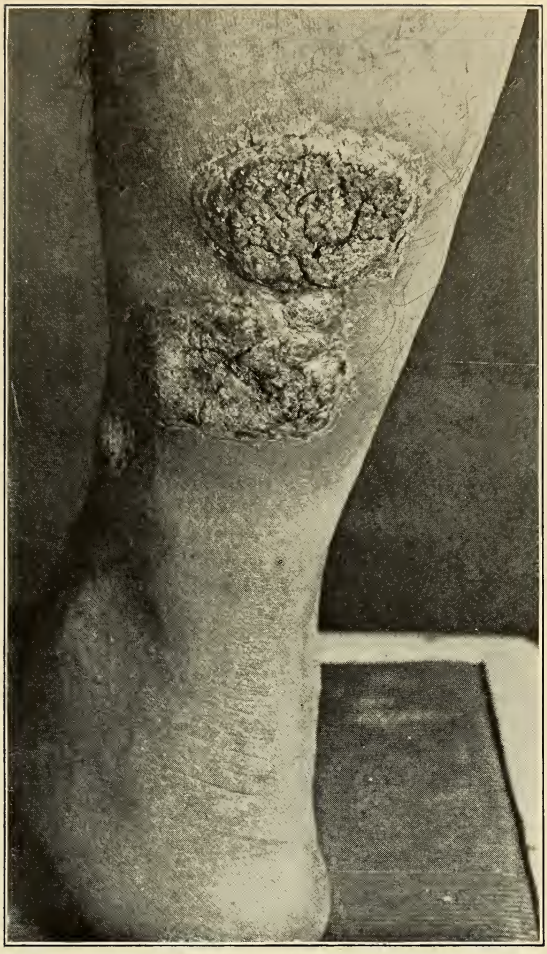

FIG. 8. BROMODERMA TUBEROSUM (CF. FIGS. 8 AND 9)

Cribriform abscesses, crusting and vegetation are the features of these lesions which are brownish or purple in hue, crusted and tender. They look like blastomycosis and sporotrichosis, which bacteriologic study excludes. Bromine is found in the urine.

CHAPTER VIII

DISEASES OF THE CUTANEOUS VESSELS; TELANGIECTASES; GANGRENE

Cutaneous vascular diseases lead to changes which differ according to the size of the vessels involved, the number, and the nature and extent of the involvement. As in other organs, the vessels may dilate, contract, and become occluded, increased or decreased in number. The pathogenesis of these changes is varied; the alterations wrought by them upon the skin are comparatively restricted. Thus, this group of diseases is an example of the relative limitation of the morbid responses the skin is capable of displaying to unrelated causes. In other words, among these diseases the doctrine of cutaneous reactions (see Chapter III) is nicely illustrated.

When capillaries dilate they show through the epidermis as minute streaks reddish or purplish in tone, according to whether they contain aerated or used blood. Such vessels are telangiectatic and the condition is known as telangiectasis. If, in addition to telangiectasis, there be a local extravasation of blood, an element of purpura is added and this, in fading, leaves pigmentation. Telangiectases are produced in a number of ways, as the following table will show.

I. Embryonic anomalies. Vascular nevi (Chapter XXXII).

II. Congenital and Familial Predisposition.

1. Onset of xeroderma pigmentosum (Chapter XXVIII).

III. Regressive Changes. Senile Skin (Chapter XVII).

Minute deep red lesions seen in adults, especially the aged; possibly nevi, the elements of which were congenitally present, the development of which was late. Both of these conditions might well be due to a degeneration of the elastic tissue either within or without the vessels, causing the vascular walls to relax, thus leading to dilatation.

IV. Inflammatory Changes in the Skin.

1. Unknown or undetermined origin

a. Seborrhoea causing telangiectases of the face.

b. Rosacea acting similarly.
c. Morphoea, Scleroderma.

2. Known or presumably specific causes
   a. Acne telangiectatica (tuberculosis?).
   b. Angiokeratoma. (tuberculosis?).
   c. Lupus Erythematosus (tuberculosis?).
   d. Lupus Pernio (tuberculosis).
   e. Pernio.
   f. Associated with Syphilis.

V. Physical Causes
   1. Mechanical Causes: Incomplete obstruction to return circulation, as for example cirrhosis of the liver; pressure of mediastinal, abdominal and pelvic masses; venous thrombosis, in the areas tributary to the large obstructed trunks capillary dilatation occurs in the skin. In the lower extremities even without such causes, the position of the limbs frequently determines the presence of telangiestases seen particularly on the thigh. Gravidity also predisposes to dilatation of the skin vessels.
   2. Actinic Rays: X-Rays, radium, exposure to sunlight (sailors and agriculturists, etc.).
   3. Heat: Burns and Scars resulting therefrom.
   4. Cold: Frost-bite (see Pernio).

VI. Chemical Agents: Acids, Alkalies, Caustics, etc.

VII. Digestive disturbances. Nasal and facial telangiectases from gastritis, the excessive use of alcohol, condiments, rich sauces. Sustained icterus also at times is responsible.

VIII. Chronic valvular disease, congenital or acquired; chronic pulmonary disease. These might also be grouped among the mechanical causes.

IX. Metabolic and ductless gland diseases; vasomotor disturbances; malar flush of myxoedema; exaggeration of rosacea and lupus erythematosus at climacteric.

X. Essential Telangiectasia
   1. Purpura annularis telangiectoides.
   2. A peculiar progressive pigmentary disease of the skin.
   3. Angiokeratoma.
   4. Angioma serpiginosum.

It is the last group with which, in the main, this chapter deals. When diseases are characterized by the meaningless word "essential," it is for the sole reason that we are ignorant of their nature. It would be both more honest and more exact to classify them as maladies of undertermined origin. An apparently clear clinical entity from among this group may be the result of any one of numerous pathogenic forces, and conversely, the same pathogenic forces may provoke different clinical pictures. This all depends upon the phenomenon of cutaneous reactions. Thus, angioma serpiginosum and purpura telangiectatica, in general accounted distinct clinical entities, may in effect not be so, an impression that cannot be escaped after reading *Stokes's* and *Wise's* respective papers on syphilitic telangiectasa and angioma serpiginosum, although it is improbable that these authors intentionally conveyed such an idea. This is not stated to confuse the student, but to prevent the misconception that certain conditions, assumed to be clinically distinct, necessarily are. Before describing these diseases, it will be necessary to devote a few paragraphs to telangiectasis as a whole.

Telangiectases may be minute, barely perceptible, bright red dots, corresponding to dilated capillaries in the papillae. These look like grains of red pepper studded over the skin and are commonly found in angioma serpiginosum, purpura telangiectatica, and *Stokes* described them in his case due to syphilis. They may also occur in any other telangiectatic disease of any nature whatever, for they simply represent the minutest possible vascular dilatation clinically recognizable, and nothing more. In slightly larger areas of vascular disturbances when capillaries in from four to twenty papillae are involved, a tiny crimson or claret colored macule is formed, or if the blood is venous a purplish shade is present. Such lesions may be nevi, or that type of possible nevi which develop in later years over the trunk. These two varieties are situated in the capillary loops.

When the process involves the longitudinal vessels in the various depths of the cutis the clinical manifestations thereof are delicately curved, or tortuous lines varying in color from brilliant crimson to purplish, according to whether the vessel is an artery or vein. Grouping of these vessels may lead to a curious lacy design. Such telangiectases are found in the diseases to be discussed, as well as in nevi, or as the result of inflammation (rosacea, lupus erythematosus, etc.), or from obstruction to return circulation (cirrhosis, cardiac, pulmonary disease, etc.). Often this and the punctate

type are seen together. Not infrequently, also, an anatomical association of the two exists with the punctate element in the centre and the longitudinal lesions radiating thence in a spider-like manner. This forms the so-called nevus araneus. At times the centre is slightly raised. These are rarely true nevi. They usually occur on the cheek, but often elsewhere, and occasionally may be due to trauma such as the sting of an insect. Finally, in a more or less restricted area all of the smaller vessels may be involved. This imparts a general redness to the affected locality, the color of which will be determined by the amount of arterial or venous blood present. A red macule is engendered which appears to be of an even tone until minutely examined, whereupon the component elements may be clearly discerned. At times in the midst of such a surface other telangiectasia may be seen.

Pathologically, these lesions are the result of congenital vascular malformation or relaxation of the vessel walls, hypertrophy of the vessels, or hyperplasia due to any cause whatever, chiefly though to some type of inflammation. A diseased vessel is capable of rupture, or it may otherwise become permeable, so that in connection with some forms of telangiectasis, as in purpura annularis, and Schamberg's pigmentary disease, pigmentation arises secondary to macroscopic or microscopic blood extravasation. Furthermore, epidermal changes, edema, atrophy and desquamation are possible.

PURPURA ANNULARIS TELANGIECTOIDES

Synonym. Telangiectasia Follicularis Annulata. (*Majocchi,* 1896.)

Definition. This disease is a rare vascular disturbance characterized by the presence of telangiectasia, purpura, pigmentation and atrophy, usually remaining confined to the lower limbs, but at times becoming universal.

Symptoms. The early lesions arise below the knees as small red puncta about the follicles. These enlarge gradually by means of an excentric growth. The eruption is symmetrical and, as the rings enlarge, they heal centrally. A slight superficial atrophy sometimes remains, as do pigmentations caused by minute hemorrhages. The aspect of a fully developed case is an admixture of telangiectatic spots about the follicles, annular telangiectatic macules, or macules formed by segments of circles, areas of purpura, pigmentation and atrophy. No subjective symptoms are present and a fine desquamation at times exists.

Course. The disease begins insidiously, runs a chronic course extending over a period of months, may become stationary at any point and remain so for a long period. In any case it gradually disappears.

Varieties. But one variety is known, although *Cramer* described a case with necrosis and deformity. *Landsteiner* mentions a variety that is not annular.

Differential Diagnosis. Angioma serpiginosum, parapsoriasis and cutis marmorata must be ruled out. The first will be dealt with below. Parapsoriasis is a papular and patchy, scaling disease without marked telangiectases. It runs a chronic course and resembles psoriasis. Cutis marmorata is a universal transitory manifestation, characterized by a tracery of congested veins forming lacy bands among which are scattered islands or normal skin.

Etiology and Pathogenesis. Young adult males are more frequently attacked than females. Auto-intoxication, tuberculosis, nervousness, trophic disturbances and familial predisposition are among the alleged causes. *Brandweiner* obtained a positive tuberculin reaction in one case. Actually, nothing is known of the causation of the disease.

Treatment. There is no rational therapy for the ailment.

Prognosis. The prognosis is invariably good. If left to itself the disease gradually disappears. Even in the rare necrotic forms the lesions disappear leaving harmless scars.

A PECULIAR PROGRESSIVE PIGMENTARY DISEASE OF THE SKIN

(Schamberg's Disease)

This malady starts gradually on the shins and involves the ankles, knees and dorsum of the feet. It is characterized by sharply defined reddish brown lesions, varying in size, and beyond the borders of which are brownish macules and tiny puncta resembling granules of Cayenne pepper. In involuting, the lesions leave a slightly diffuse brownish or yellowish stain.

ANGIOKERATOMA

This curious and somewhat rare condition is named after *Mibelli* who described it in 1889. It consists of small, roughened papular or warty lesions on the backs of the fingers and toes of young individuals. These raised lesions surmount telangiectases. They usually appear after an attack of chillblains and in delicate individuals. Although their relation to tuberculosis is not definitely proved, it is widely assumed. *Dore* reported a case associated with *erythema induratum* (*Bazin*) and the condition is classed among the tuberculides. It is not a serious disease and the therapy consists of tonics, measures directed toward curing a possible tuberculosis,

and the removal of the lesions with caustics or by electrolysis. The prognosis as to the skin condition is good. *Stumpke* described a diffuse variety in a man of twenty-eight. This is extremely unusual, although *Fabry* had already mentioned such cases. The condition is also at times found on the scrotum, persisting indefinitely, the patient remaining healthy, and certainly creating no impression of tuberculosis.

ANGIOMA SERPIGINOSUM

Synonyms. Infective Angioma; Nevus Lupus; A Peculiar Form of Serpiginous and Infective Nevoid Disease (*Hutchinson*).

Definition. Angioma Serpiginosum is a chronic inflammation of the smaller cutaneous vessels resulting in their dilatation, hypertrophy and hyperplasia, at times associated with superficial atrophy of the affected integument. According to *Wise,* but twenty-five cases have been noted in the literature. Since the publication of this paper, however, a few more instances have been reported.

Symptoms. Any portion of the body may be the starting point of the malady, which usually begins either with diffuse red macules, or cayenne pepper-like spots. These arrange themselves in groups which are symmetrically distributed and spread serpiginously. As the eruption extends, it assumes a varied configuration and is composed of several elements as follows: (1) Minute puncta resembling grains of red pepper and not very numerous; (2) Areas of diffuse uniform redness varying in shade and tone, and somewhat suggesting the congested patches caused by cold; (3) Vascular papules from the size of a pinhead to a lentil, scattered about proximally and resembling small angiomata which cannot be rendered entirely pale upon pressure; (4) Delicate vascular rings which are universal, symmetrical and the most abundant type of lesion. These rings are pink or red, not sharply circumscribed, some having depressed centres. They are from 1½ mm. to 2 cm. in diameter. (5) Meshworks of irregularly curved and crooked lines, telangiectatic in character; (6) Pigmented lesions, chiefly on the legs, yellowish or brownish in shade; (7) spots, on the neck or trunk, which are round or ovoid, white, glistening and slightly depressed.

There are no noteworthy subjective symptoms. A universal brawny desquamation sometimes is present. The composite picture is a very startling one since the various elements described exist side by side in no regular order.

Course. The onset is insidious, the progress gradual, covering a period of years until, at times, the entire body is involved. Frequently, however, the affected areas remain restricted. Given lesions

may disappear leaving no trace, or a slight superficial loss of substance simulating macular atrophy.

Varieties. The varieties depend upon the predominance of one type of the many component lesions and the extensiveness of the involvement, but the disease cannot be grouped into definite clinical subdivisions, as for instance lichen or psoriasis may be.

Differential Diagnosis. Angioma serpiginosum differs from purpura annularis mainly because it lacks purpura; from angiokeratoma because of its localization and lack of warty growths; from Schamberg's Disease because the latter is confined to the lower extremities, and lacks the variety of the lesions present in the former. Cutis marmorata has already been described and may easily be ruled out. Parapsoriasis, secondary syphilis and macular atrophy, all of which may slightly simulate the serpiginous angioma, cannot be seriously confused with it because of their definite clinical attributes and lack of telangiectases.

Etiology and Pathogenesis. This is entirely unknown. At times the eruption appears to start from a vascular nevus; far more frequently it arises apparently spontaneously. Race, age, sex and general health bear no relation to the disease. In four instances, according to *Wise,* the condition was found to exist at birth.

Treatment. There is no rational therapy. *Wise* improved his case by the use of the Kromayer lamp.

Prognosis. As regards life and general health the prognosis is good. The disease usually remains limited, often becomes generalized, and rarely involutes completely. Thus, the prospects of a cure are slight.

To dismiss the subject of "essential" telangiectases at this point were but half to discuss the matter. There is no possibility of confusing angiokeratoma of *Mibelli* with the other three, and there is a strong presumption that it may be related to tuberculosis. The remainder of the group, however, have many points in common so that to question the logic of too sharply differentiating them would not be entirely unreasonable. The Cayenne pepper spots, small papular telangiectases, telangiectatic blotches, pigmentation and desquamation are common to all. Purpura annularis is distinguished by a definite purpura, but the presence of pigmentation in the other two is very probably due to blood extravasation not clinically recognizable. Atrophy occurs in purpura annularis and angioma serpiginosum. Schamberg's disease is confined to the legs, while pur-

pura annularis usually involves the thighs and legs, although it may involve the arms. Angioma serpiginosum stands alone in the extensiveness of its distribution, and purpura annularis in the fact that the process starts about the follicles and that local necrosis may develop. On the other hand, there is a certain generic likeness between necrosis and atrophy. Thus, with the exception of the follicular involvement of purpura annularis, there is not a single feature of those enumerated that is not found in at least two, if not three, of the diseases. This makes it difficult to believe in them as definite entities.

Stokes intimated this in his paper on telangiectasia in syphilis. In the case he reported, a large number of lesions simulated the types observed in the "essential" telangiectasia. A histological study showed an inflammation of the cutaneous capillaries with infiltrations about them. The microscopic anatomy of angioma serpiginosum, purpura annularis and Schamberg's disease bears close similarity to that of Stokes's case. In discussing the general etiology of this group, *Stokes* was able to illustrate in how many instances a probable cause exists. This proves that the telangiectasia are really reactions to numerous causes, and that if ever they are to be understood, it will be necessary to attack the problem from the standpoint of causation rather than to be satisfied with the isolation and denomination of clinical entities, which after all may possibly not be entities.

The Gangrenes are given a place in this chapter because their underlying causes are roughly similar to those of telangiectasia. The main difference between the two groups is in the size of the vessels involved. Because those which play a part in the gangrenes are more important, the destruction of tissue is greater, but it must not be forgotten that telangiectases lead to atrophy and purpura annularis may lead to necrosis. Thus telangiectatic diseases and the gangrenes represent differences in degree, but are evidently not generically far apart, at least, so far as the question of vascular pathology is concerned. The causes of gangrene are:

I. Nervous Diseases
   1. Central
      a. Tabes dorsalis, syringomyelia, Morvan's disease, causing perforating ulcers.
      b. Gangrenous zoster.

2. Peripheral — Probably none. This question has not yet been cleared up. Lepra may be in this group.
3. Psychic — Hysterical mutilations.

II. Metabolic Disturbances and Diseases of the Vessels
1. Diabetic gangrene, due either to the direct effect of toxins on the vessels or on the vascular nerves (trophoneuroses).
2. Thromboangitis obliterans.
3. Raynaud's disease (Symmetrical gangrene).
4. Ergotism.

III. Cachectic
1. Dermatitis gangrenosa infantum.
2. Decubitus (bedsores). Typhoid fever, cachexias such as tuberculosis, carcinomatosis, etc.

IV. Physical Causes — Burns, frostbite, ligation of vessels.

V. Infections — Noma, nosocomial gangrene, gas bacillus gangrene, erosive gangrenous balanitis, syphilis, tuberculous lepra.

VI. Unclassified groups of Gangrene Arising in Other Diseases. Hydroa vacciniforme (Chapter IX); Gangrenous syphilis (Chapter XL); Granuloma necroticum, lepra, tuberculosis (Chapter XXV).

The divisions in this table are more or less arbitrary. Three factors appear to be important in the productions of gangrene; a diminution of resistance in the tissues, a vascular disturbance, and finally, perhaps a neurotrophic disturbance which may be central or in the vessel nerves.

I. Local Disturbances Favoring Necrosis
1. Physical
   a. Thermal changes — freezing, burning.
   b. Pressure — Decubitus.
2. Infection — Dermatitis gangrenosa infantum, noma, etc., (see V above, table).
3. Chemicals and other external agents in vocations; (Hysterical mutilations).

II. Vascular Disturbances (see II above, table).
1. Vascular disease, ligation of vessels.
2. Also chemicals as ergot, or sugar, acetone, diacetic, Beta-oxybutyric acid (diabetes) affecting the vessels.
3. Raynaud's disease.

III. Neurotrophic (see I above, table).
Probably through effect on vessels. (In hysterical mutilations, the morbid mentality impels use of caustics, etc., to produce the lesions).

Exactly what the interrelationship of these various factors may be is not definitely known. Vascular changes alone, such as obliterating inflammations, thrombosis, embolism, etc., may cause necrosis, but there must be a still remoter reason for these disturbances such as metabolic disease, injury and central nervous lesions. Pathogenic organisms require fertilized soil to become active, and thus they again may depend upon biochemical or local disturbances. The complexity of the problem might be further illustrated, but it must by now be obvious that in cutaneous gangrene, a group of dermatoses is presented which etiologically is most unclear, and in order to understand which no superficial excursion within the domain of general medicine will suffice. The most important of these diseases are diabetic gangrene, Raynaud's disease, dermatitis gangrenosa infantum, decubitus and noma.

DIABETIC GANGRENE

This starts in various ways and the commonest forms are seen on the sole, or involving the entire lower extremity, beginning somewhere about the foot. The condition may arise as an indurated, hard discoloration, a bleb, or with cutaneous hemorrhages. Necrosis attended with great pain develops. Less common forms are zosters leading to gangrene, or the onset occurs with a furuncle or carbuncle. Any portion of the skin may be the site of these changes. The diagnosis rests upon discovering the presence of diabetes. Exactly what the relationship of the disturbed sugar metabolism is to the necrosis has not yet been explained. Whether it is the excess of sugar itself, the acetone, diacetic or Beta-oxybutyric acid acting directly upon the tissues or the vessels, or indirectly through the central nervous system by means of the vascular nerves, is not clear. In the last analysis the disease is vascular. The treatment in the first place is that of the diabetes. The glycosuria must be controlled by proper diet, and when the urine is free from sugar the blood must be examined to determine a possible hyperglycemia. Acidosis must be overcome. The local treatment consists of antiseptic wet dressings, followed by suitable keratoplastics, such as Lassar's paste or diachylon ointment. Amputation of the

affected member may be necessary, but surgery is not to be undertaken lightly in this condition, and only when all hope of benefit by expectant treatment is gone. On the other hand, surgery should not be delayed until the patient is moribund. The prognosis is always grave, but with proper treatment and comparatively slight involvement many cases get well. Recurrences are common, however, and greatly to be dreaded.

RAYNAUD'S DISEASE

Synonyms. Symmetrical Gangrene of the Extremities; Local Asphyxia.

Definition. Symmetrical Gangrene is a vascular disease, usually of the fingers and toes which, after causing local asphyxia, often leads to necrosis.

Symptoms. After exposure to cold, or even, in delicate individuals, only in cool weather, pain develops in one or more of the fingers or toes, which become white and then red. As the redness develops the pain increases in intensity, the affected areas swell, grow livid, and finally, either recover or become gangrenous. Gangrene may start as such, or after the formation of a bleb, and the involved tissue becomes crusted or mummified. This process in turn may end in recovery. Although large portions of tissue may be affected, frequently only numerous minute ones are. The local temperature is low. It is rare for any other part of the body but the extremities to become involved. *Fordyce* reported a case affecting the ears. Others have observed the disease on the nose, and in a patient of *Stevenson's* spontaneous amputation of the phalanges occurred. All of the nails have been known to fall off simultaneously and suddenly. These manifestations, however, are unusual. *Ormsby* has noted telangiectasia of the face and arms, and calcareous deposits over the extensor surfaces of the fingers and elbows.

Course. The disease follows exposure. After several paroxysmal attacks of local syncopy and asphyxia, gangrene may or may not set in. As a rule even the latter is capable of improvement. Usually years elapse before the period of breaking down arrives.

Differential Diagnosis. Frostbite, the first stage of scleroderma, erythromelalgia and lupus erythematosus are the four diseases most closely simulating symmetrical gangrene. Frostbite rarely leads to necrosis unless the exposure has been prolonged, or the temperature unusually low. The absence of paroxysmal attacks, and freedom from symptoms in warm weather favor the diagnosis of

frostbite; if Raynaud's disease, it persists throughout the year, although it is modified in warm weather. Often it is impossible to distinguish these two illnesses except after years of observation. Scleroderma of the hands or feet does not lead to necrosis until the atrophic stage has been reached, and here the gangrene is not due primarily to the vascular disease, but to a stretching of the attenuated skin over bony prominences. Erythromelalgia is rosy red, rather livid, and at times purple. The color is intensified when the affected parts are dependent and becomes normal when they are elevated. No gangrene develops, although there may be atrophy, and there is hyperthermalgesia to both heat and cold. Lupus erythematosus can be confused with Raynaud's disease only when the nose, ears and cheeks are affected. The scaling of lupus and the atrophy establish the diagnosis of this disease.

Etiology and Pathogenesis. The etiology of symmetrical gangrene is not understood. It has been known to follow any serious illness and *Lustgarten* and *Klotz* thought that syphilis of the vessels was the cause.

Treatment. The therapy is purely expectant. Anodynes to relieve the pain, wet dressings, and alternately plunging the affected parts in hot and cold water are of benefit. In the eventuality of extensive gangrene, amputation may become necessary. When gangrenous areas appear, antiseptic dressings, etc., are indicated.

Prognosis. Although at times the outlook as regards life is serious, this is usually not the case. As a rule, when the involvement is slight the patient recovers from the individual attacks. The disease itself, however, is incurable.

DERMATITIS GANGRENOSA INFANTUM

Synonyms. Multiple Disseminated Gangrene of the Skin; Varicella Gangrenosa; Pemphigus Gangrenosus; Rupia Escharotica; Ecthyma Terebrans Infantum; Impetigo Cachectica; Ecthyma Cachecticum; Ecthyma Gangrenosum.

Definition. This disease, although not rare, is infrequent. It is a multiple disseminated cutaneous gangrene seen in cachectic individuals and probably caused by the bacillus pyocyaneus.

Symptoms. The disease begins in children, rarely older than two years, usually as the sequel to a serious acute illness, or in connection with marasmus. The glutei are the commonest site affected, although no area of the body is necessarily immune, and the earliest lesions are erythemas, nodules, pustules or blebs which rapidly

break down and become crusted. Removal of the crust uncovers a sharply circumscribed punched out ulcer surrounded by a red areola of variable width. In the course of time healing takes place and depressed scars remain. The lesions may be closely grouped together or sparse, and at times general symptoms — vomiting, diarrhoea and variably high fever — enter into the picture.

Course. The course of the malady is determined by the ease with which the general condition of the patient may be improved.

Differential Diagnosis. During small pox and varicella epidemics, these diseases must be ruled out. The localization of dermatitis gangrenosa infantum to the glutei, and the characteristics of the two exanthemata mentioned will serve as fairly reliable guides to prevent errors in diagnosis. Pemphigus, too, may occasionally cause confusion, but there again the restricted localization of the other disease, and the fact that pemphigus rarely causes necrosis, will suffice to allay doubt. A much more difficult disease to exclude is congenital syphilis. Syphilis, in infants, is commonly found upon the buttocks, although of course the lesions are not necessarily confined there. The Wassermann test and the general features of the disease (see Chapter XL) establish the diagnosis.

Etiology and Pathogenesis. Two factors determine the production of the disease; a preceding serious illness or cachexia, and the activity of the pyocyaneus bacillus. This organism is always found in the superficial portion of the lesions, but has never been demonstrated in the thrombosed capillaries prominent in the microscopic anatomy. Although infants are usually affected, adults may be.

Treatment. Two factors are here to be considered — the general and the local. The former involves proper feeding and other measures commonly employed to strengthen feeble infants. Locally, wet dressings of aluminium acetate to cleanse the ulcers, followed by the applications of ammoniated mercury ointment, will almost always effect a cure.

Prognosis. This is good as a rule. At times, however, death ensues, but it is doubtful whether this results so much from the local as the underlying condition.

DECUBITUS

Decubitis or Bedsore develops at points of pressure in individuals long confined to bed by protracted acute, or wasting illnesses. Thus, in typhoid fever, in the final stages of carcinomatosis, tuber-

culosis, tabes dorsalis and the like, this condition is found. Its favorite site is over the sacrum, although it often appears at any point of prolonged pressure. At first, as a rule, an erythema arises, accompanied by edema, a bleb or blebs, after which necrosis sets in, and this, increasing, forms an ulcer with red, irregular, swollen, undermined borders. A tendency to peripheral extension is manifested, and the necrotic area may become truly huge, attaining a diameter of from four to ten inches. There is considerable local pain. In typhoid fever the prognosis of the decubitus is good. In the cachexias, because no relief for the underlying illness is possible, the ulcers are practically incurable.

Treatment of the condition is prophylactic and actual. Prophylaxis lies chiefly in the domain of nursing. A bed-ridden patient should be sponged, rubbed with alcohol and liberally powdered daily; the bed sheets should be properly stretched and a daily inspection should be made by the physician to apprise himself of the first sign of decubitus. Should there be only the faintest suspicion of its onset, the affected region must be gently massaged with alcohol and powdered twice a day, a bed-ring given the patient, so adjusted as to relieve the pressure over the erythema, and the position of the patient changed from time to time. This will serve to prevent the necrosis. After necrosis has once begun, the ulcer must be cleansed daily with hydrogen peroxide, a light dressing of Lassar's paste applied, and the patient should be placed on a bed-ring. Under these conditions nearly all typhoid decubiti are curable. Such is not the case, however, with those arising at the end stages of the cachexias and central nervous diseases. Here, the most that may be accomplished is to keep the lesions clean and in a measure limit their indefinite advance.

NOMA

Synonyms. Gangrenous Stomatitis.

Definition. Noma is a gangrene of the mucous membranes, chiefly buccal, usually arising in weak, institutional children after one of the acute infectious diseases, measles being the most frequent precursor.

Symptoms. The disease begins with blebs in the mouth which rapidly extend and become gangrenous, the temperature often rising as high as 105°. The alveolar process, cheeks and lips may become involved, and at times a large part of the face is included in the necrosis.

Varieties. Occasionally the vulva is the site of noma.

Etiology and Pathogenesis. The disease is clearly epidemic and arises after acute infectious diseases in children's hospitals, the contagion probably being conveyed by means of lax nursing methods.

Treatment. Prophylaxis in the form of individual utensils and appli-

ances and towels for the patients, asepsis on the part of attendants and physicians, and daily cleansing of the mouth, are the best means of avoiding noma. When it once appears, aseptic washes should be employed. At times surgery is necessary.

Prognosis. Three-fourths of those affected die.

The gangrenes seen in central nervous diseases, barring decubitus, need not be mentioned here, but in any gangrene without an easily explicable cause, maladies of the cerebrospinal axis should be borne in mind.

Hysterical Mutilations are scarcely true gangrenes, since they are usually due to the local application of a caustic, or to some physical trauma. (See Chapter IX.) *Ergotism* is now so rare that a description of the disease may be dispensed with. Gangrenes of physical origin, save frostbite, are scarcely true gangrenes since they are temporary and end with the removal of the causes. Neither are they due to primary vascular disease, but to local tissue destruction. *Frostbite* will be described in Chapter IX. *Nosocomial gangrene* and *Erosive balanitis* are bacterial infections. (See Chapter XXIII.) The remainder of this group (Division VI of the first table) will be described in suitable chapters.

GROUP II. CUTANEOUS REACTIONS CHARACTERIZED BY SEROUS EXUDATION

The diseases of this group possess the common feature of having vesicles or bullae as their essential lesion. Etiologically, they are by no means closely allied. In fact in eczema, which is actually only dermatitis, the causes are innumerable. Intertrigo is largely a mechanical dermatosis; the nature of dermatitis repens and pompholyx is not entirely clear; while herpes and zoster are fairly definitely localized diseases of the central nervous system, or at the very least, nerve ganglia, whatever their exciting cause may be. In their course, distribution and in the grouping of the lesions, these conditions are distinct one from the other. Occasionally, admixed with vesicles, may be found bullae.

CHAPTER IX

VESICULAR DISEASES

HERPES (FIG. 9)

Synonyms. Herpes Simplex; Fever Blister; Cold Sore; French Dartre; German, Bläschenflechte.

Definition. Herpes is an acute eruption of a group of vesicles usually situated at a mucocutaneous junction.

Symptoms. With a slight sensation of tension, pain or heat, lasting from a few hours to a day, or without any prodromata, there is an outbreak of a few grouped vesicles (Fig. 9) on a slightly swollen erythematous base. The symptoms mentioned may persist after the appearance of the eruption, and slight fever may accompany the attack. The vesicles are tiny at first, tense, glistening and yellowish, from two or three to a dozen in number, and filled with limpid fluid which may become red in case there is hemorrhage, or a muddy yellow if secondary infection takes place. After a few days the vesicles become flaccid, and dry into a crust, or they rupture. In the latter event a crust also forms the color of which is that of honey, brownish or dirty according to the nature of the contents. Some days later the crust falls off leaving a pinkish epithelialized

surface below, which soon returns to its normal color. Attacks may occur anywhere in the mouth or even, it is said, on the larynx. The buccal lesions resemble aphthae.

Course. As a rule the course of the individual attack is short, rarely lasting for more than ten days. Often herpes is a symptom of an acute infectious disease. This form corresponds to the first in evolution and disappearance. Very commonly there are recurrent varieties, each attack conforming to the outline just given. These recurrences may extend over years, coming on at intervals or succeeding one another so rapidly that the patient is never without some marks of the disease. At times outbreaks regularly accompany menstruation.

Varieties. Herpes may be primary, symptomatic, or chronic, as already indicated. In addition varieties according to site, each with a Latin name, are described. Thus, there are herpes labialis, herpes nasae, linguae, palpebrarum, preputialis, clitoridis and so on infinitely. This is as absurd as would be regarding pneumonia of the upper or lower lobe, right or left lung, as separate clinical entities. A rare condition known as generalized herpes has been described.

Differential Diagnosis. Herpes resembles no other condition strongly. At times, when the lesions appear on the genitalia, it may not be simple to rule out a syphilitic primary lesion, particularly since specific infection may take place in the ruptured vesicle. Ordinarily, herpes is not indurated (although on the penis any lesion may become so) and the spirochaetae are absent. At times, labial and facial (Fig. 9) herpes when infected, simulate impetigo contagiosa. In the latter, however, the lesions are disseminated, whereas herpes is confined to one area. On the body, single small patches of vesicular dermatitis may suggest herpes, but here again the fact that herpes remains restricted while the other malady is disseminated, should prevent confusion.

Etiology and Pathogenesis. Herpes is not due to an isolated cause. It appears either spontaneously at the onset of acute infectious diseases, or may be caused by arsenic (see Chapter VII). When it appears spontaneously it is an index of some internal disturbance, possibly digestive or metabolic. This, however, is still speculative. The symptomatic forms are encountered at the onset of pneumonia, grippe, colds, scarlatina, associated with cerebrospinal meningitis, malaria, and diphtheria. It is so rarely present in typhoid fever as almost to serve to exclude this disease. Attacks

may be induced in the predisposed by slight friction. Herpes progenitalis is common after any form of erotic stimulation, chiefly however after illicit coitus especially in syphilophobics. It not infrequently occurs, nevertheless, without any apparent psychic or physical cause.

Treatment. It is common in those subject to herpes to be aware of an approaching attack through a sensation of tension at the site commonly affected. Such cases may often be aborted by a one per-cent. solution of menthol in alcohol. This is to be dabbed on every hour or so and it frequently controls the process and causes its involution before the vesicles have formed. After they have, however, a similar solution or a weak menthol and camphor salve, to lessen itching or pain, may be used. If infection takes place, a mild ammoniated mercury salve is indicated. In recurrent cases a thorough study should be made of the patient's digestion, habits, diet and general condition. Any faults should be corrected if possible. In a patient troubled with chronic constipation, recurrent attacks of herpes were prevented by regulating the intestinal function through proper diet and the use of mineral oil. This case is cited to illustrate by what simple means apparently obstinate conditions may be controlled. Herpes progenitalis must be treated with mild antiseptic dusting powders, as the sensitive genital mucosa will not tolerate well the medicaments which may be used safely elsewhere.

Prognosis. The prognosis is always good.

ZOSTER (FIG. 10)

Synonyms. Shingles; Herpes Zoster; Ignis Sacer; Zona; French, Zona; German, Gürtelausschlag; Gürtelrose.

Definition. Zoster is an acute disease characterized by pain and the sudden eruption of groups of vesicles on an erythematous base. The distribution of these groups is unilateral, restricted and along the course of a cutaneous nerve.

Symptoms. Zoster usually begins with severe pain of the type popularly described as neuralgic. After three or four days, and frequently accompanied by fever, pinkish spots appear in the painful area. The color deepens within twenty-four hours, vesicles develop, the pain increases, and burning, itching or tingling may arise. At times the pain is agonizing and has a shooting character. The tense vesicles usually contain clear yellow serum, and are closely set in patches which vary from the size of a dime to a silver dollar. Local

adenopathies are almost constant. Hemorrhage into the vesicles is not infrequent, imparting a red, purple or brown color to them, and secondary infection converts the contents into pus. As desiccation sets in the subjective symptoms diminish. Crusts form as in simple herpes and finally drop off, while healing usually ensues without scar formation. At times the patches become gangrenous, or there is a minute necrosis at the site of each vesicle. Permanently depressed scars result from this. In about a fortnight the disease has usually run its course. The aspect of a case of zoster is striking. On one side, and following the distribution of a cutaneous nerve from its point of emergence to its termination, are found discrete or closely set, red islands surmounted by vesicles which may or may not be densely grouped, and which are minute or as large as a lentil. Scars remaining correspond in their distribution to that of the original lesions.

Course. The disease begins with sudden, severe or mild pain, or none at all, the last being exceptional. After hours or days the typical lesions appear, desiccate in a week or two and leave with or without scars. Recurrences are rare.

Varieties. Aside from the varieties determined by location, and so designated, as zoster facialis, nuchae, pectoralis, and so forth, there are mild types, severe types, types in which a typical group of lesions is accompanied by disseminated varicelliform vesicles and systemic symptoms, a gangrenous, a hemorrhagic, and a most unusual bilateral form in which nerves at the same or at two different levels may be affected. *Tryr* has described a generalized form.

It is a strange fact that the lower extremities usually escape the disease. Zoster of the upper branch of the trigeminal nerve at times causes lesions to appear on the conjunctiva, sclera or cornea. This form is accompanied by excruciating pains in the eye, lachrimation and photophobia. In comparing the facial forms which are most intense with those on the lower extremities, *Montgomery* and *Culver* pointed out that many neuralgias of the lower extremities would be accompanied by zoster, but for the length of the nerve (see Etiology below). This type is called by them zoster d'emblée.

Differential Diagnosis. So far as the cutaneous lesions are concerned, zoster can be confused with practically nothing else. Before the eruption has appeared, however, during the stage of prodromal pain, all sorts of conditions may be simulated according to the site involved; torticollis, acute pleurisy, cholecystitis, gall-stone colic,

appendicitis, renal colic, *et cetera.* Frequently enough a laparotomy has been performed, the suspected viscus examined and found normal, while three days after the operation zoster has appeared.

Etiology and Pathogenesis. Zoster may be due to trauma, diseases of peripheral nerves (*Bärensprung's, Charcot's* and *Kaposi's* cases, due to tuberculous changes in the nerves, quoted by *Jarisch*), reflex neuroses, spasm of vessels in dilatation (*Kreibich*). *Haight* and *Kaposi* considered the disease infectious and the preponderance of evidence favors this view. *Bocay* reported an endemic of herpes in one family. *Audry* treated a husband and wife who were affected simultaneously. These two experiences raise the question of contagion. *Montgomery* and *Culver* offered the opinion that the infection gained access to the body from without, travelled up the nerve to the posterior root ganglion causing a hemorrhagic ganglionitis which in turn determined the zoster. This theory, fanciful as it appears, would account for the fact at least that the longest nerves, as those in the lower extremity, rarely are involved in the cutaneous manifestations of zoster, although they pain. *Head* and *Campbell* found changes in the posterior root ganglion of the nerve along the distribution of which zoster lesions were found. *Rosenow* and *Oftedal* produced the disease in animals by injecting streptococci from pyorrhoea pockets and tonsils. Hemorrhages were produced in the posterior root ganglia with a peringanglionitis. Gram positive cocci were found in the nerve lesions. True zoster produces immunity to subsequent attacks.

In addition to this it must be remembered, however, that zoster-like eruptions follow the use of certain drugs or poisons, as arsenic and atropin; general diseases; both the cachexias and acute and chronic infections as carcinomatosis, sepsis, malaria, uremia; and injuries or operations such as intraspinous injections, gunshot wounds and even vaccination. This by no means exhausts the list. Race, age and sex do not affect susceptibility to the illness, but it appears as though the aged are more seriously affected than the young.

Treatment. The local treatment of zoster corresponds to that of simple herpes. Often, wet dressings afford great relief, as aluminium acetate, Thiersch's solution and weak bichloride fomentations. For excessive local pain sprays of carbolic acid one percent., menthol one percent. in alcohol, to which about ten percent. of rose water has been added, will be found useful. A five percent. cocaine ointment, or anesthesin dusted on the open lesions are also of great service. Should the neuralgia be very severe bromides, codein or even

morphine must be employed. In ocular lesions wet dressings of boric acid must be employed and weak dionin solutions may be instilled into the conjunctival sac, but it is wiser to enlist the services of a competent ophthalmologist to control these special indications. The salicylates, salol and iodide of potassium have been used internally.

Prognosis. The prognosis in zoster is good.

Pompholyx, also called *cheiropompholyx,* is a vesicular disease of the hands and feet, prone to be symmetrical and associated with a disturbance of the sweat secretion. It favors the dorsum of the hands and fingers, or foot and toes, the interdigital spaces, palms and soles, and is more frequent on the hands than feet. The vesicles vary in size up to that of a pea, contain a clear, alkaline fluid, are at first imbedded in the skin resembling sago kernels. Later they become elevated. They rarely rupture spontaneously. Tingling, itching, burning, pain, intumescence and erythema complete the picture. Increased sweat secretion is present and the disease is most frequent in the neurotic. The treatment consists of tonics and the local measures to be described in connection with vesicular dermatitis. Roentgen ray therapy is of great service. The disease is common during summer and is one of the local predisposing causes of acute dermatitis. (See dermatitis below.) Pompholyx is possibly of fungus origin, and not, as tradition claims, a dishydrosis.

Intertrigo is an inflammation of the skin covering two permanently contiguous surfaces, as for instance, below the breast, or between the thigh and genitals. Local heat, moisture and friction predispose to the disease. The symptoms are itching, tenderness and a sensation of heat. The lesion is a diffuse erythema covered by macerated epidermis and a secretion is formed which stains but does not stiffen linen. At the folding line of the two adjacent surfaces a superficial fissure often develops and the odor from the affected area is obnoxious. Treatment consists in separating the apposed surfaces with lint, gentle daily washings with soap and warm water, dusting powders of zinc oxide, zinc stearate, talcum, burnt magnesia or starch, or ointments such as vaseline, or cold cream. Carron oil (equal parts of lime water and linseed oil) is an excellent local sedative when applied upon lint.

SIMPLE DERMATITIS AND ECZEMA

In a half-hearted and wholly illogical way dermatitis and eczema have been regarded as two distinct diseases. They are, however, identical clinically and microscopically. The chief reason for the separation of the two is the fact that in dermatitis a recognized excitant exists, not invariably demonstrable in eczema. This is as far as anyone may go to-day in divorcing the two, and this is as absurd as it would be to call enterocolitis of unknown origin eczema of the intestines, reserving the term enterocolitis only for those

inflammations of known causation. Were it not for the weight of tradition the abandonment of the term eczema would be sensible, but so much has been written under this title that such a step would still be impractical.

Synonyms. Dermatitis; German, Eccem; Ekzem; French, Eczéma.

Definition. Dermatitis (Eczema) is a catarrh of the skin possessing the pathological characteristics of an exudative inflammation. It is characterized clinically by redness, swelling, the presence of papules, vesicles, pustules, weeping, crusting and scaling in various combinations. In its course it may be acute, subacute, chronic; and its origin depends upon an interplay between various known and unknown local and predisposing causes to which the skin lesions are reactions.

Symptoms. The salient subjective symptom of dermatitis (eczema) is itching. Burning is also frequent, and rarely either tingling or pain are present. In a certain proportion of acute cases slight fever may be found. Objectively, the characteristic features are the presence of vesicles, papules, or papulovesicular lesions arising upon a swollen erythematous surface. The vesicles either rupture or dry. In the first event crusts are formed which finally fall off and leave a scaling surface; in the second event the scales form at once. The desquamation is either fine or coarse and covers a reddened or buff-colored surface, the margins of which fade gradually into the normal skin. Should the process persist, the involved integument turns red, brownish or buff, and feels leathery. Such a surface scales and may become the starting point of new vesicles or papules. The markings of the skin are exaggerated, causing a cross hatching of furrows. This process is known as lichenification, and a lichenified area may be intensely pruritic. The vesicles are usually small, about the size of a pinhead, but may be as large as a lentil, or even a kidney bean, in which case they are actually small blebs. They contain clear yellow serum, which may at times become bloody, or purulent. When the vesicles rupture and their contents flow out the disease is in the weeping stage. At body folds, over joints, or where the infiltration is thick, the skin cracks forming fissures or rhagades. Either limited areas, more extensive surfaces, or practically the entire skin may be involved, or the process may persistently be restricted to one particular site, as on the palms, soles, scalp, or about the anus. Furthermore, it may remain for a prolonged period in any one of its many stages. Thus, a chronic

thickened patch may persist about the anus, a vesicular patch or patches on the back of the hand or forearm, or a squamous patch on the palms or soles.

Course. This varies enormously. A typical case would begin with an itching or burning erythema which would rapidly become somewhat edematous, then surmounted by papules or vesicles (Fig. 11), the smaller ones of which would dry, the larger, rupture and weep (Fig. 12). Thereupon crusts (Fig. 12) would form and these would be succeeded by scales, after which involution and complete healing would take place. This series of gradations, however, in so classic a sequence is rare, except in acute dermatitis; and here it must be remembered that involution may occur after the erythema without vesiculation, and after vesicles have formed without crusting. Scaling would occur in either case. On the other hand, involution may have begun, and suddenly the entire process will flare up afresh on the partly healed surface.

The process may start as an acute one and become chronic, or may start insidiously without immediate characteristics of the acute type. In the first eventuality, after prolonged papulation or vesiculation thickened squamous (Fig. 13) patches arise; in the second eventuality, squamous patches develop gradually with apparently no vesiculation, but as a rule associated with papules seen chiefly at the periphery of the patches. Either variety is capable of becoming vesicular, and a mutation of vesicular, papular and squamous stages occurs lasting for years, and leading to chronic thickening of the skin, or lichenification. At times, the scales being friable and epithelialization imperfect, while the tissues below are edematous, a constant seeping of serum washes away the epidermis. This causes the chronic weeping forms. In addition to this, one portion of the body may exhibit vesicular patches, another squamous, another weeping, another lichenified in so varied a combination as to baffle description.

The acute types last for days, weeks, or months; the chronic for months or years. Acuity or chronicity besides being applied to the disease as a whole may be applied to the course of individual patches. Thus, in chronic dermatitis, acute patches of vesiculation may arise, or there may be an acute outbreak of grouped papules.

Varieties. The varieties of dermatitis (eczema) may be enumerated from several standpoints and are:

I. Varieties Determined by Course:

1. Acute.
2. Chronic.

II. Varieties Determined by Clinical Aspect; as vesicular, papular, weeping, etc.

III. Varieties Determined by Site; as palpebral, palmar, plantar.

IV. Varieties Determined by Causation:

1. External Origin; as ivy poisoning, occupational dermatitis, *et cetera.*
2. Internal Origin; as diabetic, nephritic, *et cetera.*
3. Local Predisposing Causes; as seborrhoeal, ichthyotic, *et cetera.*
4. Unknown Causation. (The only group to which the term eczema is applicable.)

V. Varieties Determined by Age; as infantile, senile.

I. *Varieties Determined by Course:*[1] (See Above.)

II. *Varieties Determined by Clinical Aspect*:

Dermatitis Cracquelé is rare. It is characterized by the presence of fine, large, irregularly polygonal scales on a small, erythematous surface, and is both painful and sensitive. Its appearance suggests the design artificially wrought into certain Chinese porcelains known as craquelé ware.

Dermatitis Crustosa. (See Dermatitis Madidans.)

Dermatitis Erythematosa is a common variety which may occur anywhere on the body, but favors the exposed parts, notably the face. It itches and burns intensely, scales and becomes edematous, and gradually leads to thickening of the skin. The color is usually a vivid red, but may be either dusky or purple. Traumatism, such as scratching, sharp winds or low temperature intensify the process, while scratching alone often causes vesiculation and excoriation. Crusts form on injured surfaces. The areas involved are fairly sharply demarcated.

Dermatitis Fissa, or Rhagadiformis. Over joints the thickened and inflamed skin cracks and fissures are formed. At body orifices, chiefly on the lips, and about the anus, a similar process takes place. In winter the entire cutaneous surface and exposed parts may become inflamed and the epidermis split forming a lacework of shallow fissures. This is the process known as chapping.

Dermatitis Impetiginoides or Pustulosa (Fig. 14). Any type of

[1] In the older works the word eczema would replace dermatitis, and the qualifying terms would have the neuter ending.

dermatitis, whether acute or chronic, and wherever situated may become infected. In some instances, as in the squamous forms, this is due to scratching. In acute forms the vesicles are infected by the ubiquitous skin staphylococci. Here, pustules are found admixed with the vesicular and squamous lesions. It is a form particularly common to children and leads to the formation of dirty yellowish green or yellowish brown crusts. Pruritis is usually less intense than in the squamous or papular types.

Dermatitis Lichenoides (Fig. 15) is due to cutaneous thickening caused by an infiltration indicating sustained dermatitis. It has been erroneously confounded with Vidal's *lichen chronicus simplex,* or Brocq's *neurodermitis.* It is due to diffuse itching of the integument, or to close juxtaposition of papules which coalesce into placques. The flexor surfaces are favored, but no portion of the body is immune. In color these areas vary from buff to purple, are covered with fine, adherent scales, and beyond their periphery are discrete papules. It is almost impossible to pick up the leathery skin. Chronicity is always implied by this process, but the affected sites commonly enough become the starting point of fresh attacks of the acute forms.

Dermatitis Madidans (Fig. 12). Frequently there is a sustained flow of serum from ruptured vesicles, or from imperfectly keratinized, chronic, edematous and red areas of dermatitis. This form is dermatitis madidans or rubra. When the discharge crusts the picture created is known as dermatitis crustosa. Any part of the body may be involved. The periods of crusting and weeping alternate, and the discharge stiffens linen. This is supposed to be a characteristic feature of the disease, but all dried serum from any sort of lesion may do the same thing. No other disease, however, is marked by so abundant a serum flow.

Dermatitis Nummularis is a form in which coin sized groups of vesicles on an erythematous swollen base develop. The patches itch, appear suddenly and tend to relapse. The extensor surfaces of the hands, forearms and legs are those most frequently involved.

Dermatitis Papulosa. See Dermatitis Lichenoides.

Dermatitis Pustulosa. See Dermatitis Impetiginoides (Fig. 14).

Dermatitis Rhagadiformis. See Dermatitis Fissa.

Dermatitis Rubra. See Dermatitis Madidans (Fig. 12).

Dermatitis Sclerosa is a form closely allied to the lichenoid variety and favors the legs, ankles, palms and soles.

Dermatitis Squamosa is a scaling variety of the disease. The scales are small, white, fine, and not imbricated as in psoriasis.

Dermatitis Verrucosa. In persistent forms, particularly on the lower

extremities, the affected areas tend to proliferate and assume a warty appearance. This is usually observed in the variety associated with varices of the leg.

Dermatitis Vesiculosa (Fig. 11). This is a common variety of the disease. On an erythematous surface, imperceptibly fading off into normal skin, minute vesicles arise, each containing a drop of serum. The entire area is swollen, hot, itching, or painful, and as the vesicles rupture, flaky honey-colored crusts form. In involuting, a squamous, or chronic weeping stage is reached, or if the vesicles become infected, impetiginization occurs. The sites favored by vesicular dermatitis are the flexors where the integument is thin, or if the excitant be an external agent, as rhus, chemicals and the like, the vesicles develop upon the area exposed, and thence may spread to other parts of the skin. In such cases, too, the vesicles may be so large as to be rather small bullae. Vesiculation may arise on a patch of chronic dermatitis, as has already been stated.

III. *Varieties Determined by Site.*

To some extent the picture of this disease is modified by the site affected. Essentially, its clinical features are uniform, but a dermatitis of the scalp differs in aspect from one in the interdigital spaces of the feet purely because of peculiar mechanical, physiological, and anatomical conditions, as the warmth and moisture in body folds, the presence of hair on the scalp, or all three, as in the axilla. Structurally, too, the skin is not alike in all areas. On the scalp, about the nose, on the chest and between the shoulder blades, there are more sebaceous glands than elsewhere. At these sites and in most of the body folds, especially in the axillae, and on the palms and soles, sweat glands are found in greater number. The epidermis of the last two sites is thicker than anywhere else. These variations in hair, sebaceous and sweat structures, and thickness of the epidermis are responsible for many of the superficial variations in the picture. A similar influence is exerted at the mucocutaneous junctions. With these facts in mind, it will be well to reclassify dermatitis (eczema) from the topical viewpoint.

The Head. On the scalp two stages of the disease are common, the vesicular and squamous. The vesicular form is oftenest observed in infants, the squamous in adults. In infants the vesicles readily become infected causing pustules to develop. The secretion of the vesicles and pustules dries among the short hairs which become matted into the crusts. On removing the latter, denuded integu-

ment is exposed, the areas so affected varying in size from a lentil to a dime. These are not ulcers, but patches devoid of epidermis and they give off serum. Interspersed among the crusted and vesicular lesions are scaling patches and excoriations. In adults scaling predominates, but the other features enumerated are not infrequently present. Very often, too, regional lymph glands are enlarged, and in children, in whom the condition is commonly associated with pediculosis capitis, suppurative, cervical and postauricular adenitis develops.

The Ears may be the site of the vesicular, scaling and lichenified stages of the disease. When the process is acute the lobes swell. In the external meatus the scaling variety is common. Behind the ears, where they join the head, both the scaling and vesicular forms are very frequent, and any irritation, such as the wearing of spectacles which curve about the ears, augments the intensity of the process.

The Eyelids present three types of the malady: the scaling, lichenified and vesiculopustular, all of which itch. The first is associated with a subacute erythema; the second is due to chronicity and renders the lids, particularly the upper, red, thickened and inelastic; the third chiefly attacks the ciliary margin causing folliculitis with a seropurulent discharge which produces swelling and agglutination. When the lower lid is involved this organ and the adjacent skin, because of the looseness of the cellular tissue, become strikingly edematous. When both lids are involved they make the patient look as if each eye were encased in a red disc of swollen, vesicular or scaling skin. In infants and children in whom the process is severe the disease has been regarded as connected with scrofula. This, however, remains unproved.

The Nose. In patients with chronic coryza there is sometimes a chronic inflammation of the nares. This is usually of the squamous and fissured variety. A vesiculopustular form, often reaching as low as the upper lip, is encountered in acute coryza.

The Lips are frequently involved. In acute forms there is puffing, and the lesions hurt or itch. A subacute form, radiating from the lips to the adjacent integument, is associated with the use of tooth powders and mouth washes containing wintergreen and peppermint. These forms itch intensely and are extremely disfiguring. Of course, they denote a hypersusceptibility to the essential oils used in the lotions and powders, or nearly all of the human race would be thus afflicted. A fissured variety is common in winter. This

involves the vermilion border and is known as chapping. It belongs to the clinical group of dermatitis fissa.

The Face. In people from thirty to sixty years old, particularly in firemen, sailors, agriculturists, in fact in all exposed to extremes of wind, weather and temperature, the forehead, temples and cheeks become the site of what is called facial (eczema) dermatitis. The skin is thickened, a dusky to purplish red, itches, and alternately weeps and scales. When associated with seborrhoea the scales are greasy. The patient tends to rub or scratch his face and this produces acute exacerbations of the process, as the result of which weeping and crusting occur. This form is most obstinate.

The Neck is often involved in an extension of a persistent variety of the above. Any type of the process may be present, most frequently, however, the lichenified and papular.

The Upper Extremities present all of the varieties mentioned, but there are also some which merit special consideration. The nummular variety is the first of these, and it favors the dorsum of the hands and the extensor aspect of the forearms. (See nummular dermatitis (eczema) for a discription of the clinical appearance.) The hands are the site of occupational or professional dermatoses of this type and the condition may assume several forms. The commonest of these is a vesiculosquamous eruption of the back of the hands which is recalcitrant to treatment while the causal irritation is sustained. Squamous, lichenified and erythematous varieties are also often observed, or a mixture of these forms. Surgeons, nurses, chemists, factory hands, charwomen, painters, printers, candy manufacturers, in fact, those engaged in any manual vocation are subject to the disease. A vesiculopustular and erythematous form usually due to fungus infection is associated with dysidrosis, particularly pompholyx, and a chronic squamous palmar variety accompanied by fissure formation is often encountered. The nail beds may be reddened and swollen, the nails are gray, longitudinally ridged, and often brittle, thickened and distorted. The toe nails may suffer similar changes.

The Lower Extremities too have their peculiar forms. The nummular variety is rare. Associated with varicosities a peculiar dermatitis develops, accompanied by minute cutaneous hemorrhages and followed by ulceration with scaling or hypertrophy of the skin. This is the common dermatitis (eczema) so often associated with ulcus cruris. The hypertrophic form is commonly known as eczema verrucosum, and there is a sclerotic variety known as eczema scleroti-

cum, all of which have already been in part described. At first a vesicular, squamous, or erythematous weeping or crusted dermatitis appears, which itches or pains intensely and is often excoriated. Vivid petechiae then develop which, in fading, turn brown and leave a pigmentation, ultimately permanent, which successive crops of hemorrhagic lesions intensify. Gradually the skin breaks down and an ulcer forms, usually on the outer aspect of the lower third of the calf, or over the malleoli. The ulcer has rolled, thickened, undermined margins and a granulating base which secretes a seropurulent substance, often very foul in odor. In the hypertrophic forms at the sites mentioned in connection with the ulcers, verrucoid plaques develop. Surrounding both the warty patches and ulcers, papules are often seen. Rarely, the entire leg becomes elephantiastic and studded with verrucoid proliferations from the knees to the feet, or with inflammatory lymphatic cysts.

The Soles are the site of a vesiculobullous (Fig. 16) and a squamous form. This is identical with the form described on the hands, caused by fungi. The commonest fungus is the epidermophyton inguinale, and the vast majority of cases formerly regarded as dishydrotic in origin are actually infections. Between the toes vesicular, erythematous and scaling lesions develop, as well as between the toes and the ball of the sole. Maceration causes the thickened epidermis to exfoliate in large, caseous, pure white sheets, exposing painful, red, denuded areas of skin which sometimes look glazed, and lead to the formation of fissures. A similar form occurs in the interdigital spaces on the hands, especially in occupational dermatitis. All of the forms seen on the feet are intensely pruriginous and sometimes exquisitely painful.

Anal dermatitis (eczema) itches extremely, is either lichenified, weeping, or rhagadiform in type and often associated with hemorrhoids, fissures and fistulae. Equally often, however, it is associated with constipation or acid stools, particularly in infants with carbohydrate fermentation. In children with thread worms the condition too is found, and in adults scratching, for the relief of pruritus ani, may precipitate the eruption. Whatever the cause, the process need not remain limited to the anal region, but may extend to the perineum, along the entire intergluteal fold, and thence to the adjacent portion of the buttocks. In infants, because of careless toilet, an acute erythematovesicular dermatitis of the napkin region often arises. This, in nurse-maid parlance, is known as chafing and reflects largely on the carelessness of the child's attendant.

Genital Dermatitis (Eczema). The penis, scrotum, vulva and adjacent areas vary in their response to dermatitis according to certain anatomical peculiarities. When the prepuce is involved acutely it swells and may cause phimosis and balanitis. Acute and chronic forms on the shaft of the penis and scrotum conform to the general type, but the weeping and lichenified varieties are the commonest, and the scrotum often becomes elephantiastic. All forms are seen on the vulva, the most frequent being the weeping and lichenified. Vaginal discharges intensify and protract the disease, and involvment of the mucous surface of the greater and lesser lips and the introitus are the rule rather than the exception. Perspiration, too, augments the condition and, almost invariably, the itching is intense. Maceration results, and this with weeping and scaling greatly increases the difficulty of epidermal regeneration. These regions are involved in a great proportion of the infantile forms of the disease.

The Umbilicus presents a variety modified by maceration. Anywhere else the form most frequent here would simply scale, but the moisture, as between the toes and about the genitals, converts the scales into lamellae which are easily removed, leaving a humid red surface.

The Nipples are attacked by vesicular, scaling and particularly rhagadiform types of the malady and the process ordinarily extends to and beyond the areola. Women are attacked almost exclusively and particularly during the period of lactation. The relationship of this form of dermatitis to Paget's disease will be discussed in connection with epithelioma (Chapter XXIX).

The Flexor and Body Folds. Surfaces in constant contact, as below the breasts, abdominal folds in the obese, the femoro-scrotal, and labial junction, the axillae, because of friction and moisture, are liable to erythematous, weeping, macerated, scaling and rhagadiform types of the malady. The elbow and knee flexures, where the integument is particularly thin and sensitive, are similarly involved, and also frequently by lichenified forms which simulate lichen simplex, and which, in fact, were considered identical with the latter by the Germans who called the condition eczema flexurarum. Forms developing in cutaneous folds are known as intertriginous.

IV. *Varieties Determined by Causation.*

To introduce this grouping at this point is perhaps a trifle premature since the question of etiology has not yet been discussed. Thus, for the present, slightly dogmatic statements will have to be

permitted. In the paragraphs on causation many features which appear unrelated will be brought into harmony.

1. External Origin; *Dermatitis Venenata.*

This condition is produced by a great many external agents of which several examples are appended.

A. Chemical.

(a) Vegetable; poison ivy, poison sumach, nettles, primroses, croton oil, cantharides, mustard, arsenic, turpentine, tars, vegetable dyes, resins, *et cetera.*

(b) Animal; rancid fats as in soap and ointments; certain insects as the brown-tailed moth; secretions and excretions as urine, feces; discharges from the body emunctories and cutaneous glands.

(c) Chemicals; anilin dyes, mercury, salts, iodine and its derivatives, hair dyes, hair washes, picric and chromic acids, phenol, formalin, perfumes, mouth washes, chiefly those containing peppermint and wintergreen, dichloramin T and Carell-Daikin solution, *et cetera.*

All substances employed in art, science, and the most diverse vocations are capable of provoking a dermatitis in given individuals.[1]

Two types of the disease, because of their frequency, require more detailed mention. The first of these is *ivy poisoning,* the second is *occupational dermatitis,* also known as professional eczema. The majority of external irritants which cause dermatitis produce that variety which is best illustrated by a description of poisoning by rhus toxicodendron. It is supposed that toxicodendric acid is the excitant. After exposure to this ubiquitous plant the patient becomes aware of intense burning or itching. In mild cases a few vesicles appear, while in severe ones the affected skin becomes red, swollen and thickly studded with vesicles and small bullae. The face frequently is edematous beyond recognition, the eyelids closed, the lips puffed. After from two to five days the vesicles rupture, crusts form, the swelling diminishes, the erythema fades and desquamation begins. In a fortnight or three weeks recovery is complete. At times the disease persists much longer, however, in a subacute or chronic form, which tradition still compels us to call eczema.

Carell-Daikin solution and dichloramin T when improperly used

[1] For a complete list of these substances consult James C. White's extensive Monograph —"Dermatitis Venenata," Boston, 1887, Cupples and Hurd.

may cause an erythematous, papular or even a papular and vesicular rash. This occurs in susceptible individuals in the neighborhood of sinuses being treated with the above substances, when the surrounding skin has not been protected by petrolatum or a paste of the Lassar type, and while in the army, I often saw examples of the eruption in question. Normal tissue seems often to be adversely affected by these antiseptics, although they certainly do not appear to irritate pathological tissue. In one instance dichloramin T was used by an officer as a mouth wash for an attack of herpes. Within an hour or so he got an acute exudative cheilitis, and his entire buccal mucosa was swollen and painful. A young officer with osteomyelitis of the small bones of his feet invariably, when dressings of these substances were locally applied, developed not only a dermatitis at the sites thus dressed, but a generalized erythomato-vesicular eruption which lasted for a few days, and itched and burned intensely. Such experiences were rare, however. The common eruption seen was that of a weeping, infiltrated dermatitis framing the wound in a margin up to an inch in breadth. This always slowly disappeared when treated with Lassar's paste or a bismuth paste. Nor have I ever seen the condition arise at all in patients, the neighborhood of whose wounds has been protected with petrolatum or some other fat. As a corollary to the above it is obvious that these substances have no value in the treatment of pure dermatoses.

Occupational dermatitis is usually confined to the hands, in particular the dorsum, and is seen among factory workers, artisans, surgeons, trained nurses, *et cetera*. It may and often does resemble ivy poisoning. Equally often it is more like nummular, or interdigital dermatitis, or a rhagadiform squamo-vesicular dermatitis of the fingers and palms. The subjective symptoms may be intense.

B. Physical.

Cold may cause a scaling fissured dermatitis known as "chapping." The face, lips and hands are subject to this, as well as the leg above the shoe tops. Heat and particularly sunburn, for which the actinic as well as heat rays are responsible, causes erythematobullous dermatitis followed by scaling and hyperpigmentation known as tanning. The Roentgen rays rarely cause vesiculation, but among other changes produce erythema and increased pigmentation. Pressure, of clothes or artisans' implements, is also capable of producing the disease. A common example of this form is seen in individuals wearing braces, trusses, splints and surgical dressings, and in the

last type irritating secretions from wounds augment the intensity of the process.

2. Internal Origin.

The commonest variety of this group is that associated with diabetes. It may assume any clinical form. Nephritis, chronic constipation, particularly in the presence of proteid putrefaction, probably sensitization to certain foods, and endocrinous disturbances are also more frequently at fault than we suspect, or with our present methods of study, are able to demonstrate.

3. Local Predisposing Causes.

A great deal has been written of seborrhoeal eczema and dermatitis. The nature of seborrhoea will be discussed subsequently (Chapter XI). Suffice it to say that although seborrhoea is no eczema, eczema or dermatitis often develops on seborrhoeal areas. Ichthyosis is a rare cause of the disease. Hyperidrosis, sudamina and pompholyx are frequent contributing causes, and the malady is often observed with prurigo, dermatitis herpetiformis and scabies. Varicose veins predispose to dermatitis of the leg.

4. Unknown Causes.

Probably the term eczema, if justifiable at all, is so only as applied to varieties of undetermined causation. My own belief is that it would be sounder to eliminate the term and substitute dermatitis of unknown origin, since dermatitis is a descriptive word and eczema a symbolic expression connoting nothing definite. This, however, is a heresy for which we are not yet ripe. The entire question will be referred to in greater detail under the heading of etiology.

V. *Varieties Determined by Age.*

Although no period in human life is characterized by immunity to dermatitis, the process varies somewhat according to the patient's age. Infancy and senility both possess fairly striking forms.

Infantile Dermatitis (Fig. 14) usually begins in the first months of life and may be limited or very extensive. As a rule it arises on the scalp, extending thence to the face and neck, and frequently to other parts of the body, favoring the flexors and those other skin folds peculiar to babies. It readily weeps and crusts, and because of the susceptibility of the infant skin to pyogenic organisms, impetigo

frequently develops. When the malady is severe the baby's face, head and neck are covered with a seropurulent discharge from innumerable vesicles, pustules and weeping surfaces. Crusts, scales, erythematous patches and bleeding excoriations form. Similar manifestations may arise anywhere else. Lichenification is rarely if ever present.

The etiology of the disease has been discussed in a voluminous literature and has stimulated a great amount of investigation. The result of all of this effort in terms of actual knowledge is slight. Infants, in general, live either on maternal or cow's milk so that a dietetic fault, it would appear, should be easy to demonstrate. It has been held that either the fats, carbohydrates, proteids, or salts are responsible. Sensitization to the various milk proteids has also been considered. Another explanation has been the famous exudative diathesis. No one theory satisfies all cases, and each view seems correct in some instances. When these have been totaled, however, a substantial balance remains about which we have no certain knowledge. Undoubtedly, this ignorance will vanish as methods of biochemical investigation improve. Local cutaneous disturbances, too, are involved in the production of the disease, the chief of which is seborrhoea. Very few infants fail to show some evidence of this condition, from a mild crusting of the scalp (the so-called milk crusts) to a more extensive involvement of the face, and even the body, and this favors exudative inflammations.

Senile Dermatitis. The salient feature of the disease at this age is a degree of pruritus often out of all proportion to the extent of the area involved, or the severity of the lesions. Any or all of the clinical types may be present, the squamous and lichenified being the most frequent. The face, genital organs, arms and legs appear to be the sites of election, but any other portions of the body may be and often are affected. The intractability of the disease forces the conclusion that it depends upon profound metabolic disturbances incidental to old age, and the problem of relief is one with that of the discovery of the fountain of youth.

Differential Diagnosis. A disease capable of assuming so many aspects imitates and must be differentiated from many other conditions. To facilitate the process of differentiation, the various clinical stages will be compared in order with the diseases simulated.

Erythematous .....

Localized on Face .....
Rosacea — (Chapter XXXV).

Disseminated .....
Scarlatina, Measles and Various Toxic Eruptions — (Chapter VI).

Vesiculobullous .....

Localized .....
Erysipelas — (Chapter XXIII).
Herpes — (This Chapter, above).
Zoster — (This Chapter, above).
Pediculosis Capitis — (Chapter XX).
Ringworm — (Chapter XXXVII).
Favus — (Chapter XXXVII).

Disseminated .....
Scabies — (Chapter XX).
Pediculosis Corporis et Pubis (Chapter XX).
Pemphigus — (Chapter X).

Vesiculopustular .....

Impetigo Contagiosa — (Chapter XXIV).
Impetigo Bockhardt — (Chapter XXIV).
Sycosis — (Chapters XXIV. and XXXV).

Papular .....

Acute Lichen Planus — (Chapter XIII).
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Squamous .....

Localized .....
Lupus Erythematosus — (Chapter XXV).
Squamous Palmar Syphiloderm — (Chapter XLII and XLIII).
Eczema Marginatum — (Chapter XXII).
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Disseminated .....
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Parapsoriasis — (Chapter X).
Pityriasis Rosea — (Chapter X).
Pityriasis Versicolor — Chapter XXII).
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Premycosis — (Chapter XXVI).
Pityriasis Rubra of Hebra — (Chapter XII).

Rosacea (Fig. 79) is a red eruption of the face, confined to the nose, the adjacent areas of the cheeks, the chin, and the forehead. It is characterized at first by dilatation of vessels, and these persist showing through a diffusely erythematous integument. A more or less marked oiliness of the skin develops and frequently pustules arise resembling those seen in acne vulgaris. After meals, particularly when spicy or hot food has been eaten, the face flushes. No scaling is present, however; the disease is of insidious onset without vesiculation or weeping, and it does not itch, although it often is accompanied by a sensation of burning. Dermatitis (eczema), even when present at the sites usually affected by rosacea, has, amidst the erythematous elements, vesicles and scales, but no telangiectasia. Itching is frequent, rather than burning; there is no flushing and no acne pustules develop. It must be remembered, however, that sometimes irritants employed in treating rosacea may excite a secondary dermatitis.

Scarlatina and *Measles* resemble the mottled erythematous forms of dermatitis (eczema), especially in children. The characteristic systemic symptoms of the exanthemata, their duration, and their failure to show vesicles clinically should prevent confusion.

Erysipelas is at times strongly suggested by acute vesiculobullous dermatitis (eczema) when the latter is limited in extent. The resemblance is due partly to the vivid red of erysipelas, but this alone, without vesiculation, could cause no doubts. When vesicles and bullae form, the diagnosis of erysipelas depends upon the systemic reaction, fever, malaise, or depression, the sharply limited margin, induration, edema, and characteristic peripheral advance of the lesion. Fever in dermatitis is low, if present at all; the patches fade into the normal skin by gradation; edema may be marked, but there is little or no induration, and it is comparatively rare to have serious systemic symptoms. The vesicles and bullae are much more numerous in dermatitis than in erysipelas.

Herpes (Fig. 9). Small patches of dermatitis (eczema) when composed of groups of vesicles, strongly simulate simple herpes, when areas are involved in which the latter is prone to occur. Herpes, however, is preceded by a sensation of tension and burning, rather than pruritus. The vesicles are deep-seated and rupture with difficulty and the patches are usually single, or, when multiple, follow a line of nerve distribution. In dermatitis and eczema the patches are numerous and do not follow the course of nerves. Iso-

lated islands of this condition are rare. Herpes does not weep or scale, but involutes by desiccation.

Zoster (Fig. 10). The sudden outbreak of a rash after a prodromal period of several days of pain, usually severe and often agonizing, the typical distribution and mode of fading are all sufficiently distinctive to rule out dermatitis, save for the fact that both are vesicular diseases. The grouping of the vesicles in zoster is compact and the lesions are deeper and prevailingly larger.

Pediculosis Capitis. At times pediculi of the scalp cause a vesicular dermatitis of the face, neck, ears and scalp, itself. This differs in no way from any other dermatitis, save that it more readily becomes impetiginous. It is important, however, to bear in mind that dermatitis at the sites mentioned may be due to the epizoa and a conscientious investigation to establish their presence, or that of nits, should be made.

Ringworm (Fig. 48). The vesicular forms of ringworm remotely simulate dermatitis (eczema) in circular patches. Ringworm prevailingly occurs in circular lesions, the centre of which tend to be scaling or crusted, and the periphery vesicular or vesiculopustular. The site most frequently involved is the scalp, although the glabrous areas are also affected. The diagnosis is established by demonstrating the presence of the micro-organisms. Recently *Ormsby* has reported the presence of epidermophyton inguinale in the vesicles of a form of vesiculobullous dermatitis of the hands and feet resembling pompholyx. (Chapter XXII.) This has already been mentioned in paragraphs dealing with the hands and feet.

Favus simulates dermatitis in only one stage. When the latter is vesicular and golden crusts appear, these suggest the sulphur-colored scutula of favus. The crusts are not cup-shaped, however, and the achorion is absent.

Scabies (Fig. 43). Generalized pustulovesicular dermatitis (eczema) somewhat resembles scabies, since scabies frequently becomes "eczematized." The localization, the nocturnal itching, the burrows caused by the acari, and finally, the microscopic demonstration of the latter are the points upon which the diagnosis rests.

Pediculosis Corporis (Fig. 44) *et Pubis.* People afflicted with the body louse at times acquire dermatitis. The parallel scratch marks, chiefly over the shoulders, but also wherever else the clothing presses against the body, the general thickening and pigmentation of the skin rather than the presence of papules or vesicles, furnish an ample basis for differentiating pediculosis corporis from dermatitis. Find-

ing the lice in the folds of the underclothing clinches the matter. Pediculosis pubis, itself, does not cause dermatitis nearly so much as does the blue ointment, so universally used in the treatment of the disease.

The inflammation corresponds to the involved hairy areas and the diagnosis rests partly upon the history, and partly upon the discovery of nits or pediculi in the hair.

Pemphigus (Fig. 17) may but rarely be confused with dermatitis (eczema), and then only when the latter exhibits large bullae. Even in such an event, however, the fact that pemphigus blebs arise from a non-inflammatory base, and the presence of buccal lesions are too distinctive to permit of much confusion. Pemphigus foliaceous may remotely resemble the weeping and scaling stages, but the admixture of blebs and the severe constitutional reaction would exclude this possibility. In this connection it must be remembered that dermatitis herpetiformis, closely related to pemphigus, may also resemble simple dermatitis. The vesicles in dermatitis herpetiformis are inclined to be grouped herpetically, and involute by desiccation.

Impetigo Contagiosa in its pure form does not resemble dermatitis (eczema) but the latter is capable of becoming secondarily infected, or impetiginous. It is important, in connection with impetigo, to determine its underlying cause. Any vesiculobullous, rhagadiform, or excoriated dermatosis may be the starting point. Obviously, dermatitis is among these. Impetigo and dermatitis are both vesiculobullous, serum exudes from the lesions, and crusts form. The lesions of impetigo are prevailingly bullous, however, rapidly become purulent, the crusts are honey colored, the weeping transitory, the course short, the disease is almost entirely restricted to children, and favors the face or scalp. Ecthyma is a form of impetigo which involves the skin more deeply, leaving punched out ulcerations. In general, though, what is true of impetigo holds in ecthyma as well.

Impetigo of Bockhardt is a suppurative folliculitis of the scalp and is closely related to sycosis non parasitaria better called folliculitis staphylogenes barbae. Because of the similarity of the small pustules to infected vesicles, a certain resemblance exists to vesiculopustular dermatitis. Close inspection of the lesions, their intimate restriction to the follicles, their pure pustular character, and the fact that the lesions are pierced by hairs, rule out any other diagnosis than that of impetigo or sycosis.

Lichen Planus. Save for the acute generalized form of lichen planus there can be no confusion between this disease and dermatitis,

and only the disseminated small vesicular or vesiculobullous form of the latter can create such confusion. Close inspection of the nature of the lesions immediately indicates how easy of differentiation the two are. The lichen lesion, in the disseminated forms of the disease, is a minute, glistening, waxy, chrome papule, umbilicated and highly pruriginous. No vesiculation exists. The dermatitis papule is a small, solid, dull red and slightly lenticular lesion. In lichen, the buccal mucosa is frequently the site of minute, pure white lesions, totally wanting in the other disease under consideration.

Prurigo is papular urticaria. It usually begins in childhood, persists indefinitely, and is characterized by the presence of minute, shotty papules, some of which have a pinpoint vesicular summit. It is localized prevailingly upon the extensor aspects of the limbs, the buttocks and belly, and the skin as a whole becomes thickened. It is unattended by large areas of vesiculation or scaling, and the glands enlarge throughout the body.

Lupus Erythematosus. For the most part this disease involves the face and scalp, usually in bilateral symmetrical distribution. The lesions are scaly placques of purplish red, the margins have greasy adherent scales the under surfaces of which possess short projections or plugs, casts of the patulous follicles in which they arise. Ultimately, an atrophy remains at the site of the lesions. In dermatitis the scaling occurs on a generally slightly erythematous surface, and often evidences of vesicles remain. No atrophy occurs.

Squamous Palmar Syphiloderm (Figs. 90 and 95). Squamous lesions are seen in the secondary and tertiary stages of syphilis. Those observed in the former cannot readily be confused with squamous dermatitis; those arising in the latter can, and it is often almost impossible to differentiate the two. The syphiloderm is usually, but not always unilateral, and it never itches, but at times the scaling dermatitis of the palm does not either. Frequently the syphiloderm has the characteristics of grouping known to syphilis. Often enough the outline is irregular, fissures form, the epidermis is thickened as in dermatitis, and the scales are irregular in size and shape. A positive Wassermann reaction and other evidences of syphilis are required to settle the diagnosis, but if the serum test should be negative as sometimes is the case in the tertiary stage, vigorous general, without local, antisyphilitic therapy as a diagnostic procedure, may clear up the question.

Eczema Marginatum (Fig. 52) (Epidermophytia Inguinalis) is a disease of the scrotum, more rarely the vulva, the adjacent portion of

the thighs, the axillae, and the folds formed by the breasts and chest. It is a red squamous slightly itching disease, closely resembling dermatitis of the areas mentioned, and it consists primarily of nummular lesions which gradually coalesce into placques with a festooned outline. The diagnosis can be settled by demonstrating the causative fungus — one of the trichophytons. The cause of this variety is the epidermophyton inguinale and it is related to the form seen on the hands and feet, already described.

Erythrasma affects the same areas as eczema marginatum, and consists of diffuse patches of a buff or copper color which scale and have a sharply circumscribed margin. It is caused by a fungus, the microsporon minutissimum. The diagnosis often can be made only by demonstrating the organism.

Psoriasis resembles patchy, scaling dermatitis (eczema) particularly when associated with seborrhoea. This is especially true when itching is present, as occasionally occurs in psoriasis. The scales in the exfoliative stages of dermatitis are finer than in psoriasis, not laminated and not clear white, and associated with them may be other elements of a process such as vesiculation, which are totally lacking in psoriasis. The purpura and punctate bleeding, on removing scales, are characteristic of psoriasis, but lacking in the other condition. Finally, in even the least characteristic forms of psoriasis, some typical elements are present at the classical sites, which clearly indicate the nature of the disease.

Parapsoriasis does not itch, never has a vesicular stage, runs a chronic and prolonged course, has very fine scales, and only on most superficial inspection can be confused with dermatitis. Parapsoriasis always evades the head.

Pityriasis Rosea resembles that form of dermatitis or eczema which arises on seborrhoeal skin. At times, the most experienced dermatologists are unable to differentiate between the two conditions. In typical cases of pityriasis rosea the early lesions are pink, the later ones have a tan or buff centre, and a scaling pink periphery. It is rare to see lesions above the neck, or below the middle third of the thigh, and somewhere on the body, usually on the lower abdomen or back, one lesion, much larger than the rest, is seen. This lesion is the starting point of the disease. In general, too, the condition appears to be epidemic in the spring and fall.

Pityriasis Versicolor also remotely resembles those forms of scaling dermatitis which arise in connection with seborrhoea. The like-

ness is superficial and if necessary a demonstration of the pathogenic agent of pityriasis versicolor, the microsporon furfur, clinches the diagnosis.

Lichen Acuminatus in its exfoliative stages, particularly when it forms placques, can resemble lichenified scaling patches of dematitis. The finding of typical papules admixed with patches of lichen acuminatus, particularly on the dorsum of the fingers, the scales adherent to the papules, the absence of vesiculation determine the diagnosis of lichen.

Premycosis: Mycosis, or Granuloma Fungoides, has an initial stage one form of which is indistinguishable clinically from chronic dermatitis. The prefungoid patches, however, are a trifle more circumscribed and much more pruriginous than are those of the other condition, and microscopically, often possess a granulimatous structure. Many cases of mycosis begin with what at first seems to be a harmless dermatitis, and only the course of the disease reveals its actual nature.

Pityriasis Rubra of Hebra and the other erythrodermas are capable of simulating a generalized dermatitis. It is indeed rare, however, for the latter to be universal as are the erythrodermas. Moreover, the scaling in these exfoliative dermatoses is coarser, more profuse and more persistent, and is not consecutive to vesiculation. Their constitutional symptoms are pronounced, the skin in general thickens, and weeping is rare. The entire integument from top to toe is covered, and while dermatitis may involve all areas of the body it does so by virtue of wide distribution of the lesions, which practically never exist universally and uninterruptedly.

Etiology and Pathogenesis. [1] The problem of the etiology of the disease has stimulated much investigation and more writing. Our views concerning the question are not yet in accord. It is variously contended that:

1. Eczema and simple dermatitis are two distinct conditions;
2. Eczema is invariably of internal origin;
3. Dermatitis is invariably of external origin;
4. Either disease may be of either origin;

[1] For a detailed review and bibliography of this question, see Pusey's "Text-Book on Dermatology"; Brocq's "La Question des Eczemas," Annal de Dermat. et de Slph., January — March, 1900; Besnier, "Pract. Dermat.," Vol. II., P. 1; Heimann, "A Critical Review of Eczema and Dermatitis," *Journ. of Cutan. Dis.* Vol. XXXIV., No. 4, PP. 259–284; and *Jour. Amer. Med. Assoc.*, Vol. LXVII.

5. Dermatitis is a cutaneous catarrh of known external or internal origin, and eczema a cutaneous catarrh of unknown external or internal origin;
6. Eczema is dermatitis of unknown origin.

The internal causes of eczema have been regarded as:

1. Diathetic — heredity, constitutional weakness, gout, diabetes, *et cetera;*
2. Physiological — infancy, dentition, pregnancy, lactation, senility;
3. Nervous;
4. Functional and Organic — digestive, urinary and assimilatory:
5. Disturbances of metabolism and of the endocrinous glands.

The local causes of eczema have been considered:

1. Cutaneous anomalies such as ichthyosis, hyperidrosis, seborrhoea, skin folds, body orifices;
2. Chemical and physical irritants;
3. Micro-organisms.

The local causes of dermatitis have been considered:

1. Chemical and physical irritants;
2. Micro-organisms.

Before proceeding with a discussion of these numerous and dissimilar factors, it is necessary to understand the pathology of the two conditions. Clinically, as has been pointed out, they are identical. Microscopically, the same holds true. Both are catarrhal inflammations; both are exudative. Thus, histologically, eczema is simple dermatitis. What then are the differences, if any, between the two processes? In the disease commonly known as simple dermatitis, there is a history of known exposure. Is this enough to differentiate it clinically from eczema? For an answer, the doctrine of cutaneous reactions (Chapter III) must be re-emphasized. Just as wheals may be due to innumerable causes constituting the picture of urticaria, so are the lesions of (eczema) dermatitis. Precisely as wheals are provoked both by external and internal causes, so are the lesions of (eczema) dermatitis.

Further facts must be borne in mind. All individuals are constantly exposed to cutaneous irritants — whether physical or chemical. A relatively small proportion of the human race ever shows any ill results therefrom, else every surgeon would have bichloride

dermatitis, every workman some occupational dermatosis, and everyone exposed to rhus would have dermatitis. Examples might be cited indefinitely. All of this indicates that the majority of human beings are incapable of being harmed in the manner mentioned. They are immune. From this it follows that susceptibility means predisposition, and predisposition indicates a variation from the normal which may be congenital or acquired. Two factors then are necessary to produce dermatitis, a predisposing or general and a precipitating or local cause.

Diabetes is one of the general diseases capable of causing dermatitis. There are many others also, but diabetes will excellently illustrate the influence of internal derangements in the pathogenesis of the malady. Not every diabetic, however, acquires eczema. Why? Because the skin is not equally liable to the disease in all individuals. This means that a precipitating cause must exist in those who become afflicted. What then is the difference between two conditions which are identical clinically and microscopically, but one of which is due to a known local and undetermined general cause, and the other to a known general and undetermined local cause? Actually none. Inability to detect the unknown quantity does not exclude the existence thereof, but simply denotes ignorance or inadequate scientific methods on the part of the observer. Such a deficiency supplies no rational grounds for the creation of two disease entities, and we are forced to the conclusion that since eczema is dermatitis, and since not all the causes of dermatitis are known, eczema, if we wish to use the term at all, is dermatitis of undertermined origin. Thus, it ceases to be rational to retain such a word as eczema, but it is inevitable that we must remember what it signifies, since the term has been used in all previous literature. We may restrict its scope to dermatitis of unknown origin. We might as well, however, call nephritis, or colitis, or gastritis of unknown origin, eczema of the kidneys, colon, or stomach.

The local causes of dermatitis are:

1. Chemical. Acids, alkalies, salts, caustics, cleansing fluids, powders, soaps and cosmetics — in short, all materials used in any vocation or phase of life.
2. Vegetable Substances. Rhus, primrose, poison sumach, wood oils, and many others — forty-six families in all.
3. Animals and Animal Substances. Brown-tailed moth, pediculi, acari, cantharidis, ants, and many less common varieties.
4. Micro-organisms.
   a. Bacteria, staphylococci (?). This on the whole has never been

proved. (See the Transactions of the Fourth International Dermatological Congress.) Possibly bacterial toxins or the bacteria themselves may intensify the process. Engman's infectious eczematoid dermatitis is due to a staphylococcus.

b. Fungi. Ormsby and others have isolated a fungus from lesions resembling pompholyx.

5. Physical Causes.
   a. Actinic rays — X-ray and sun dermatitis.
   b. Thermal — Burns and frost-bite.
   c. Trauma — friction from clothes, implements, *et cetera*.
6. Undetermined.

The general causes are:

1. Metabolic — Disturbances of nitrogen metabolism (nephritis), sugar metabolism (diabetes), or of the glands of internal secretion, particularly hyperthyroidism.
2. Digestive and Assimilatory — Gastritis, hyperacidity, achylia, bread dyspepsia, carbohydrate fermentation, proteid putrefaction, constipation, enterocolitis, fat indigestion.
3. Eliminatory Diseases — Chiefly nephritis.
4. Hematogenic — The anemias and leukemias.
5. Anaphylactic — Intolerance to proteids.

The so-called reflex causes — dentition, menopause, menstruation and nervous — can properly be included in the metabolic group. It is difficult to conceive of the way in which the nervous system by being deranged might cause dermatitis, unless some forms thereof were trophoneuroses, a view for which no valid evidence exists. My own experience leads me to believe that the commonest internal causes of conditions with which dermatitis is associated are disturbed nitrogen or sugar metabolism, hyperthyroidism, the entire group of digestive and eliminatory disturbances, badly tolerated food-stuffs, and sensitization to certain proteids. Of all of these, the most frequent are proteid putrefaction and constipation.

Local predisposing causes are ichthyosis, hyperidrosis (related to pompholyx and prickly-heat), skin folds (related to intertrigo), body orifices (related to dermatitis about the mouth, anus, vulva, perineum). The scalp, eyebrows and eyelids are also peculiarly vulnerable, and finally it must be remembered that in certain individuals areas far from body folds or orifices, or free from any anomalies or other cutaneous disturbances clinically recognizable, are subject to the malady. Whether this is due to chemical or anatomical peculiarities is hard to determine. In interpreting the contents of this paragraph it is obvious that local predisposing causes must be

summed up in the scientific symbol of points of lowered resistance. Local predisposing causes may act in three ways. They may assume the function of precipitating causes in the presence of general disturbances; retain their rôle of predisposing causes in the presence of external precipitating causes; or, act as the hypersusceptible medium in which external causes operate with maximum intensity in individuals with a general predisposition. Thus, eczema is dermatitis, and dermatitis depends upon an interplay between various known and unknown local and predisposing causes.

Treatment. The external treatment of dermatitis or eczema differs according to the stage of the disease, its situation, according to individual peculiarities of the lesions, and symptoms. The internal treatment depends upon the general cause, if there is one that can be determined. The first point of importance to settle is the presence of a local precipitating cause. Thus, the patient must be interrogated as to occupation, habits, pastimes, the recent acquisition of new clothing, the use of cosmetics, hair lotions, soaps and powders. By this means only may occupational dermatoses, and those of chemical and physical origin be recognized. Scales and secretion from vesicles must be examined for fungi, so that dermatitis due to such organisms may be recognized. Inquiry and clinical and pathological examinations must be made to determine the presence of digestive, eliminatory, gynecological, metabolic, and endocrinous disturbances. If the work of *C. J. White* in the percutaneous test in the disease bears out its promise, a valuable diagnostic aid will have been established. Internal treatment must be guided by the above considerations.

The digestive tract appears most frequently at fault, but it is difficult to be sure whether this is cause and effect, or whether the skin and intestinal conditions are both expressions of a common underlying cause. In any event, the method of attack here is to modify the diet subject to the presence of carbohydrate fermentation, proteid putrefaction, simple constipation, hyperacidity, achylia, *et cetera.* In addition, the use of mineral oil for constipation, daily colon washings, particularly for chronic intestinal intoxication, and the suitable employment of cathartics and eliminatives are indicated. For intense pruritus, especially in high-strung patients, the bromides and valerian preparations are of great value. It must be remembered, however, that the bromides and opium derivatives, useful as they usually are, themselves may cause itching in certain people, and unless they produce a very marked and prompt

antipruritic effect, they are to be discontinued. Furthermore, constipation frequently results from the use of opium which must therefore be employed guardedly. If nephritis should appear to be responsible for, or associated with the dermatosis, suitable treatment of the kidneys obviously is indicated, but diaphoretics are not, since stimulation of the sweat apparatus would probably irritate the skin. When excretion of nitrogenous end substances of metabolism is lowered by renal impairment, the patient must be put to bed, and the main indication becomes that of the renal disturbance. After restoring the powers of elimination so far as possible, the treatment of the skin again becomes the more prominent problem.

Local Treatment. Acute Dermatitis (Acute Eczema). The therapeutic problem is the control of an acute exudative inflammation, the phenomena of which are swelling (edema and vesicles), congestion (erythema), pain (itching and burning) and local heat. The means of accomplishing this correspond with those employed for any similar process anywhere within the body. So far as possible the affected part must be put at rest. Thus, when the involvement is extensive, the patient should go to bed, or at least lie down. The remedies indicated are wet dressings, soothing lotions and pastes. The use of ointments is contra-indicated. The following medicaments are the best to be used as fomentations; viz.: Burrow's solution (alum acetate), resorcin lotion, magnesium sulphate, or witch hazel. Burrow's solution should be diluted with nine volumes of cool water, and bulky dressings should be applied to the affected parts constantly for from twenty-four to forty-eight hours; or until the swelling and erythema are reduced. This treatment should not be used at night if it interferes with sleep. Impervious substances to cover the dressings are emphatically contra-indicated, as the value of fomentations in acute vesicular processes depends in a large measure upon evaporation. Weak resorcin solutions are keratoplastic and antipruritic. Wet dressings of this substance in one to two percent. strength may be employed precisely as Burrow's solution, but resorcin should not be used about the eyes as it may cause severe conjunctivitis. Magnesium sulphate is employed in half saturation in water as hot as the patient is able to bear, and the dressings are applied for from fifteen to twenty minutes every two hours, followed by a soothing lotion. Witch hazel is diluted with equal parts of hot water and used exactly as magnesium sulphate.

During such periods as the wet fomentations are interrupted,

as at night, or in the intervals following the magnesium sulphate or witch hazel dressings, the skin is to be coated with a soothing, shaking lotion of which the following are types:

Powdered Calamine aa... 10.0
Zinc Oxide
Alcohol 50.0
Rose Water .....to 200.0

Sig.

Zinc Oxide
Powdered Calamine aa... 10.0
Alcohol 50.0
Rose Water 20.0
Lime Water .....to 200.0

Sig.

A lotion of this sort must be thoroughly shaken up, poured into the hollowed palm of the attendant and splashed on the affected surface where it dries. It leaves a powdery coating which should not be removed save that it is wise to cleanse the skin once in twenty-four hours with olive oil. In doing so, however, no great force should be employed and only such crusts and coatings of dried lotion should be removed as come off easily. Burnt heavy magnesia may be substituted for the calamine, and it is somewhat more soothing. Ichthyol, in a concentration of from five to ten percent., may also be included in the formula. This substance tends to constrict the vessels and thus diminishes the congestion. Of the two formulas given the second is more astringent and the first more soothing. Thus, the second is preferable in the more vesicular and erythematous types of lesions, but its use must be restricted, as it may dry the skin excessively and make it too brittle. At the onset of the disease it is often found beneficial to paint the skin with equal parts of water and ichthyol.

When the process has begun to subside wet dressings and lotions are no longer indicated, and zinc paste (equal parts of zinc powder and vaselin) is substituted. The proper way to employ this substance is to spread it evenly and thinly on strips of unbleached muslin four to six inches broad which are then applied to the affected areas and bound on lightly with ordinary bandages. It may be spread directly on the affected surface itself and bandaged loosely with muslin. The former method is better. A change of the dressings should be made every twelve or twenty-four hours, preferably the latter, and the parts lightly cleansed with pure,

fresh olive oil, if the paste has dried upon the skin. If the parts look clean, however, it is far wiser to omit the oil washing in order to irritate the skin as little as possible. Within a week to ten days the acute process should have subsided, and subsequent dressings with ordinary zinc ointment will suffice until exfoliation ceases. Should the process have become chronic, the treatment to be pursued is outlined below.

Acute weeping and chronic vesicular and weeping dermatitis are at first treated as the acute vesicular form. Weeping in general is due to imperfect keratinization and thus resorcin fomentations are peculiarly useful. Painting of the affected areas with a twenty percent. silver nitrate solution is of great value since it promotes the growth of epithelium and protects the denuded surfaces. This treatment, however, must not be too long continued, or too extensively applied, because of the danger of argyria. Lassar's paste dressings, to be changed every eight hours, are of great value. The paste must be applied thickly in order to take up the serum. Diachylon paste and ointment are also of signal utility, since the lead plaster is a great stimulant of epithelial growth.

Chronic scaling dermatitis must first be stimulated. The best revulsives are tar (oleum Rusci, oleum cadini, liquor carbonis detergens, technical coal tar, resorcin, salicylic acid). An excellent base is zinc ointment. Oleum Rusci is best used in a strength of from two to five percent., the other tars in from five to twenty percent., resorcin in from two to five percent., salicylic acid in from two to three percent. The ointment is rubbed in fairly vigorously once or twice a day, and when the skin has become thoroughly irritated Lassar's paste dressings are used. Roentgen therapy, 1 Holzknecht unit weekly, hastens the disappearance of these lesions. In lichenified eczema, in addition to the treatment just suggested, chrysarobin, pyrogallol, eugallol, the first two in five to ten percent. strength in vaselin or lanolin, and the last in ten percent. solution in acetone are valuable. These provoke a more or less marked dermatitis leading to exfoliation. The patient must be warned of the danger of these medicaments to the eyes, and of the destructive effects of the first two on clothing. Thus, the surfaces treated are to be protected, and the patient must be seen daily so that not too profound a dermatitis will be excited.

Special indications arise according to the nature, site and cause of the lesions. Dermatitis craquelée is best treated with daily inunctions of mild boric acid (two to five percent.), or salicylic

acid (two percent.) ointments once or twice a day. The fissured form must be stimulated with silver and dressed with emolient ointments. Impetiginous forms are best treated at first, in the manner of vesicular types, and then with ammoniated mercury ointment, two to ten percent. The treatment of lichenoid, weeping, nummular and sclerotic types has already been described. Verrucous dermatitis (eczema) must be converted into the scaling or lichenified type first. This is best accomplished by the use of ten to twenty percent. salicylic plaster. Then the treatment becomes that of the lichenoid or scaling type. Roentgen rays used as above, are of great value.

When the scalp is involved in males wet dressings may be employed, but this is almost impossible in women. Calamine and zinc lotions cannot be used anywhere where there is a long growth of hair, nor can pastes. Mild resorcin and boric acid lotions, mild boric and ammoniated mercury salves (when these are tolerated) are our only methods of attack. The eyelids may be treated with wet dressings of boric acid, salves of mild, ammoniated mercury ointment (two percent.), or yellow mercuric oxide (one percent.). When the nares are involved, silver nitrate for the fissures, external wet dressings, and white precipitate salve are indicated, particularly if there is also impetigo. Labial involvement may be controlled by the general methods outlined. Tylotic forms (palms and soles) are first treated with salicylic acid plaster as above, followed by salves and Roentgen rays. Anal eczema is best controlled according to the general rules outlined, but if hemorrhoids, prostatitis, or carbohydrate fermentation are the basis of the skin manifestations, these faults are the essential ones to correct. In children with thread worms colon irrigations and white precipitate salve should be used. When the genitals are the site of the disease the treatment corresponds to that outlined, and in men, balanitis, and in women, vulvovaginitis or diabetes are often the basis of the disturbance. When these conditions are relieved the cutaneous manifestations often vanish without local treatment. Scrotal elephantiasis is intractable. Umbilical forms, and many of those seen in body folds, are treated according to general principles and those set forth in connection with intertrigo. The important point is to reduce friction as much as possible.

Special etiological factors also modify therapy. Exposure to any external excitant must be prevented. At times, even occupation must be changed. Hyperidrosis is best controlled by means of the Roentgen rays; seborrhoea, with sulphur or resorcin lotions or

salves, or white precipitate ointment. Infantile forms of the disease are often due to digestive disturbances which must, if possible, be corrected. That variety due to fungi is best treated by inunction twice daily with Whitfield's ointment.

℞.	Acid salicylici	2.0
	Acid benzoici	4.0
	Ung. aq. Ros. q.s. ad.	30.0
	Sig.	

Prognosis. The prognosis as to life is good. In the majority of instances the disease is curable. Forms depending upon occupation and habit are amenable to treatment only when these are changed. Elephantiastic scrotal forms, and those due to varicose veins, or varieties dependent upon incontrollable metabolic disorders are incurable, but may be improved by proper therapy. Hyperidrotic forms may be improved only when the disturbance of the sweat apparatus is amenable to therapy. Here the X-rays are of great value.

DERMATITIS TRAUMATICA

Scratching, the rubbing of apparel, bites of animals, the use of implements and tools, may injure the skin, causing the various well-known types of cutaneous traumata.

Dermatitis Calorica is produced by intense heat, either through heat rays, or actual contact with hot substances, such as steam, boiling water, oil, *et cetera,* and fire itself. Slight exposure causes an erythema; more exposure, an exudative inflammation with vesicles and bullae; and still more exposure, an eschar with involvement of the deeper tissues. These stages are respectively first, second and third degree burns. The last two may become infected. Healing takes place with scar formation. When half of the body is involved the patient usually dies. The treatment of first and second degree burns is that of acute dermatitis. An excellent way of handling second and third degree burns is to cover them lightly with a single layer of sterile gauze, otherwise leaving the affected areas exposed. Extensive third degree burns should be treated as surgical wounds. If necessary, skin grafting is indicated.

Dermatitis Congelationis, freezing, or frost-bite, usually involves the nose, ears, fingers, toes, feet and cheeks. This process is also divided into three stages. In the first there is erythema following ischemia; in the second, vesicles and bullae; in the third, gangrene. The intensity of the process depends upon the intensity of the cold, and the duration of the exposure. Treatment consists of the restoration of circulation by friction with snow, or ice. Otherwise, it corresponds to that of dermatitis, or that of gangrene, or a wound. People subject to chillblains may prevent these if, beginning in September or October, they practise vascular gymnastics

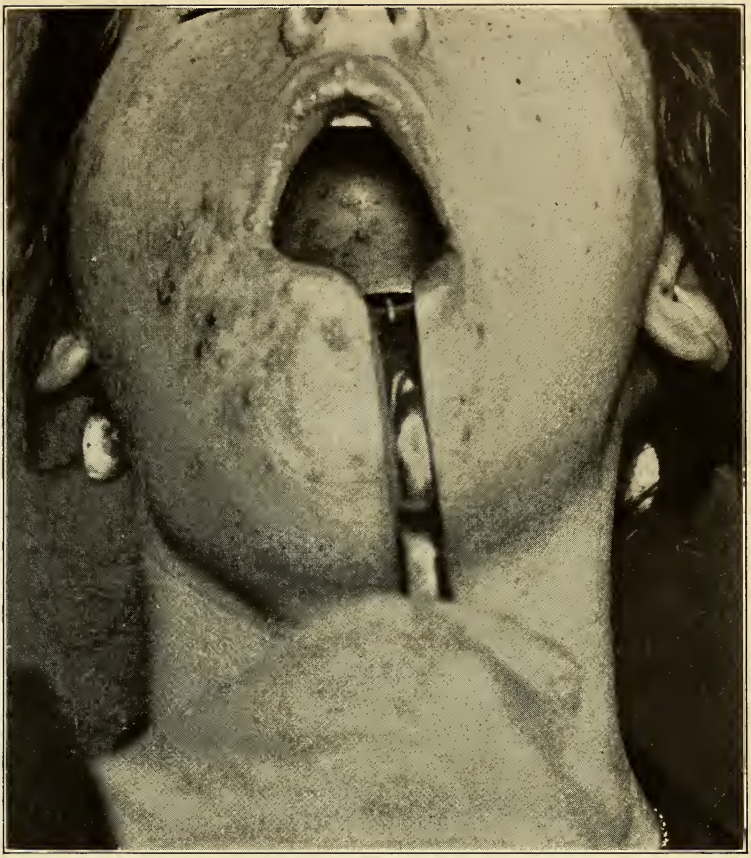

FIG. 9. HERPES

Herpes, also known as herpes simplex, cold sore or fever blister, consists of small areas of grouped vesicles, usually occurring on one side. The commonest location is on, or near, the lips or nose. At times, as in this case, the buccal mucosa is involved either alone, or in conjunction with the skin. At times, the genitals are involved. In contradistinction to zoster, herpes is recurrent and symptomatic, often being secondary to an acute infectious disease, notably pneumonia; or following the use of drugs, notably arsenic. Simple herpes is usually qualified in description by the name of the site, as herpes labialis, progenitalis, etc.

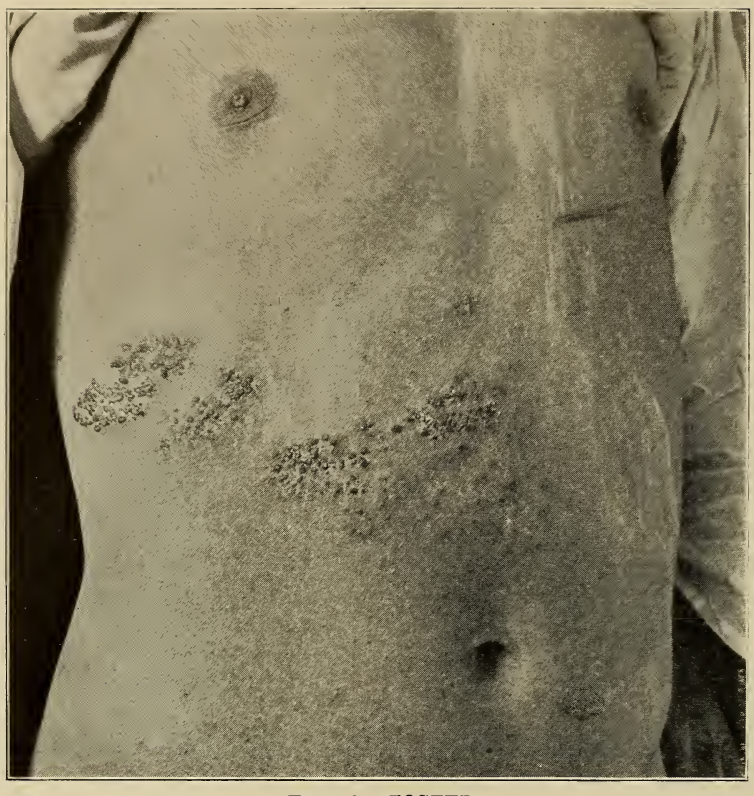

FIG. 10. ZOSTER

Zoster, herpes zoster or shingles, is unilateral and follows the distribution of a cutaneous nerve. It thus overruns the mid-line, as this picture shows. The lesions are grouped vesicles. In true zoster, recurrences are rare, but in symptomatic forms, due to arsenic, or perhaps focal infections, there may be recurrences.

FIG. 11. DERMATITIS (ECZEMA), ACUTE VESICULAR

Except for the large bleb on the third finger, this is a typical example of any acute exudative inflammation of the skin, whether called dermatitis or eczema. Any type of external irritant whether chemical, physical or bacterial or fungus; or any internal condition, capable of producing this form of skin reaction, produces this picture. It might equally well be due to rhus, epidermophyton, or a hypothetical metabolic derangement.

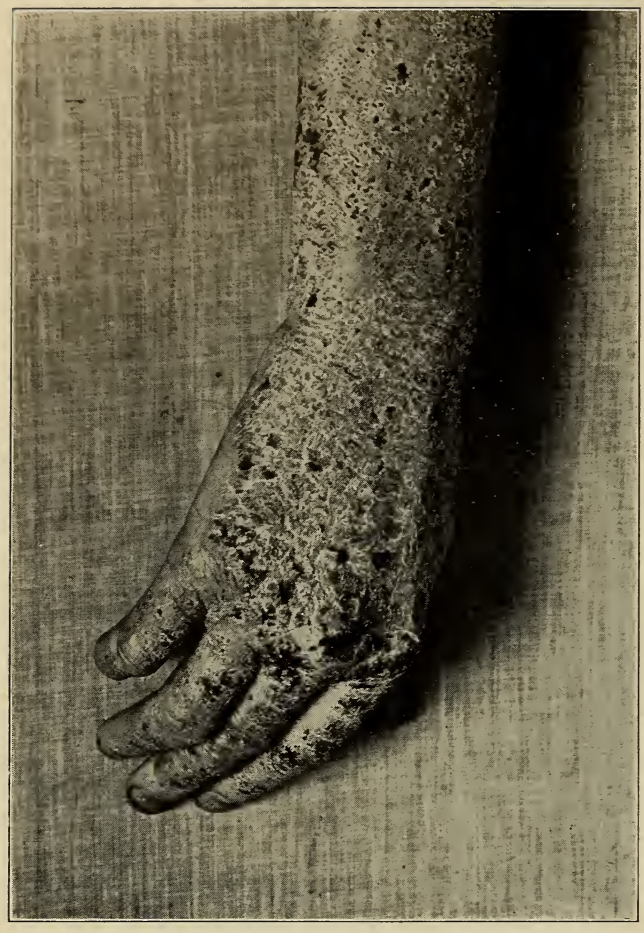

FIG. 12. DERMATITIS (ECZEMA), CRUSTOSA OR MADIDANS

Involuting simple dermatitis crusts and cracks. In chronic forms, the skin is thickened. All of these features are here illustrated. Crusts are due to serous discharge or weeping.

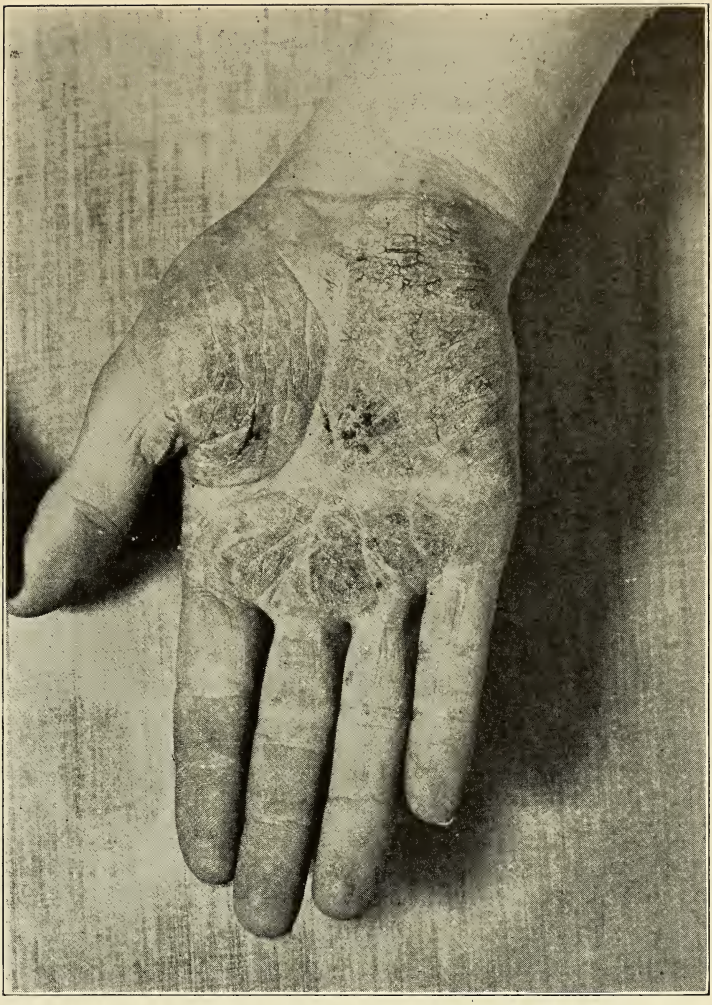

FIG. 13. DERMATITIS (ECZEMA), PALMS

The integument is thick, scaling and fissured. It differs from palmar syphilis in that there are no nodules or large subepidermal papules, and there is no festooning of the margins. The soles are similarly affected in this form of dermatitis.

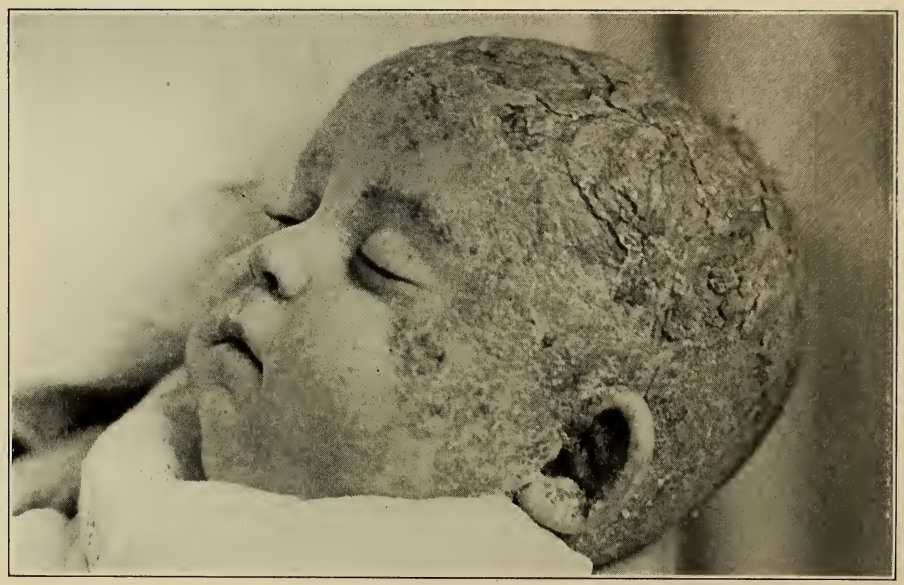

FIG. 14. DERMATITIS (ECZEMA) IMPETIGINOSA

The crusting, the unruptured vesicles, as seen on the cheek, and evidences of scratching here and on the forehead (small, dark points), are characteristic.

FIG. 15. DERMATITIS (ECZEMA), THICKENED OR LICHENIFIED

This illustrates the cutaneous change determined by sustained irritation. It is secondary to habitual scratching or rubbing to relieve chronic pruritis; or it results from any chronic inflammation. In this instance, the cause was specifically dermatitis (eczema). Another term is lichenification. The process may be secondary; or, if no ascertainable cause is found, may be primary. In the latter event, it is closely related to, if not indentical with, neurodermite or the so-called lichen chronicus simplex.

FIG. 16. POMPHOLYX

This condition is also called cheiropompholyx and dishydrotic dermatitis; although there is no real association with sweat disturbance. It is a vesiculobullous, scaling and crusted dermatitis, and was originally confused with dermatitis venenata or eczema of metabolic origin. In recent years its frequent causation by fungi has been established. Often the hands, groins and axillae are involved.

as to the parts usually affected. Thus, the feet every night should be plunged alternately five or six times, first into extremely hot and then into iced water. Before and after the hydrotherapy the members should be massaged with lanolin or vaseline.

DERMATITIS FACTITIA

Synonyms. Hysterical, Neurotic, or Spantaneous Gangrene; Feigned Eruptions.

These manifestations are self-inflicted cutaneous injuries provoked by the use of caustics, acids, alkalies, various implements, or heat. Erythemas, vesicles, blebs, superficial gangrene and necrosis are thus caused to appear on areas within the reach of the patient's hands. The diagnosis depends upon the lack of similarity of the lesions to those of recognized dermatoses, and upon the fact that areas are free outside of the range of the patient's hands. Often, when caustic fluids have been employed to cause the disease, the line of flow indicates its artificial nature. Patients have been known to simulate pemphigus, and other skin diseases. Malingerers, mendicants, people endeavoring to mystify physicians, to excite sympathy, or to evade their duties, are among those in whom the disease occurs. At times, no actual reason may be ascertained, but in any event the malady indicates mental or moral obliquity. The treatment consists in skillfully ascertaining the nature of the disease and confronting the patient with the facts. Actual treatment of the lesions varies with the nature of their cause, but roughly corresponds with that of dermatitis or traumas.

DERMATITIS REPENS AND ACRODERMATITIS PERSTANS

These two diseases are probably identical. Both begin after a trauma, some occupational injury, a pin-prick, a surgical operation or burn. The disease usually starts on a finger or toe, with a more or less persistent vesicle or bleb, although frequently without such a prodromal lesion. Peripheral spreading of vesicles, and undermining of the advancing margin, central denudation of the epidermis, with more or less weeping, characterize the process. At times, a whole extremity may become involved before healing finally takes place. In acrodermatitis perstans, after healing, the bleb tends to return at the original site and the process begins afresh. In this disease, too, distant areas may be affected, apparently by inoculation. The cause of the disease is probably an infection and the treatment consists of that of dermatitis, and the use of strong silver solutions.

CHAPTER X

BULLOUS DERMATOSES

The bullous dermatoses are etiologically not related. Indeed, their causation is not definitely explained and they are associated solely because of certain similar clinical points. Pemphigus, dermatitis herpetiformis, epidermolysis bullosa, and hydroa vacciniforme with their subclasses constitute this group. Other and not related dermatoses have bullous phases, notably erythema exudativum bullosum (Chapter VI), urticaria bullosa (Chapter VII), impetigo contagiesa (Chapter XXIV), variola and varicella (Chapter VI), lepra (Chapter XXV), and syphilis (Chapter XLII), and certain drug eruptions of both external and internal causation. The common feature of the group is the bulla, the characteristics of which have already been described (Chapter IV). These lesions again illustrate the doctrine of skin reactions, as do wheals and vesicles, and in another day when the nature of dermatoses is better understood, so superficial an attribute as the appearance of a lesion will cease to suffice as a bond for associating maladies which are fundamentally unrelated.

PEMPHIGUS

Synonyms. Pompholyx,[1] German Blasenausschlag.

Pemphigus is an acute or chronic bullous dermatosis occurring in several varieties, characterized by clinical or prognostic differences. *Ormsby* recognizes four types: pemphigus acutus, vulgaris, foliaceus, and vegetans. *Finger* recognizes benign pemphigus, or pemphigus vulgaris, and malignant pemphigus of which there are four types: the acute malignant, pruriginous, foliaceus, and vegetating. These two will serve as examples of the usual ways in which the disease is classified. For simplicity, *Ormsby's* makes the greatest appeal.

PEMPHIGUS ACUTUS

Also known as Acute Febrile Grave Pemphigus, or Acute Infectious Bullous Dermatitis.

[1] This term is obsolete as applied to pemphigus; it is commonly used as an abbreviation for cheiropompholyx.

Definition. Acute pemphigus is apparently an infectious disease characterized by a prodromal period and an eruptive stage. It is usually fatal, or goes into a chronic form conforming to the vulgar, exfoliating, or vegetating types. In rare instances the disease is supposed to end in recovery.

Symptoms. After an inaugural stage of malaise, chills and fever, an eruption of blebs appears. The lesions vary in number and size. There may be but few, or they may be numerous and widely disseminated, including the visible mucous membranes, and they may be as small as lentils or as large as an egg. At first, the contents are clear and serous; later they become cloudy and purulent, and when they rupture crusts form. At times the mucosa is spared; at times even the conjunctivae are involved, causing edema of the lids. Blebs in the mouth usually rupture promptly and their residue appears as a superficial gray lesion at times covered by a membrane. At times the blebs contain blood as well as serum. As a rule the lesions arise from inflamed skin, but occasionally a red halo surrounds their bases. The prostration is frequently severe, and high temperature, at times even hyperpyrexia has been noted.

Course. The course varies. Beginning as an acute infectious disease, a bullous eruption develops. The duration of the malady may be from several days to a few weeks and it usually ends fatally. At times the disease merely represents the beginning of a chronic type.

Varieties. There is a supposedly benign form, which, however, may not belong with this disease, but rather with pemphigus vulgaris.

Differential Diagnosis. Only dermatitis herpetiformis, erythema bullosum, urticaria bullosa, varicella and variola may be confused with this malady. Dermatitis herpetiformis never starts so suddenly, there are slight or no constitutional symptoms, the course is prolonged, and the lesions fairly characteristic. (See below.) Erythema bullosum is mild, and lesions characteristic of the erythema group are present. (Chapter VI.) The presence of wheals and factitious urticaria differentiate hives from pemphigus. Varicella is a disease of childhood and crops of papules as well as umbilicated blebs are present. Variola is the disease most likely to simulate acute pemphigus. Variola occurs in epidemics, as a rule is divided definitely into well-recognized stages (Chapter VI). the fever curve and double period of eruption, the umbilication of the blebs, should all render the diagnosis relatively simple.

Etiology and Pathogenesis. Very little is known of the cause

of the disease. After wounds, in those engaged in handling dead animals, preeminently in butchers and anatomists, the syndrome appears so that there is a great possibility of its being due to infection. It may possibly be related to foot and mouth disease as was suggested by Bowen twelve years ago.

Treatment. Quinine and arsenic have been used. Symptomatic, in want of rational, therapy is all that there is to offer. One is utterly helpless before the problem of treating pemphigus.

Prognosis. The patients usually die.

PEMPHIGUS VULGARIS (FIG. 17)

Definition. This is a rare chronic eruption of bullae, few in number as a rule, extending over a period of months or years.

Symptoms. After a period of slight malaise, or without prodromata, a few blebs (Fig. 17) arise which either dry, or break and crust. The lesions are tense or flaccid, contain serum, pus, or blood, or some combination of the three, always, however, at first serum alone. Successive crops of one or more blebs arise either just before, just after, or at a variably long interval after a given series has healed. Thus, at times there is a polymorphous picture,— blebs and crusts, the color determined by the contents, and pigmentation where the lesions were. There is no inflammatory zone about the bullae. On the visible mucosa and conjunctiva ruptured blebs with grayish bases are often to be observed. Accompanying the outbreaks, there may be a rise in temperature of from two to four degrees, and considerable prostration, decompensation of the heart, nephritis, enterocolitis, and a slight hyperleukocytosis and relative increase in the neutrophiles.

Course. The disease may last for months or years, and end as an acute, grave, or as foliaceus, or vegetating pemphigus. Comparative health may persist for a long time before the more serious phases of the illness develop.

Varieties. The varieties of this form of the disease are those of incidence, such as pemphigus solitarius when but one lesion recurs at a given site, or pemphigus diutinus, disseminatus, hemorrhagicus, or conjunctival or buccal pemphigus. *Dr. Martin Cohen* referred a case of pemphigus vulgaris of the conjunctiva and cornea to the Dermatological Clinic at the New York Post-Graduate Hospital. This patient, a middle aged widow, also had buccal lesions. *Fabry* in 1915 reported similar ocular lesions in pemphigus foliaceus.

Differential Diagnosis. Because of the character of the lesions,

their non-inflamed base, the slow course of the disease, and the absence of other signs of multiform erythema or urticaria (the only two conditions which might cause confusion) the diagnosis of pemphigus vulgaris is simple.

Etiology and Pathogenesis. Actually, nothing is known of the cause of the disease. There are three theories as to its origin, infectious, toxic or metabolic. A series of micro-organisms has been described, the bacillus pyocyaneus, and several peculiar organisms reported by *Lipchütz* in 1911 ad 1912. No confirmation exists. *Johnston* favors the metabolic view, and this appears reasonable but remains unproved. In an analysis of thirty cases, neither age, sex, occupation nor general health had any relation to the disease. Twenty-nine of the patients were Hebrews. No laboratory work threw any light on the etiology. Dr. Hermann Goldenberg, with whom I had the pleasure of collaborating, considers the disease infectious.

Treatment. Arsenic is supposed to be of value, and in the case of conjunctival pemphigus noted above, appears to have been. On the basis of its being a toxic disease, colon irrigations are indicated, and tonic treatment should be used. The local therapy consists of employing dusting powders, Lassar's paste and diachylon ointment, and perhaps the permanent bath as it is employed in Vienna.

Prognosis. All of the thirty cases mentioned above died. Among them were about a dozen patients with pemphigus vulgaris. The patients may survive for years, but they ultimately succumb to an incurrent infection, cachexia, or a general lighting up of the process. At times the condition evolves into pemphigus foliaceus or vegetans. A benign form is known which after weeks or months disappears. It is questionable whether this is really pemphigus for it is almost unanimously agreed that pemphigus is always fatal.

PEMPHIGUS FOLIACEUS (FIG. 18)

Definition. Pemphigus foliaceus is a rare form of pemphigus in which the primitive lesions are flaccid bullae which appear in great numbers and lead to an almost universal exfoliation, so that the condition actually appears to consist of a mixture of blebs and large scales.

Symptoms. The onset may be preceded by prodromata such as weakness, malaise, mild fever, one or two blebs (Fig. 17) on the body or in the mouth, or the disease may start suddenly as pemphigus acutus, or gradually with the clinical picture of dermatitis herpeti-

formis, or after pemphigus vulgaris. A patient with the disease in its typical aspect presents a fairly characteristic picture. The facies are anxious, and an impression of great suffering is imparted, due to the actual pain in the skin, the discomfort of the inflamed integument, and the agony caused by friction of areas denuded of epidermis. Analysis of the lesions reveals several points. Isolated flaccid blebs of sudden evolution are present. These discharge their contents of seropurulent secretion which in drying forms fragile crusts. Below the latter, as well as below the blebs themselves, inflamed skin denuded of its epidermis is seen. This bleeds slightly. As the lesions are covered with ill-formed epithelium, new blebs develop below, lifting off the imperfect covering. Thus, large stratified scales and crusts are formed. Besides this, the epidermis easily peels off even without the presence of blebs. This is known as *Nikolsky's* sign and is due to serous saturation of the epiderm, lifting the upper layers of the latter aloft. The scales thus formed dry rapidly and resemble pie crust (Fig. 18). The picture consists of two essential elements, bullae and scales. As the disease extends, the entire skin is involved, and it all appears to be exfoliating, the surface below being red, and at the margins of the scaling areas residual bullae are seen with their crusts and scales. The mouth, vulva and body folds are included, and moisture causes maceration. Fissures (Fig. 18) form all over. The extremities swell, as do also the ears, which thus lose their normal contour, the eyelids grow puffy, purulent conjunctivitis often develops, the nails become distorted and may even fall, while the hair usually falls out also. In the course of time palpebral ectropion develops, and at points of pressure decubiti may arise. An odor suggesting the charnel house is common in pemphigus foliaceus.

Fever accompanies the picture, but at times when the patient appears well it is as high as 103 or 104, while during periods of extension it may be normal. There is no relation between the fever and eruption, nor is there anything typical about the curve. It is purely capricious and simulates neither the fever of typhoid, malaria nor sepsis. Bodily function may be undisturbed for prolonged periods, but diarrhoea commonly is present, and the evacuations are often foul and contain mucous. Vomiting, too, is not rare and renal involvement may also be seen. Restlessness is common and periods of mental flightiness are not infrequent. In a case I had the opportunity to observe closely the patient at first appeared well, the appetite was good and the intestinal function regular. After

ten weeks the stools became clay-colored, these alternating with foul green evacuations. Two weeks later the feet became edematous, and a day or two after this albumin and hyalogranular casts were found in the urine. In a fortnight more there was glycosuria which lasted for two days; then, suddenly for no reason, the albumin and casts disappeared and the diarrhoea became controlled by the use of paregoric. Preceding this chain of developments the afternoon temperature had been between 101 and 102; as the disease grew severer the temperature became and remained normal. The pulse in pemphigus foliaceus is weak and rapid, about one hundred and twenty, the respirations about twenty-six and the leukocyte count about ten thousand, but the relative proportion of the various white blood cells remains unchanged.

Course. The disease lasts for weeks or months, exceptionally for years. Exhaustion, intractable enterocolitis, or an intercurrent infection, often pneumonia, sometimes sepsis, ends the patient's agony.

Differential Diagnosis. The presence of blebs, exfoliation, and *Nikolsky's* sign characterizes the disease. Dermatitis exfoliativa (Chapter XII) may remotely simulate it, but the points above enumerated should prevent error.

Etiology and Pathogenesis. It is valueless to speculate on the probable cause of the malady. It is obviously either toxic, metabolic or infectious in origin, but there are insufficient data to justify belief in one possibility to the exclusion of the others. The bacillus pyocyaneus has been regarded by *Hazen* as the cause in some cases.

Treatment. The treatment is similar to that of pemphigus vulgaris and quite as expectant and futile.

Prognosis. Death is inevitable, usually no later than within two years of the onset.

PEMPHIGUS VEGETANS

Synonyms. Pemphigoides Maligna, Herpes Vegetans, Condylomatosis, Erythema Bullosum Vegetans.

Definition. Pemphigus vegetans is a rare (about seventy cases on record) malignant illness, characterized by the formation of bullae from the bases of which spring vegetations which become condylomatous.

Symptoms. The disease begins in one of two ways. Either there are prodromata such as malaise, languor and mild fever followed by bullae in the visible mucosa of the body orifices, or the early stages of the condition simulate pemphigus vulgaris, or derma-

titis herpetiformis. In any event the eruption soon limits itself to the mucosa at the sites mentioned, the axillae, elbow flexures, groins, base of neck, umbilicus and the cutaneous areas immediately adjacent. Any or all of the sites enumerated may be involved simultaneously. Most frequently, however, the process is for a long time restricted to the mouth, anus, or vulva. The lesions themselves are at first bullae of various sizes, and are numerous and close-set, or sparse. They are either tense, containing serous fluid, or flaccid, containing pus. They soon rupture and on mucous surfaces are covered with a dirty gray membrane, while on the skin they are crusted. Underneath the membrane or crusts arise papillomatous or condylomatous vegetations, surrounded by a red zone, and in this stage the flat, macerated, vegetating papules of syphilis, called condylomata lata, are closely simulated. An overpowering fetor is exhaled from the mouth, and the lesions elsewhere have an equally revolting odor. The nails and hair may fall out.

Course. The progress is one of alternate periods of remission and recrudescence, with each accession severer than its predecessor, until after months or years the patient succumbs to asthenia, intercurrent infection, or to the severity of the illness itself. There is no regular fever curve characteristic of the disease, although more or less increase in temperature at times exists without, however, in any way running parallel to the severity of the illness. Occasionally, periods of vomiting, diarrhoea, coughing and the like arise, and do not seem to bear any direct relation to the disease.

Varieties. There are no true varieties of the disease other than those due to accidents of site, severity, intensity, and height of the fever, save that there are two ill-conceived groups of cases, the malignant and benign. The former is true vegetating pemphigus; the latter is not pemphigus at all, but probably vegetating dermatitis herpetiformis.[1]

Differential Diagnosis. Only syphilis simulates this malady,

[1] This distinction will hold good as long as we differentiate between pemphigus and Duhring's disease. Evidence is accumulating, however, that there is no great difference between the two diseases. The description of pemphigus given in the text reflects the accepted views. In an analysis of thirty cases studied by Dr. Goldenberg and me in the wards of the Mt. Sinai Hospital we found that the disease was always fatal, nearly always began in the mouth, and that pemphigus vulgaris always preceded the foliaceous and vegetating variety. We thus regarded the described types as clinical variants of a single disease, and we gained the impression that its causation was more likely infectious than anything else. Cases simulating pemphigus that recover are not pemphigus. Hence in this sense dermatitis herpetiformis and pemphigus may be divorced.

and actually it is the lesions alone of which this is true, not the entire process. The benign nature of syphilis, other characteristic lesions thereof, the positive Wassermann test, the presence of spirochaetae in syphilitic condylomas, the absence of blebs as well as other signs of pemphigus eliminate confusion.

Etiology and Pathogenesis. Nothing is known of the causation of the malady. The long array of alleged causative microorganisms, the numerous conventional explanations of the condition, amply indicate the incorrectness of any.

Treatment. There is no rational therapy. It is possible only to confine our efforts toward increasing the comfort of the patient and maintaining asepsis as to the parts affected, along the lines laid down in pemphigus vulgaris.

Prognosis. The disease is fatal.

DERMATITIS HERPETIFORMIS (FIG. 19)

Synonyms. There are numerous other names for this disease. It is important however to remember only Herpes Gestationis, Duhring's Disease, and the French term, Dermatite Polymorphe et Douloureuse.

Definition. The disease is uncommon, if not actually rare, and is characterized by an eruption of vesicles, blebs, pustules and papules which appear in several crops, variable grouping, and inconstant distribution. They are attended by burning, pain or itching of variable intensity. Occasionally, malaise or slightly more marked systemic disturbances, even low fever accompany the picture. From this the French term, painful and polymorphous dermatitis, appears the most aptly descriptive.

Symptoms. The onset of the disease may lack constitutional symptoms, but more often is characterized by malaise, chills, chilliness, fever, and at times rigors followed by itching, burning, or pain. Within a day or two a polymorphous (Fig. 19) exanthem appears consisting of macules, papules, tubercles, vesicles or blebs, either on the skin alone (Fig. 19), the mucosa or both. The possible combinations and number of these lesions are boundless, and they may be restricted, universal, disseminated, discrete, coalescent, or grouped in a manner suggestive of herpes. Features of multiform erythema, pemphigus and acute vesicular dermatitis are simulated. In long standing cases the extensor surfaces of the limbs, the sacral, trochanteric areas, and shoulders are involved. As one crop involutes others arise, and new lesions tend to group themselves about the

remains of the old so that festooned, circinate and gyrate areas develop, consisting of one or more of the component types of lesions. When the mucosa is involved the lesions are indistinguishable from those of pemphigus. This is also often true of the skin. The bullous lesions dry and form crusts and occasionally become purulent, in which case the crusts assume the characteristics of dried pus. In addition to this the papular lesions may be scratched open, and therefore the picture is further complicated by the presence of excoriations. Areas of lichenification too may develop (Chapter XIII). Although the process itself causes no scarring, at times deep scratching and local infection may leave superficial scars which not rarely are pigmented or depigmented.

Course. The illness may last for months or years and is noteworthy for its varying periods of activity, partial or complete quiescence. Although usually it does not shorten life, occasionally malignant forms end fatally, simulating either variola, or exfoliative dermatitis. Such cases are indistinguishable from malignant pemphigus vulgaris, or pemphigus foliaceus.

Varieties. A form of this disease seen during pregnancy is known as herpes gestationis. Another variety, impetigo herpetiformis, also almost entirely restricted to this class of patients, is very severe. It is characterized by an exanthem of pinhead pustules with yellow or green contents, and an inflammatory base. The buccal mucosa is also involved, and half of the patients die. A few cases have been observed in men.

Differential Diagnosis. To differentiate this from pemphigus is almost impossible. Only observation of the patient throughout his life can lead to a positive conclusion. It is known that many cases of pemphigus begin as dermatitis herpetiformis, and that many cases of dematitis herpetiformis end with the clinical aspect of any form of pemphigus. Only the polymorphous attributes of Duhring's disease indicate the possibility of its being a clinical entity, and I am not convinced that this alone is sufficient to separate it from pemphigus, nor has the Viennese school ever accepted the distinction. Although the terminal stages of malignant dermatitis herpetiformis simulate variola, the case history makes confusion impossible. Simple dermatitis resembles dermatitis herpetiformis only as to certain lesions, but the picture in its entirety is quite different, unless lichenification arises. Then chronic dermatitis (eczema) is mimicked, but only remotely. Bullous multiform erythema is sometimes hard to differentiate from dermatitis herpetiformis, but

the typical lesions of the former interspersed among the bullae should indicate, if not actually determine, the correct diagnosis.

Etiology and Pathogenesis. No one explanation has yet been accepted to account for the disease. *Johnston* and *Schwartz* have found nitrogen retention preceding outbreaks in chronic cases. The usual sophisms concerning reflex neuroses and the like still have their advocates, as is always the case when the etiology of a disease is unknown. Herpes gestationis may be due to pregnancy, but there is no proof of it yet, and it is more likely simply to be dermatitis herpetiformis coincident with pregnancy. Although still unproven, impetigo herpetiformis is evidently a systemic infection. The cases are rare and blood cultures have not yet been done, but the behavior of the patients suggests the probability of sepsis with pyoderma probably due to bacterial embolism.

Treatment. Arsenic is the only efficacious drug in this illness and more recently it has been the experience of all that cacodylates have been the most useful. Injections should be given every other day starting with one or two grains and increasing the quantity to the point of tolerance. Salicylates and thyroid extract have been praised by some. *Johnston* forbids excessive proteid diet in cases with nitrogen retention. The local treatment corresponds with that of pemphigus. In impetigo herpetiformis induction of labor is indicated, and symptomatic local therapy, as in pemphigus.

Prognosis. In dermatitis herpetiformis, and excepting in malignant cases, or in those cases which evolve into pemphigus, the prognosis as to life is good, and as to a permanent cure almost uniformly bad. About half of all of the cases, either correctly or incorrectly regarded as dermatitis herpetiformis, die. It is probable, however, that those ending fatally do not belong in this group, but are pemphigus.

HYDROA VACCINIFORME

This disease is also known as recurrent summer eruption, or herpes aestivalis. It is a vesicular disease restricted almost entirely to boys and the lesions appear on exposed surfaces (hands, face, and forearms) every summer. Either one or more crops come with the hot weather, and the characteristic lesions are vesicles or small bullae which break, crust, and leave depressed pigmented or non-pigmented scars. With adolescence the disease ends. It is due to the effect of actinic rays on a skin, either sensitized to their action, or lacking some protective elements which make normal skin resistant. There is no cure for the condition during childhood, but spontaneously at adolescence the attacks cease.

EPIDERMOLYSIS BULLOSA HEREDITARIA

This is a rare hereditary and familial disease characterized by the formation of bullae which arise as the result of the slightest trauma. The bullae are flaccid, rupture easily, and leave depressed scars. Gradually, the entire body may be covered. The general health remains unaffected by the disease, although there is no cure known for it. It is hereditary and due to a malformation of the elastic tissue. Usually, the first signs of the malady appear shortly after birth, at times, however, later in life. *Wise* has recently described *epidermolysis bullosa acquisita* appearing in adults and resembling the hereditary form clinically, but not etiologically.

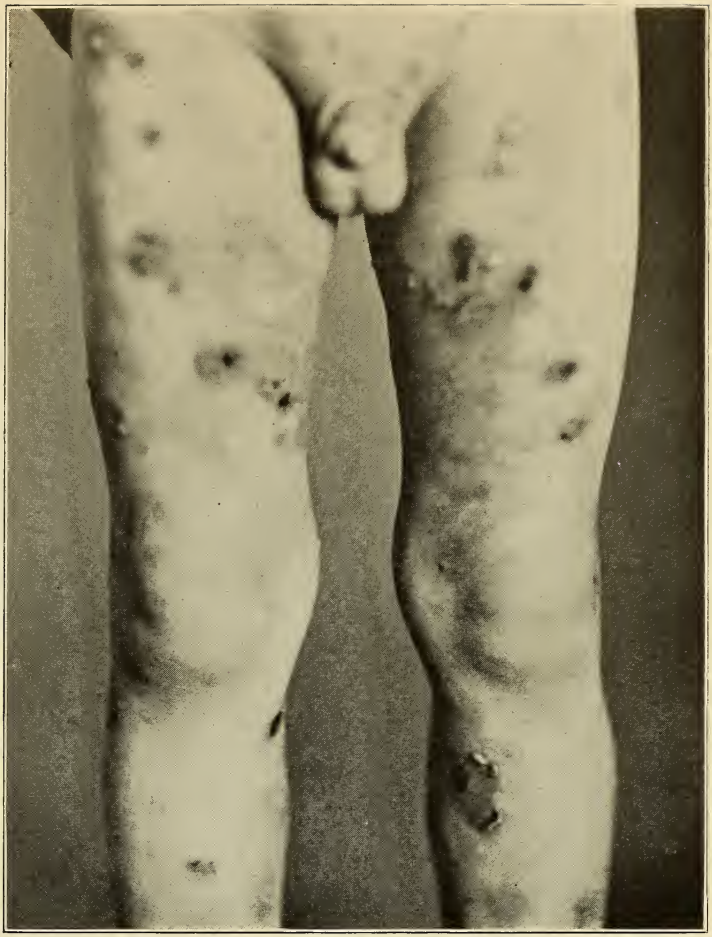

FIG. 17. PEMPHIGUS VULGARIS

Unruptured bullae, without inflammatory base, appear near the groin. A ruptured one is seen below the left knee. Here and there are seen healing, crusted and healed lesions, and hyperpigmentation. Other conditions suggested are dermatitis herpetiformis, bullous urticaria, bullous erythema multiforme, and bullous bromoderma, all excluded by the nature of the blebs near the groin.

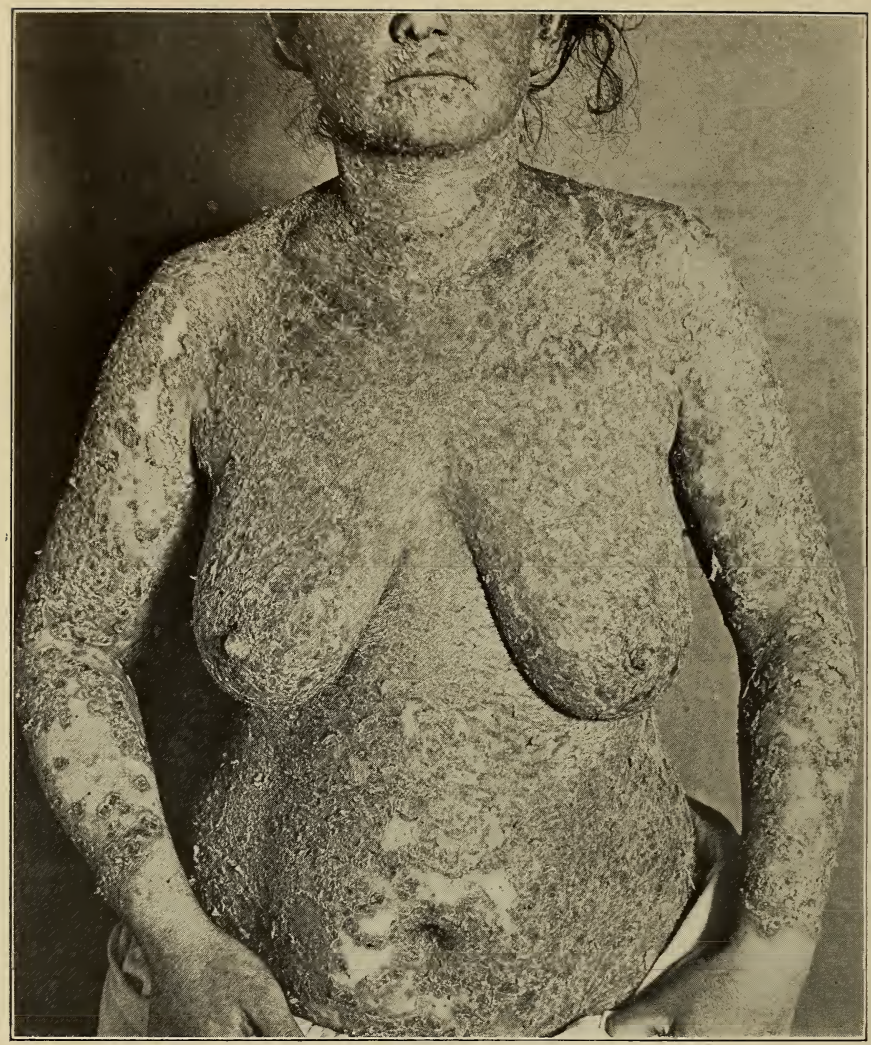

FIG. 18. PEMPHIGUS FOLIACEUS

In this type of pemphigus, the entire skin peels in large flakes. The integument is red and tender, and exfoliation results from large, flat, flaccid blebs. Dermatitis exfoliativa of the Hebra and Wilson-Brocq type differ in the absence of blebs, mucous involvement and greater thickening of the skin.

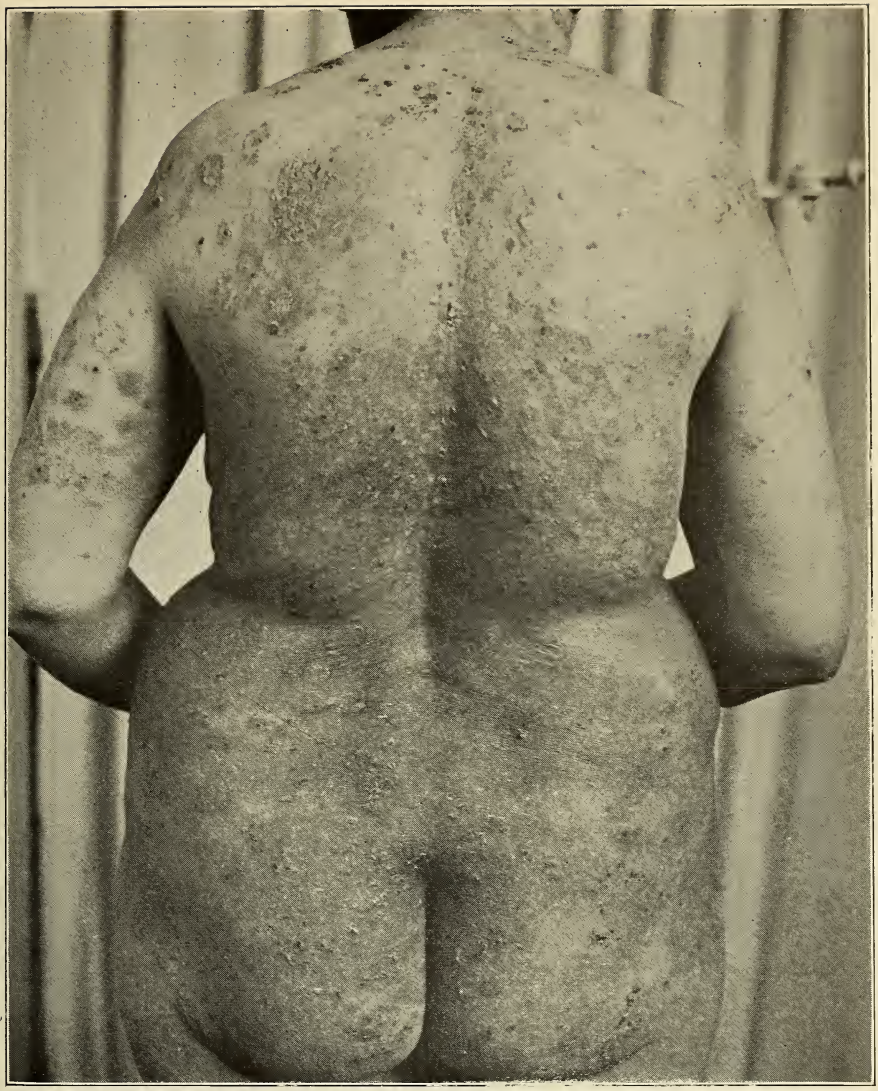

FIG. 19. DERMATITIS HERPETIFORMIS

This rare disease is also named for Duhring, and called polymorphous and painful dermatitis in France, a most descriptive term. There is grouping of vesicles, as over the left arm and shoulder, and bullae and papules appear as between the scapulae. Crusting, scaling, festooned margins, patches, excoriations, and lesions in all phases of development and disappearance are encountered. A certain relationship to pemphigus is admitted. Itching, burning and pain are subjective features.

GROUP III. CUTANEOUS REACTIONS CHARACTERIZED BY SCALING OR LICHENIFICATION

Three classes of dermatoses constitute this group — the psoriasiform, erythrodermal and lichenous. They are all clinically characterized by scaling, the majority without any deep seated infiltration, some, however, having this attribute. The psoriasiform diseases are macular or maculo-papular, the macules often coalescing into placques in psoriasis itself, and sometimes in seborrhoea. The erythrodermas are usually diffuse. The lichens are infiltrated papules, and lichenification is a primary or consecutive infiltrating process.

CHAPTER XI

PSORIASIS AND THE PSORIASIFORM DERMATOSES

There are four grand classes of psoriasiform dermatoses: psoriasis, seborrhoea, parapsoriasis, and pityriasis rosea. Of these, only psoriasis is a clean-cut entity, standing by itself in the center of the group. Although perhaps premature at this point, it must be stated that parapsoriasis unites the scaling dermatoses with granuloma fungoides (Chapter XXVI), pityriasis rosea forms a link with exudative multiform erythema and the toxic dermatoses (Chapter VI), and seborrhoea is the starting point of numerous forms of simple dermatitis (Chapter IX), notably those classed as eczema.

PSORIASIS (FIG. 20)

Synonyms. Lepra alba, psora, alphos; German, Schuppenflechte.

Definition. Psoriasis is a scaling dermatosis, inflammatory in nature, usually chronic in course, more frequently gradual than sudden in onset, and characterized primarily by a red papule surmounted by white scales.

Symptoms. As a rule, psoriasis makes its appearance in the second or third decade of life. It also frequently begins late in adult life, and rarely in infancy. Although it may start with a sudden diffuse and generalized eruption, the vast majority of cases

begin gradually with the appearance of roseate, just perceptible papules, surmounted by a white, silvery, or nacreous imbricated scale (Fig. 20). These papules increase in size and number. The larger the papule, the deeper its color, so that there is a wide range of hues varying from barely recognizable pink to deep purplish or brownish red. The scales are usually not firmly adherent, but may be. More often it is easy to scrape them off with the finger nail. A bare, inflammatory zone surrounds the scale (Fig. 20), and the entire lesion is sharply demarcated from the normal skin. The lesion is infiltrated, and the older ones are distinctly raised, sometimes an eighth of an inch. They are impliable and somewhat harder than normal integument; in fact, frequently leathery (Fig. 21). At times the scales, particularly in older lesions, are yellowish or gray. Upon their removal, bleeding puncta appear which correspond to engorged papillae, and which are almost pathognomonic.

The sites affected are various. On the whole, the extensor surfaces are more profusely involved than the flexors, and in these forms which exhibit but few lesions, there seems to be a preference for the scalp, joints of the elbows and knees. Thus, both extreme limitation and wide distribution of the lesions are known. The sites rarely involved are the palms and soles, while the lips and other orificial mucosa never are.

A limitless series of aspects is a feature of the disease. It may consist of widely disseminated (Fig. 20), small, pale lesions, or such lesions in only certain areas. Or the spots may be larger, even veritable plaques (Fig. 21), all over or just on the favored areas; or, there may be a mixture of the types. This has caused the creation of a series of terms purely descriptive of accidents of size, shape and arrangement of the component elements; as *psoriasis punctata* (Fig. 20), when the lesions are tiny (Fig. 20); *guttata,* when they are the size of a drop of liquid; *nummularis* or *discoidea* (Fig. 21), when coin-sized; *circinata* or *orbicularis,* when they enlarge peripherally and heal centrally; *figurata* or *gyrata,* when the circinate lesions coalesce forming festoons; *geographica,* when the lesions simulate maps; *diffusa,* when large areas of the skin are involved; *rupioides* or *ostracea,* when the scales are thick and resemble oyster shells; *follicularis,* when about follicular openings; and *inveterata,* when persistent, thickened, or fissured. Of course, this is all purely artificial. There are also psoriasis acuta, chronica, pruriginosa, capitis, *et cetera.*

As a rule, itching is absent, but in some cases may be intense.

The general health usually remains good, and there are no chemical or biological alterations which are diagnostically characteristic of the disease, as the Wassermann reaction is characteristic of syphilis, or the blood picture of leucemia.

Although the scalp (Fig. 20) is a favorite locality for psoriasis lesions, the hair rarely falls. The scales on the scalp are generally powdery or crusted, and often the lesions are numerous. The nails are affected in one of two ways; either the papule develops under the nail raising it, or it arises in the nail bed, and as the nail grows it is distorted, being indented with minute pin-point depressions such as might be produced by puncturing paper with a fine point. The nails are often brittle, dull, or misshapen because of the involvement of the matrix during the developmental period.

For the sake of emphasis, certain features of the psoriasis papule will be recapitulated. It is dry, scaling, never weeps and rarely itches. Upon the removal of the scale hemorrhagic dots are seen, sometimes so minute as to require magnification to detect their presence.

Course. Psoriasis usually begins between the eighth and twentieth year of life, often later, almost never earlier, and is capable of following one of several courses. It may start suddenly with a widely disseminated papular rash, or insidiously with a few spots which enlarge and increase in number. There may be periods of remission during which the patient is apparently free for months or years, but close examination will usually reveal a few lesions somewhere — either on the elbows, knees or scalp. At the onset the lesions may be punctate or guttate, and later assume any of the aspects already mentioned. After the period of chronicity has set in, the lesions may persist indefinitely in any form and neither advance nor recede. The process is unattended by grave complications or sequelae, but in rare instances epitheliomata are said to have arisen from inveterate patches, particularly in patients who have received arsenic. Hyperpigmentation or depigmentation also are known, and this, too, may be connected with arsenic therapy.

Varieties. There are no varieties save those due to accidents of appearance, localization and course. Rarely, the lesions may become verrucoid.

Differential Diagnosis. Psoriasis is more or less closely simulated by syphilis, pityriasis rosea, parapsoriasis and seborrhoea, the exfoliating erythrodermas and premycosis, and remotely by pityriasis rubra pilaris, lichen planus, lichen simplex and ringworm.

Disseminated psoriasis resembles papular syphilis in the secondary stage. The psoriasis papule has definite attributes which that of syphilis lacks. The scale of the latter is not imbricated and silvery, but consists of a single layer, nor does its removal cause any punctate hemorrhages.[1] In syphilis, in this stage, other factors exist which are lacking in psoriasis, such as mucous patches, flat condylomas, glandular enlargement, and the positive Wassermann test, as well as constitutional symptoms. Syphilis, also, is prone to involve the palms and soles, and favors the flexor surfaces in general, while the hair often falls out.

Old plaques of psoriasis which are festooned, gyrate or circinate in character must be distinguished from similarly arranged forms of tertiary syphilis, chiefly tubero-serpigenous gummata. The color of psoriasis is more vivid, that of syphilis brownish or buff. Psoriasis scales are white, those of the late syphiloderm gray or muddy and almost crust-like. The consistency of the psoriasis lesion is leathery, that of the luetic lesion more distinctly a deep infiltration. Other evidences of syphilis may be present, particularly the serum reaction, and these serve to clear up the question. Instances in which confusion might arise in differentiating palmar or plantar psoriasis from squamous syphilis at these sites are infrequent, since psoriasis here is rare and syphilis common; the Wassermann reaction is absent in the former and likely to be present in the latter, and finally, the therapeutic test in syphilis by the use of salvarsan or mercury should establish the correct diagnosis.

Pityriasis rosea, parapsoriasis and seborrhoea will be discussed later in this chapter, and the points of differentiation will then be enumerated. Universal psoriasis may closely resemble the exfoliative erythrodermas, and the so-called premycotic dermatoses. Indeed, the most expert are at times at a loss to find points of differentiation, and only close study of the cases will lead to correct interpretation. In general, it may be stated that universal psoriasis runs a milder course as to general symptoms than do the erythrodermas. Psoriasis, finally, involutes at least partly, while the erythrodermas, although punctuated by remissions, grows progressively worse, the patient's general health deteriorating, while gradual wasting leads to death. It is hardly fair, however, to await a fatal issue in order to make a diagnosis, and the only points upon which to base an opinion of psoriasis would be the facts that somewhere on the body

[1] It is inadvisable to scratch syphilis papules with the finger nails, because of the danger of infection.

a few characteristic lesions of this disease might be demonstrable, the absence of general glandular enlargements such as are seen in many of the erythrodermas, and finally the pervading impression of good health.

Lichen acuminatus and lichen planus resemble psoriasis only when they are in the forms of coalescent patches. There is more infiltration than in psoriasis, more pruritus, and the color of lichen acuminatus is a rather more decided buff, while that of lichen planus is violet or purple. The scales are more adherent in the lichens, not silvery, and their removal does not occasion punctate hemorrhages. In lichen planus, too, there is often mucous involvement, which in psoriasis is wholly unknown, while in psoriasis the scalp and face are involved which never occurs in lichen planus. Finally, typical lesions of the various maladies clear up the diagnosis. (Chapter XIII.) Lichen simple chronicus, or the *neurodermite* of the French, also known as eczema papulosum or lichenification, for the most part favors the flexor surfaces, notably those of the knees and elbows, the small of the back and nape of the neck. Particularly the first two sites are rarely affected in psoriasis, while the scalp is affected by this disease, and practically never by any form of lichenification. The patches of lichenification are thickened, circumscribed, furrowed by transecting lines, red, buff, or violet in color, and outside of their periphery are minute, red, lenticular papules. Often, too, lichenification is associated with some other disease such as dermatitis, dermatitis herpetiformis, scabies, prurigo, and the like. Several forms of ringworm resemble psoriasis. Here the diagnosis must depend upon a demonstration of the fungus.

Etiology and Pathogenesis. Psoriasis is equally common in both sexes, in all social classes regardless of occupation and race, save that it is unusual in negroes. It rarely occurs before ten or after forty-five. Heredity, in the loose manner in which this factor has been conceived in medicine, has been held responsible. *Knowles,* however, found but six familial cases in hundreds which he studied and decided against heredity, a view with which I emphatically agree. A disease observed in four percent. of all patients with dermatoses must occasionally appear in several members of a family, and there is no more proof of the heredity of psoriasis than of colds in the head, in fact less, since colds are almost universal.

Pollitzer in a brilliant analysis of the subject favors the idea of infectious origin. There is no proof of this and all reasoning is purely by analogy. On the other hand, there is perhaps no con-

vincing opposite evidence. Rheumatism and gout have been widely regarded as the cause in this country, and particularly in England. In the first place, there is no uniformity in our knowledge of these two diseases. They are not alike, and both are so common that it would be astonishing if they were not occasionally associated with so ordinary an illness as psoriasis. Nor is there any logical evidence of a nervous origin. It is not a cutaneous neurosis, nor is it a trophoneurosis, for it has never been found coupled with lesions in the central nervous system. A certain suspicion exists, however, that it may be due to disturbed nitrogen metabolism, although the work of *Johnston* and *Schwartz* is against this. They made nitrogen estimations on urine in various stages of the disease and found no variation from the normal. On the other hand, a most exhaustive and pretentious study of the subject by *Schamberg* indicates an abnormal nitrogen retention. There is no direct evidence either that disturbances of the endochrinous glands are at fault, although the occasional instances of improvement following the use of thyroid point to the possibility of hypothyroidism, at least in selected cases.

It is a matter of common knowledge that involution and recrudescences occur in the disease without apparent rhyme or reason. Thus some patients are better or worse during given seasons; women may improve or get worse during parturition, lactation, or the menses; a change in diet appears to affect one group of patients favorably and not another, *et cetera*. From this, one must inevitably conclude that the oscillations in the course of the malady depend upon periodic and transitory changes in the body, or in the last analysis, that the disease is due to a disturbance in metabolism. Even if it should ever be shown that the cause is a specific microorganism, the facts just enumerated would still remain important in their causal relation to the process as shifting influences determining the fertility of the soil to the infection. Such an infection might be local or general. The latter view may be excluded because there are no evidences of a systemic infection as we to-day understand the interactivity of pathogenic agents and their hosts. After all, lepra and syphilis act differently, nor is there any evidence of a local infective agent in psoriasis, nor are there any serologic studies which serve to illuminate this point.

Cases present themselves with all kinds of associated diseases or conditions, as constipation, colitis, bronchitis, tuberculosis, malaria, syphilis, neurasthenia and many others; but it is rare to find one in which the psoriasis and general disturbance coexist in a manner which

to any reasonable being would suggest cause and effect. And yet there is no doubt that controlling a general disturbance often seems to ameliorate an associated psoriasis. The improvement, however, it must always be remembered, may simply be due to one of the curious remissions so characteristic of the condition, and neither to internal therapy nor to improvement as to the general existing disease. There is no dermatosis in which *post hoc ergo propter hoc* reasoning is less apt and more fraught with danger. In short, no definite views may exist as to its etiology for all such views must inevitably be born rather of faith than fact.

Treatment. The treatment of psoriasis must be considered under two heads, general and local. General treatment consists of diet and the internal administration of certain drugs, the choice of which will be determined by such indications as may appear to bear a causal relation to the disease. On the assumption that an excessive ingestion of nitrogenous food, or nitrogen retention, causes the condition, or some of its forms, it has become popular to restrict or entirely eliminate the proteid intake, including meats, fish, eggs, and vegetables high in nitrogen content such as beans, peas, lentils and the like. It has never seemed entirely rational to me to do this, as I know of no instances in which improvement on such diet has been occasioned in such a manner that it could not be explained either by efficacious local therapy, or a spontaneous remission. Such treatment, too, would be indicated if gout were present. *Bulkley's* rice diet represents an extreme in the application of these principles, justification of which has never seemed valid in view of results obtained. So rigorous a limitation of food causes symptoms of starvation, and one instance has come to my notice in which an over-rich carbohydrate diet rapidly made the patient's skin worse, and caused a carbohydrate fermentation and mucous colitis. When the patient was put upon a bland, mixed diet all of her general symptoms, as well as her extensive and infiltrated psoriasis vanished (the latter within a fortnight), although the only local remedy employed was vaselin to keep the skin pliable. Should the patient have chronic nephritis, of course the diet and elimination should be the cardinal factors in deciding upon the mode of therapy.

Various medicaments are employed, the favored of which are arsenic, the salicylates, colchicum and thyroid extract. Of these, only arsenic is very important. This substance is used orally or by injection. When administered by mouth the forms of choice are Fowler's solution, Asiatic pills or arsenic trioxide. The first is

given in the conventional manner, in increasing doses, or sodium arsenite may be selected. Arsenic trioxide is given in doses of 0.0016 to 0.0033, three times a day after meals in pills. Asiatic pills are a favorite preparation once popular in Vienna, and very good.

℞
Acid arsenosi 0.75
Pulv. pip. nigrae 6.00
Gum. accaciae 1.50
Pulv. altheae rad. 2.00
Aquae qs. .....
M. Div. in pill No. C.

Roughly each pill contains of arsenious acid Gr. 1/8 (0.0075). One pill should be given after each meal and the daily number increased by one until the patient reaches the point of tolerance. *Jadassohn* taught that arsenic alone would cure psoriasis, provided the patient were able to stand the required quantity, which might be extreme. Although there is no doubt that the substance is valuable, I am unwilling to take so optimistic an attitude regarding its utility. Sodium cacodylate is also useful and should be injected daily in increasing doses, beginning with one grain and stopping at the point of toleration. Symptoms of arsenical poisoning should be closely watched for and the drug stopped upon their appearance. The possibility of hyperpigmentation, hyperkeratosis and epithelioma should never be forgotten when arsenic is employed. Arsphenamin and atoxyl are useless.

The salicylates and colchicum are employed in rheumatic and gouty subjects with psoriasis. I am not sure that their use is reasonable, although no harm can arise from it. Within the last few years *Gottheil* has advised autoserum in this malady. He stands practically alone in his enthusiasm. Other investigators, notably *Trimble,* deny its value, and *Ravitch* and *Howard Fox* recanted in later papers enthusiasm expressed in earlier ones. At present the chief claim of *Gottheil* is that autoserum enhances the efficacy of chrysarobin. Ringer's solution and normal salt solution have been administered intravenously without benefit. Thus the only internal therapy having approximately universal approval is the employment of arsenic.

Local therapy consists of the use of medicaments capable of removing scales and infiltrations by producing a more or less severe dermatitis. Such substances are known as revulsives and keratolytics and those which are of greatest efficacy are chrysarobin and some

of its derivatives, pyrogallol and some of its derivatives, salicylic acid, certain alkalies, sulphur, resorcin and the tars. Ammoniated mercury is also of great value and radiotherapy has a wide field of utility. The choice of substances and the form in which they are to be dispensed is determined by the extent, size, depth of infiltration and site of the lesions.

Before describing the methods of applying the materials enumerated their various general characteristics and modes of use must be outlined. Chrysarobin is an irritant, particularly in the presence of water. It is as bad in acute psoriasis as it is good in chronic, whether the latter be extensive or limited. It cannot be used upon the hands, face, or scalp, partly because it stains the skin brown and partly because, when brought in contact with the eyes, there is danger of exciting marked cunjunctivitis. It rapidly removes the eruption, leaving at the site of the papule a white stain rimmed by a chocolate colored margin. Neorobin is a chrysarobin derivative discovered by *Schamberg* who finds it more efficacious and less irritating than other similar substance. Pyrogallic acid is toxic, sometimes causing hematuria, or even fatal nephritis when employed over extensive surfaces. Thus, its value is confined to the treatment of small areas. Salicylic acid is particularly good in cases in which the scales are especially thick, as are also alkalies, the most useful of which is potassium hydroxide as incorporated in green soap. Sulphur and resorcin have a limited sphere of utility, at times hastening the involution of given lesions. Tar, particularly birch tar, oil of cade and pix liquida, of which the first is the best, are widely recommended. More recently carboneol (really technical coal tar) has come into use. The odor of these preparations is unpleasant and the tars and chrysarobin have the disadvantage of spoiling clothes. When applied universally in ointments dermatitis may be caused and even severe nephritis has been known to occur. It is safe, however, to employ it as did the Viennese a generation ago. Their method which was both pleasant and efficacious will be subsequently described. When tar works it has several advantages over chrysarobin, the most important being that it does not discolor the skin.

The precise methods of using the medicaments above outlined are as follows:

Chrysarobin. After a warm bath a five percent. ointment should be rubbed into the skin vigorously. Lanolin, or equal parts of this substance and vaselin, make a good base. This concentration may

be too weak in some cases and double or triple the strength may be required. Instances frequently arise in which even thirty or forty percent. salves are required, but this is exceptional and dangerous because of the possibility of general poisoning. Nor, as has been mentioned, should chrysarobin ever be used in acute forms, but only in the chronic, whether disseminated or localized, and whether the lesions are small or large. For localized patches the drug may be incorporated in flexible collodion or traumaticin, and painted on with a small, soft brush or cotton applicator. It is wisest to remove the scales first by washing with soap and water. In inveterate patches it is found useful to rub in the salve vigorously for ten or fifteen minutes with the blunt end of a clothes pin covered with chamois. At times the chrysarobin may be fortified with two or three percent. of salicylic acid. Neorobin is best used as a salve, or in any other manner in which chrysarobin is employed. It seems quite as efficacious as the latter and less irritating.

Pyrogallol. Except in isolated inveterate patches, this substance should not be used. When indicated, it is to be prescribed in ten percent. salves with any suitable base, and rubbed in vigorously once a day. In extensive cases eugallol (ten percent.) in acetone should be painted on once a day, after a hot soap bath. This substance does not stain the skin or clothes, dries almost as soon as applied, and often causes rapid involution of the lesions. It does discolor tiles and enamel, so that the patient should be cautioned not to spill it. I have seen it produce dermatitis only once.

Resorcin and *Sulphur* are valuable in psoriasis of the scalp. They should be prescribed in ointments, resorcin in two to five percent. strength and sulphur in from five to ten percent. An application should be made once a day, and the patient instructed to shampoo once or twice a week. Although in general tincture of green soap is one of the worst things on earth for the skin, it is valuable in psoriasis because it softens the scales. On non-hairy parts of the body sulphur is often employed, particularly incorporated in Wilkinson's ointment.

℞ Flor. sulphuris
Ol. Rusci .....aa 10.
Sapon. virid.
Adipis .....aa 20.
Cretae alb. 2.25
Sig.

This preparation should be rubbed thoroughly into the affected

areas, and the addition of a little water enhances its value. Salicylic acid may be added to sulphur and resorcin salves up to five percent.

Mercury. The ammoniated salt up to ten percent. is indicated in treating the scalp; the yellow oxide in treating lesions near the eyes. In the treatment of individual patches it is often useful to remove the scales, paint the affected area with a one percent. alcoholic solution of sublimate, and cover the lesion, after it is dry, with collodion. Salicylic acid when combined with ammoniated mercury enhances the latter's value. All of these substances are to be used once a day.

Tars. These may be used in simple ointments applied daily, or in combination with other medicaments, and in fluids. The first have already been described. They are most efficacious when applied after bathing. Wilkinson's ointment is an example of the combined variety, as is also Dreuw's.

℞ Acid salicyli
Chrysarobin .....aa 10.
Ol. Rusci .....20.
Sapon. virid.
Petrol. flav. .....aa 24.
Sig.

This is used precisely as is Wilkinson's ointment. Tar in fluid form is employed pure or diluted. An example of the latter is

℞ Ol. Rusci ..... 25.
Alcohol 95% .....ad 100.
Sig.

After a bath the patient is painted with the lotion whereupon he bathes again for from one-half to two hours. The tar is finally washed off and the skin anointed with a bland zinc salve. This is very efficacious treatment, does not stain the skin, and the patient does not go about smelling of tar. Pure tar often inflames the skin, and I prefer to use it diluted in alcohol because it does not irritate and dries more quickly. In scalp treatment, too, simple tar salves are useful.

Light Therapy. Fractional doses of Roentgen rays, one Holzknecht unit a week, are of great service. Care must be exerted when they are used in treating the scalp because of the danger of alopecia. The Uviol lamp, giving an ultra-violet ray, is of limited value, and the Kromayer lamp, which irradiates only small surfaces, may be employed in treating inveterate patches.

It may be of service to recapitulate the above from the standpoint of regional therapy. Psoriasis of the scalp may be treated with ammoniated mercury salve, or this combined with a little salicylic acid. Tar salves and lotions are also useful. On the eyelids or brows two percent. ammoniated mercury, or one percent. yellow mercuric oxide are useful. On the face, these and tar salves are indicated, and on all of the areas just mentioned chrysarobin is to be avoided. All other forms of local treatment outlined are indicated on the rest of the body, but on the delicate covering of the scrotum, penis and vulva, chrysarobin, pyrogallol and salicylic acid must be employed with the utmost caution.

PARAPSORIASIS

Synonyms. Resistant maculo-papular Scaling Erythroderma.

Definition. Parapsoriasis is a designation applied to a loosely united group of cutaneous diseases characterized clinically by an eruption of faintly yellow to buff, or pinkish-buff macules, papules, nodules or plaques, which have fine scales and which usually do not itch. According to *Darier* there are three main classes of this disease, and this point of view is generally accepted. These subdivisions are: (1) Parapsoriasis en gouttes; (2) Parapsoriasis lichenoïde; (3) Parapsoriasis en plaques.

Symptoms. (1) (Fig. 22) Parapsoriasis en gouttes, Brocq. (Guttate variety; Dermatitis psoriasiformis nodularis, Jadassohn; Pityriasis lichenoides chronica, Juliusberg.) On the trunk, arms and thighs pin-point to lentil sized papules develop, usually at adolescence. They are buff or red and resemble syphilitic papules, but scale rather more than the latter, practically never involve the neck and face, are not accompanied by adenopathies and mucous patches, and the Wassermann reaction is absent. They do not itch, never involute, and from time to time new lesions develop.

(2) Parapsoriasis lichenoïde (Fig. 22); (Lichen variegatus, Crocker; Parakeratosis variegata, Unna, Pollitzer and Santi; and perhaps Jadassohn's and Juliusberg's diseases as above). This variety is seen in the third and fourth generations and the dermatosis involves all parts of the body below the clavicles, usually not extending far beyond the knees and elbows. The eruption consists of yellowish scaling papules of a lichenoid character which coalesce and form a generalized lace-work. Amidst these small, glistening lesions, resembling those of lichen planus, are seen. There is no itching or involution.

(3) Parapsoriasis en plaques (Fig. 23); (Xantho-erythroderma perstans, Crocker; Erythrodermie pityriasique en plaques disseminées, Brocq). The body becomes covered with scaling, moderately infiltrated plaques, varying in shade from fawn color to reddish brown or yellow. The patches are irregular in outline, sharply circumscribed, vary in number, persist, and do not itch. The skin does not feel infiltrated and the lesions never involute.

Course. The disease begins insidiously in adult life or adolescence, increases slowly, and never disappears. The general health is not impaired.

Varieties. These have already been mentioned.

Differential Diagnosis. Psoriasis, lichen planus, scaling seborrhoea, syphilis, and mycosis fungoides are simulated by parapsoriasis. In psoriasis, the typical localization, involvement of the face and scalp, periods of remission, and the silvery scales are distinctive. Lichen planus (Chapter XIII) itches intensely, the lesions are umbilicated, violet or yellow, and glistening, and the buccal mucosa is frequently involved. Seborrhoea is seen on the scalp, in the folds of the body and flexor surfaces, possesses dense greasy scales, usually itches, causes loss of hair, and is prone to give rise to vesicular dermatitis. Syphilis may be ruled out along the lines mentioned in describing guttate parapsoriasis. Mycosis fungoides in the so-called premycotic stage simulates the plaque formed variety. Mycosis itches, and has a significant if not constant microscopic picture. Usually, only time will serve to make the diagnosis, and the likelihood of mycosis must always be seriously considered in cases of parapsoriasis *en plaques*.

Etiology and Pathogenesis. The cause of the disease is unknown.
Treatment. No treatment influences the disease.
Prognosis. This is good as to life, but the malady cannot be cured.

PITYRIASIS ROSEA (FIG. 24)

Synonyms. Herpes Tonsurans Maculosus, Pityriasis Circinata; French, Pityriasis Rosée (Gibert).

Definition. Pityriasis rosea is a common macular, and at times maculo-vesicular, scaling cutaneous disease, which, but for lack of evidence of infection would be classed among the acute exanthemata, but which, because of its clinical features, must still be grouped in with the psoriasiform dermatoses.

Symptoms. Although seen at all ages, the disease usually attacks young adults. It is rarely preceded by chills, chilliness and low fever, with slight glandular enlargements. At first a single lesion appears on the body which grows larger, and after from a few days to several weeks, other spots appear on the trunk (Fig. 24), arms, thighs, and rarely on the legs, forearms and neck. Facial involvement is exceptional. The first lesion, known as the primitive patch, is pink, and spreads peripherally with a pink advancing margin upon which are situated fine scales. Central healing takes place leaving a fawn colored puckered area which gradually becomes normal. The other patches vary in size up to that of a dime or a quarter, are circular or oval, and tend to arrange themselves, particularly on the back, in sloping lines parallel with the ribs. Elsewhere they also follow the lines of cleavage. They, too, heal centrally and spread

peripherally as does the main lesion which they resemble in all respects but size. Itching is usually present and often intense. In from three to ten weeks involution is complete. *Udo Wile* has described vesicles in the centre of some lesions.

Course. The disease is self limited. It usually is over in six weeks, but may last longer if not properly treated. It begins with a single patch and more lesions appear after a few days, or a fortnight. These increase in number during a second fortnight and then involution gradually takes place.

Varieties. Save the vesicular form, no other varieties are known.

Differential Diagnosis. Seborrhoea is the one disease closely simulated. At times, the most expert fail to make the diagnosis. Seborrhoea tends to be localized to the scalp, face, sternum, interscapular regions and body folds, but may be elsewhere. The scalp and face are practically always free in pityriasis rosea. Seborrhoea is more infiltrated and usually lacks the fawn colored centre, the entire patch being yellowish or brown, and the scales coarse, dense and greasy rather than fine. Ringworm and epidermophytia inguinale (Chapter XXII) may somewhat resemble pityriasis rosea, but a demonstration of the pathogenic organisms of these maladies clears up the diagnosis.

Etiology and Pathogenesis. A few facts concerning the disease are clear. It occurs prevailingly in spring and fall in an epidemic manner. Thus, it is clearly infectious. The fact that in a few instances fever and constitutional symptoms are present indicates the possibility of internal infection. On the other hand, the primitive patch preceding the generalized eruption points to the probability of an external agent. This patch might possibly be the source of a toxaemia, accounting for the early systemic symptoms, but such a view is unlikely as the disease is too mild. Micro-organisms of one kind or another have been described, but the work is unconvincing. Some authors regard it as a skin reaction to a general metabolic disturbance, and ally the condition with erythema multiforme. This is probably wrong because it looks different, acts differently and is localized differently from the latter. There are also those who consider the disease as evidence of a dyscrasia or of scrofula. These facts are mentioned only to be dismissed.

Treatment. Purely external therapy suffices. I have yet to see a case which failed to disappear within three weeks when the following lotion was used:

Resorcini	6.0
Zinc oxid	20.0
Liquor calcis	50.0
Alcohol 95% qs ad	200.0

Sig. Shake well and apply twice daily.

After about a week the skin may become dry and brittle. Then, an emolient salve (U.S.P. zinc ointment) should be employed, and after the dryness of the skin is better, the lotion is to be resumed. Irritating salves, such as the tars, salicylic acid, resorcin ointments, et cetera, are contra-indicated. They usually intensify the pruritus and protract the disease. Should the itching be severe, bromides are to be used, and locally, mentholated (two percent.) zinc stearate powder.

Prognosis. This is always good.

SEBORRHOEA

Definition. To define seborrhoea taxes ingenuity to its utmost. Opinions still vary greatly as to its nature and place among dermatoses. It is considered by some primarily an affection of the sebaceous glands, by others a dermatitis, by others non-inflammatory. There is no doubt that it favors sites at which the pilosebaceous organs are most numerous or most active, and it would be idle to deny the etiological significance of this circumstance. The question is further complicated by the fact that seborrhoeal soil readily gives rise to a dermatitis which was named seborrhoeal eczema by *Unna,* a term whose application has grown loose with time and which has been made to include all stages of the disease. Nor have those who, endeavoring to simplify the tangle by coining the expression seborrhoeal dermatitis, accomplished aught but confusion. *Sabouraud* takes the stand that seborrhoea, pure and simple, is non-inflammatory. This is wrong, as a most cursory inspection of the tissue shows.

Seborrhoea is a mild, superficial inflammation at any time capable of becoming severer and developing into one form of dermatitis (eczema). In its quiescent phase it may exhibit the inflammatory signs of scaling, the functionally overactive signs of steatorrhoea, or both. In its more active phase, all of the signs of skin catarrh, but chiefly weeping and crusting, may be added to the above, even to the extent of so completely masking the latter as to preclude a correct diagnosis until the secondary signs have vanished. It is probable, too, that the disease may be, at least in part, parasitic.

By piecing all of these facts together into a tentative patch work a crude conception of the process may be reached. Seborrhoea is a disease whose starting place at least is in areas rich in pilosebaceous organs. It is possible that parasites participate in its causation, and it is characterized by scaling, or over-activity of the skin glands, or both, and is prone to be the starting point of an exudative inflammation by no means limited to the sites originally affected.

Symptoms. Seborrhoea usually begins on the scalp (Fig. 25) with scaling and itching. The scales are white or gray, fine or coarse, and often greasy and adherent if there is marked over-activity of the sebaceous glands. The drier form, also known as seborrhoea furfuracea, pityriasis simplex, or eczema seborroeicum, is the common dandruff, the symptoms of which are known to everyone. At times the disease spreads to the neighboring skin over the forehead (Fig. 25), neck, behind the ears, *et cetera,* as a yellow to red, scaling inflammation which when irritated becomes vesiculo-papular and pustular, and weeps. This is the form which may truly be called seborrhoeic dermatitis, and which clinically and anatomically resembles any other dermatitis, the seborrhoeal soil forming the precipitating cause. (See Dermatitis, Chapter IX.)

Other areas of the body subject to the malady are the front of the chest, the interscapular (Fig. 26) region, body folds, eyelids (Fig. 25), eyebrows, umbilicus and genitalia. Scaling and itching are common to all, and the involved skin is often only slightly reddened, but not rarely deeply inflamed. Underneath the scales there are sometimes plugs which are hyperkeratotic casts of the patulous follicles below, and at times there are crusts of pure, dried grease when the flow of sebum is excessive. Such crusts are waxy and yellowish. When true dermatitis supervenes the crusts and scales assume the aspect of those which characterize the latter disease. Often in long-standing processes lichenification occurs.

Course. The malady usually begins at adolescence, often in childhood, and not rarely in infancy. One form, milk crusts, characteristic of babyhood, starts shortly after birth with a yellowish crusting of the scalp probably representing a continuation of the vernix forming propensity, and it usually vanishes within three or four months. The other forms, if not properly treated, persist for life. Seborrhoea of the scalp often causes baldness. If allowed to progress unchecked, seborrhoea of the face leads to slight thickening of the skin, and the pilosebaceous follicles become permanently

widened so that the integument looks coarse grained. These enlarged glands are constantly filled with exfoliated cuticle, sebum and dust, the entire mass forming comedones which will be further described in connection with acne. In addition to these the skin is often greasy. Acne vulgaris is frequently associated with seborrhoea, and the latter is always found with the former. Rosacea and some forms of simple dermatitis find their origin in seborrhoea.

Varieties. Without too great elaboration of this phase of the disease certain varieties must be mentioned.

Seborrhoea sicca is the commonest variety. The best example of this is dandruff.

Seborrhoea oleosa is an oiliness of the skin, usually of the scalp and face, often over the sternum and between the shoulder blades. The dry and oily form may be associated, whereupon the scales become greasy. On the face, under the scales and crusts, are found inflamed patches. A similar process, not unlike psoriasis, is seen about the navel and on the rest of the body.

Seborrhoeal dermatitis occurs at the sites already described in which ordinary seborrhoea is found. It may also remain restricted to the scalp, or spread to any of the areas mentioned. It is characterized by scaly macules, circular, oval or festooned in outline, or papules covered by a scale, or plaques, or a combination of the three, or it may be perifollicular, and it may weep, as ordinary dermatitis does, or there may be a free flow of oil so that the scales and crusts which are formed may either be greasy, or of the usual type seen in catarrh of the skin. Needless to say all of these forms have their special fanciful designations which it is useless for the practical student of dermatology to know. The scales near the skin are adherent, the upper layers being less so. All hairy portions of the body seem peculiarly vulnerable, but alopecia rarely occurs excepting on the eyebrows or scalp. When the body is involved, all sorts of diseases may be simulated, especially, however, psoriasis, pityriasis rosea, and ringworm. When the disease becomes universal it mimics the erythrodermas.

Seborrhoeal baldness or alopecia has been alluded to. It may be partial or complete. The hair first falls out about the frontoparietal regions and vertex, growing thinner and thinner. When the scalp is entirely bare it becomes glossy, pink and tense and the follicles are widened, but because they are made shallow by the traction on the scalp, comedones usually do not form. Lanugo hair

usually remains. As a rule a band of true hair survives reaching from temple to temple and from the nape of the neck to the occipito-parietal junction.

Diagnosis. Simple dandruff is easy to recognize. In children, seborrhoea must be differentiated from ringworm. This is usually easy since ringworm produces baldness either in large or small patches, and the areas involved are covered with gray, dry scales, or vesicles, or broken hair, are bald and finally are caused by fungi which may be demonstrated microscopically. There is also a diffuse form of tinea which closely simulates seborrhoea, and which can only be diagnosed by finding the fungi. Finally, the two diseases may co-exist. In adults, the diagnosis rests upon the presence of other symptoms of the disease, notably itching which is relieved by shampooing. It must be remembered that the normal skin desquamates; we wash or rub off the scales constantly so that we do do not know we are scaling. On hairy parts of the body the scales cannot fall off so readily. Thus, we must rule out physiologic before we venture the diagnosis of pathologic scaling.

Psoriasis favors the extensor surfaces, the scalp, and finally the rest of the body. It is a drier process, the scales are characteristic, and there are punctate hemorrhages on removing the scales, *et cetera.* (See this chapter above.) Only psoriasis of the scalp causes serious difficulty in differentiation, which is impossible without other evidences of either disease. Psoriasis, however, does not cause alopecia, and on the whole itches less than seborrhoea. Pityriasis rosea is characterized by an advancing, pink, scaling margin and a buff centre. There is a primitive patch larger than the rest, and the process rarely occurs above the clavicles. Ringworm is sparser and a demonstration of the fungi clears up the diagnosis. The diffuse erythrodermas may start as an apparent seborrhoea. The differential points will be taken up in considering these diseases (Chapter XXII).

Etiology and Pathogenesis. Simple seborrhoea is found in nearly all whites of both sexes from puberty up. Ethiopians have normally an oilier skin than whites, and among the Malay races the disease seems to be somewhat less common than among Caucasians. The commonness of the disease argues that it is either infectious, or inherently characteristic in the human race. *Sabouraud, Unna* and *Melassez* have described bacteria which they consider pathogenic. These are the famous morococcus, bottle and acne bacillus. Without entering too greatly into the details of the question it may be stated

that their evidence is largely circumstantial, but it is difficult to prove the pathogenicity of any cutaneous bacteria, and there is little to distinguish *Unna's* morococcus from *Welsh's* white staphylococcus which is always found all over the skin. The acne and the bottle bacilli present in seborrhoeal lesions are indistinguishable from similar organisms found elsewhere. Nor is there the remotest evidence that any of the alleged organisms is specific. All that can be said is that they are found in seborrhoea, and it may be stated with equal force that they are also found where seborrhoea is not.

Certain other factors are important. Seborrhoea begins or gets worse at or about puberty. It favors areas where the sebaceous glands are largest, most numerous and active. It is at puberty, too, that these glands increase in size and activity. Whether this is due to endocrinous changes peculiar to the period in question or not, it is at present impossible to state. If so, the disease is primarily due to internal secretory changes. Should a specific organism ever be proved to be the causative agent, it would be so only in a secondary sense, a precipitating cause, the changes of puberty in the broadest meaning, being the predisposing one. The possible connection of disturbed sugar metabolism, which is after all probably due to an endocrinous disturbance, has been indicated in the work by *Schwartz* and myself; we found a large number of instances of hyperglycemia in seborrhoeic people. In close relation with this problem lies that of alimentary hyperglycemia, excessive ingestion of carbohydrates, and perhaps carbohydrate fermentation and indigestion. These matters are all relatively vague, and only the crudest clinical corroboration of their possible etiological bearing exists; but there is no doubt that in many cases the proper regulation of diet, restriction of carbohydrates, and curing of gastro-intestinal disturbances improves and often actually cures seborrhoea.

General causes of the usual variety, sedentary habits, constipation and the like have been mentioned, as they always are where opinions on etiology have no sounder foundation. Probably, in given instances these may aggravate or predispose to the malady, but definite proof is lacking. The most striking fact after all is the relation of the process to puberty.

Treatment. Internal treatment consists in the main in an attempt to regulate the disturbed functions enumerated above. Of these, only the digestive group is amenable to therapy and here the matter resolves itself into the proper handling of hyperacidity, achylia and carbohydrate indigestion by diet and the use of a diastase,

or pancreatic extract and mild alkalies. Constipation must be cured by diet, and often mineral oil is of great value. In more complex digestive disturbances, the co-operation of a skilled internist or gastro-enterologist should be sought.

Local Treatment. The seborrhoeal scalp is differently treated in its varying aspects. Itching, scaling, evidences of acute inflammation and falling of the hair constitute the clinical indications. At times there is a marked loss of hair without much scaling and itching, or a great deal of scaling without much loss of hair. In general, the patient should be directed to shampoo once a week with some simple soap, but never green soap. Twenty-four hours before shampooing one of the following salves is to be applied sparingly to the scalp; either a five to ten percent. sulphur, a two to five percent. resorcin, a two percent. salicylic acid, or a five to ten percent. ammoniated mercury, in a base of cold cream, or equal parts of cold cream and lanolin. Vaselin is contra-indicated because it is hard to wash it out. Salicylic acid and ammoniated mercury together are very good. The salve loosens the scales and the shampoo removes them. Fine combing is bad. Every day, save the one on which the ointment is employed, a scalp lotion may be used. Beta naphthol, salicylic acid, euresol, resorcin, and spirits of formic acid combined with one of these, represent those usually employed. A little bichloride of mercury in a strength of one to five thousand may be included. It has seemed to me advisable to eliminate cantharides and quinine, for in my experience I have found that the former sometimes provokes dermatitis, and the latter, urticaria in the susceptible. Their value does not compensate for these unpleasant possibilities. A very excellent lotion which may be regarded as a type upon which to model others is

℞ Euresol
Spirit. acid. formicarum aa..... 10.0
Glycerin 15.0
Alcohol 95% qs ad 200.0
Sig. Apply to scalp once a day.

Salicylic acid 4.0 (two percent.) may be added to this, and if the patient desires the fluid to be perfumed, fluid extract of violets (0.6) or spirits of cologne (15.0) may be incorporated. The best method of applying a fluid of this sort is with a medicine dropper, the patient having been instructed to part the hair in parallel furrows separated by an inch, and to drop the fluid in the part. Thus the

liquid will spread evenly over the entire scalp. The procedure requires only about ten minutes, and after the scalp is entirely covered, it should be rubbed lightly for a moment or so, but not massaged.

Such treatment stops the itching and scaling, and usually the hair ceases to fall out within a month. New hair, however, does not grow readily after thirty. To promote the growth of hair many spurious methods of treatment have been devised, but the ranks of the bald-headed still remain unthinned. Massage, vibration with electric apparatus, and the like, all benefiting the treasury of the "hair specialist" more than the scalp of the patient, have been urged upon an eager public, and wave succeeds wave of credulous victims who despondently return to legitimate physicians after having been exploited by those who, without knowing medicine, make promises which it is not within the power of man to redeem. Recently the Kromayer and Alpine lamps have been widely employed in treating baldness, and although I can attest to their frequent value, I approach with utmost skepticism the claim that they can clothe the desert with verdure, or make the atrophied hair follicle give forth hair. Perhaps no better proof of the limitations of treatment is necessary than an inventory of the bald-headed dermatologists in existence.

Upon the non-hairy parts of the body sulphur, resorcin,[1] ammoniated mercury salves, and resorcin, euresol and sulphur lotions are of use in five to ten percent. strengths. Deeply scaling lesions should be treated precisely as psoriasis. X-Ray therapy is excellent, and in infiltrated patches the Kromayer lamp is useful also. The Roentgen rays are particularly valuable in oily seborrhoea. They atrophy the glands and thus lessen their over-activity and diminish the hyperkeratosis. This tends to overcome desquamation.

When vesicular dermatitis is grafted upon seborrhoea, the treatment must be first directed to the secondary process. It is conducted along the lines indicated in the chapter on simple dermatitis. When the superimposed condition is well, the seborrhoea should be treated as outlined above.

Prognosis. The individual attacks and the lesions of seborrhoea are easily cured, but recurrences are likely and numerous. In men, particularly, loss of hair is difficult to stop. At this point I should like to mention a view which I have held for years and which *Pollitzer* once expressed at a meeting of the American Dermatolog-

[1] In red-headed and blonde people resorcin should be used circumspectly since it sometimes turns the hair green. In white-haired people this usually happens.

ical Association, namely, that not all baldness is pathological, but that it is normal in a large percentage of men, at least. It is biological in that the hair is a secondary sexual characteristic, useless after a certain age. To the student of Darwin this point will be clear; to one versed in modern medicine it will be apparent that this is equivalent to asserting in another form that the disease is due to a change in the internal secretions.

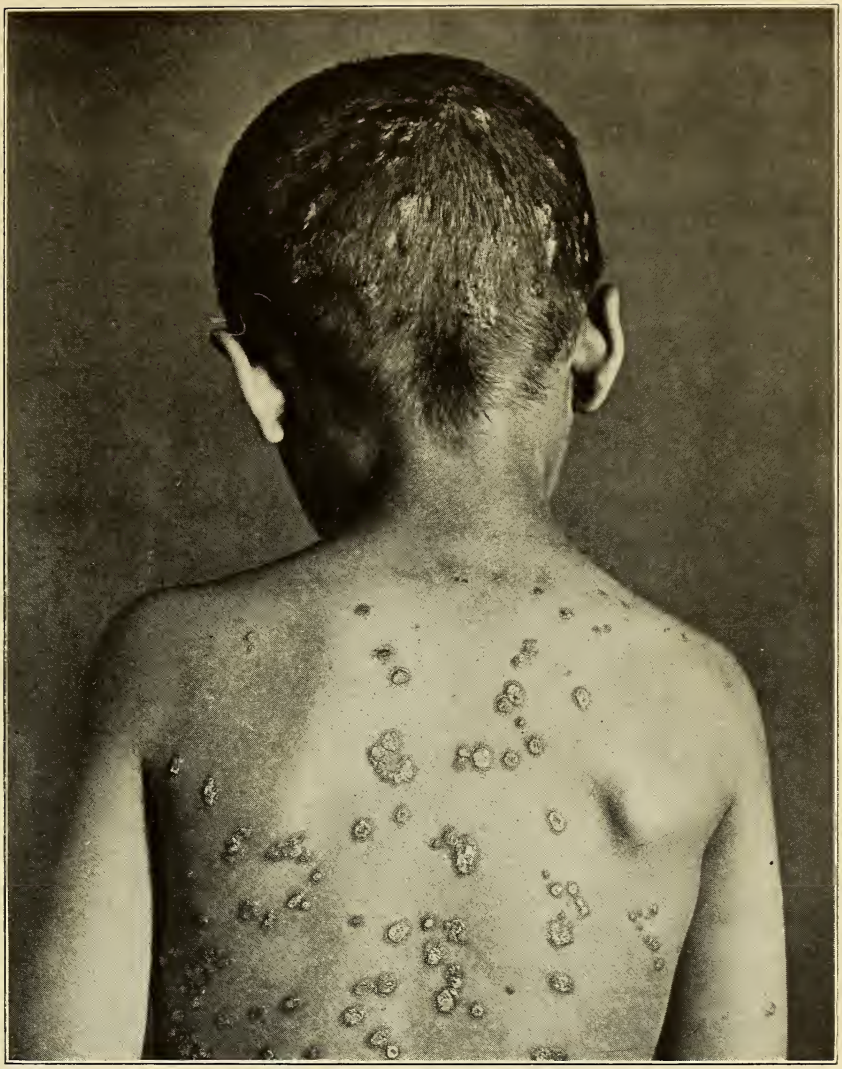

FIG. 20. PSORIASIS

Scalp involvement and the small, or guttate type of lesion are illustrated. The white, delicate mica-like character of the scales is shown. Near the left scapula, a patch is forming through coalescence. The scalp involvement alone excludes pityriasis rosea and parapsoriasis; the nature of the scales, seborrhoea.

FIG. 21. PSORIASIS IN PATCHES

In this form, as here shown, large, thickened, red plaques form. White scales develop. The margins are festooned by coalescence with smaller outlying patches. Here and there, small isolated papules are seen.

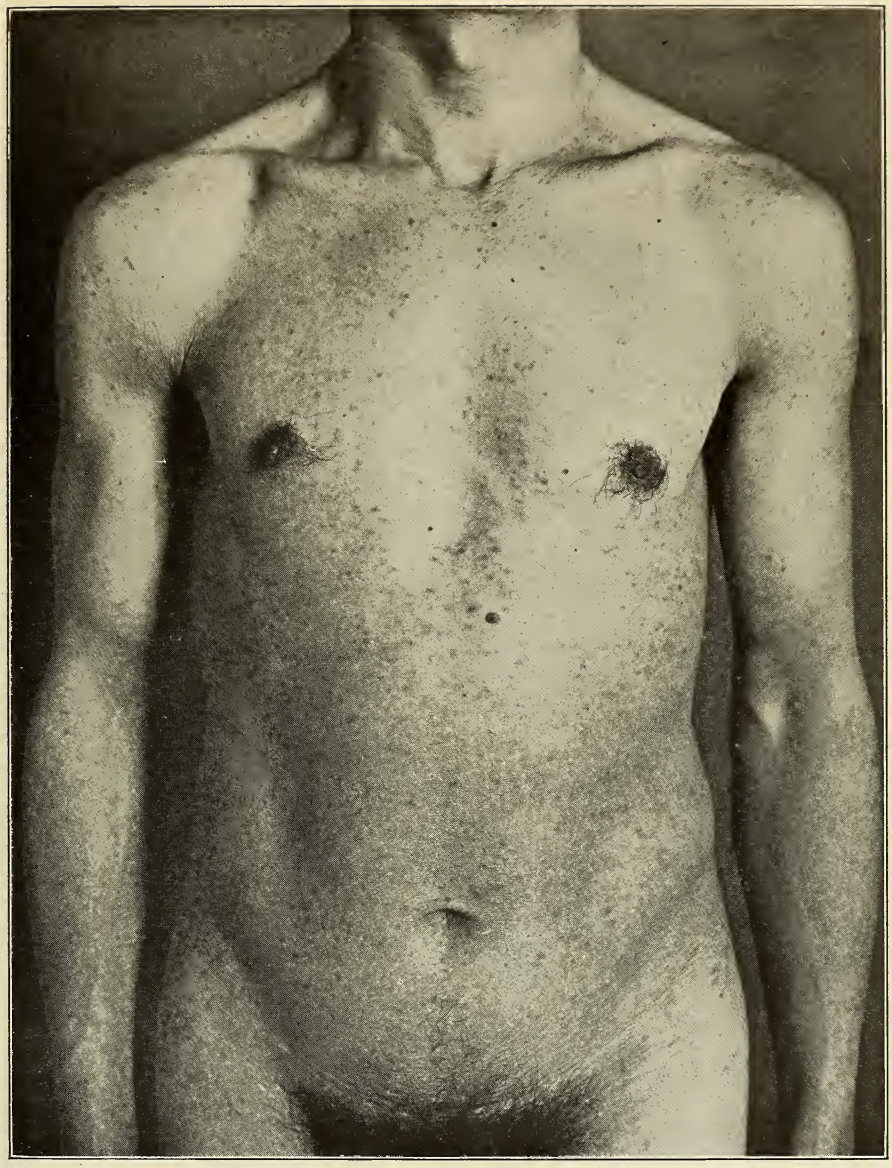

FIG. 22. PARAPSORIASIS

This variety of parapsoriasis is characterized by small buff papules some of which suggest lichen, others psoriasis. Both have small fine scales. The lesions rarely appear far above the clavicles or much below the lower third of the thigh. There are no subjective symptoms, and great chronicity distinguishes the affliction.

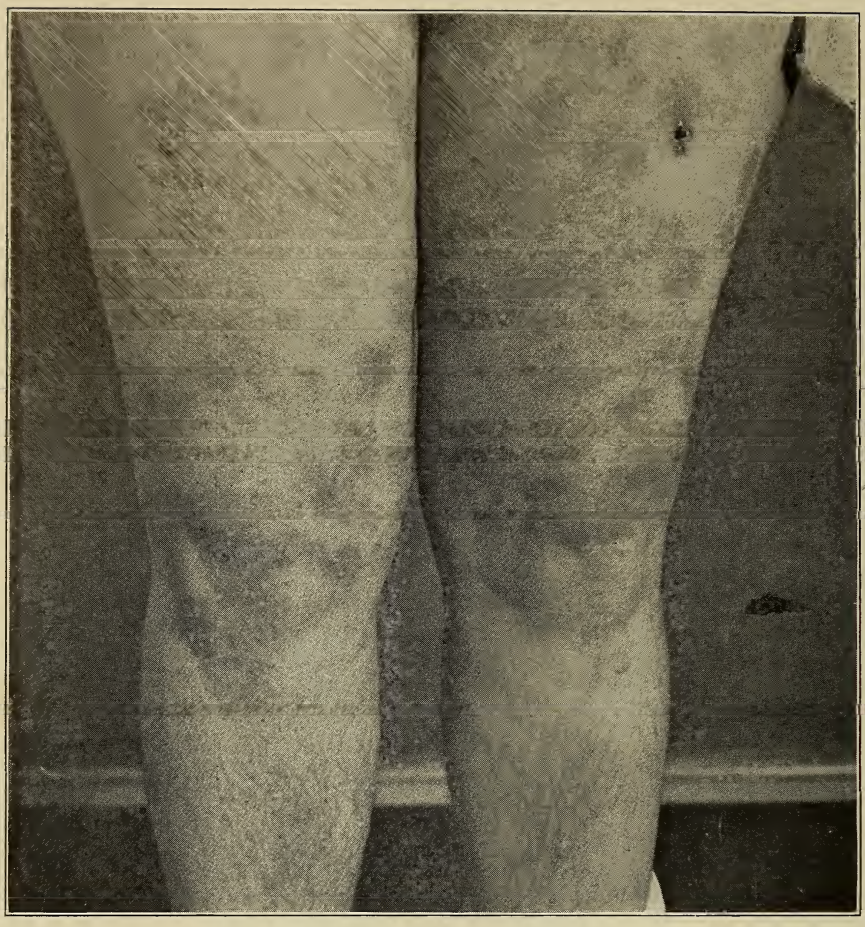

FIG. 23. PARAPSORIASIS

This is the plaque form of parapsoriasis, or erythroderma pityriasique en plaques disseminées. The lesion is extensive, yellowish, slightly infiltrated and scaling. There is no itching. This condition at times precedes mycosis. The face is usually free in parapsoriasis. The black spot on the left thigh is the result of a biopsy and has nothing to do with the disease.

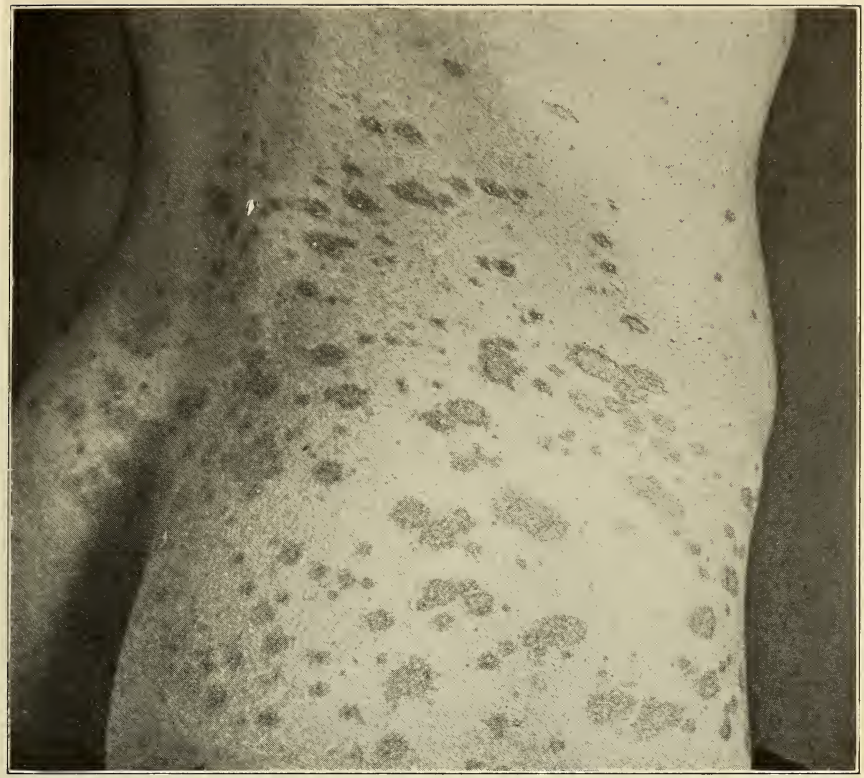

FIG. 24. PITYRIASIS ROSEA

This condition is characterized by oval or rounded flat papules interpersed among which are minute lesions. The long axis of the oval lesions parallels the skin's cleavage lines. A red, swollen margin encloses the yellow, or buff, slightly puckered, scaling centre. Usually the general outbreak is preceded by one large patch. The face, legs and forearms are rarely involved. Thus seborrhoea is ruled out. The scales are finer and not so white as in psoriasis, or so greasy and yellow as in seborrhoea.

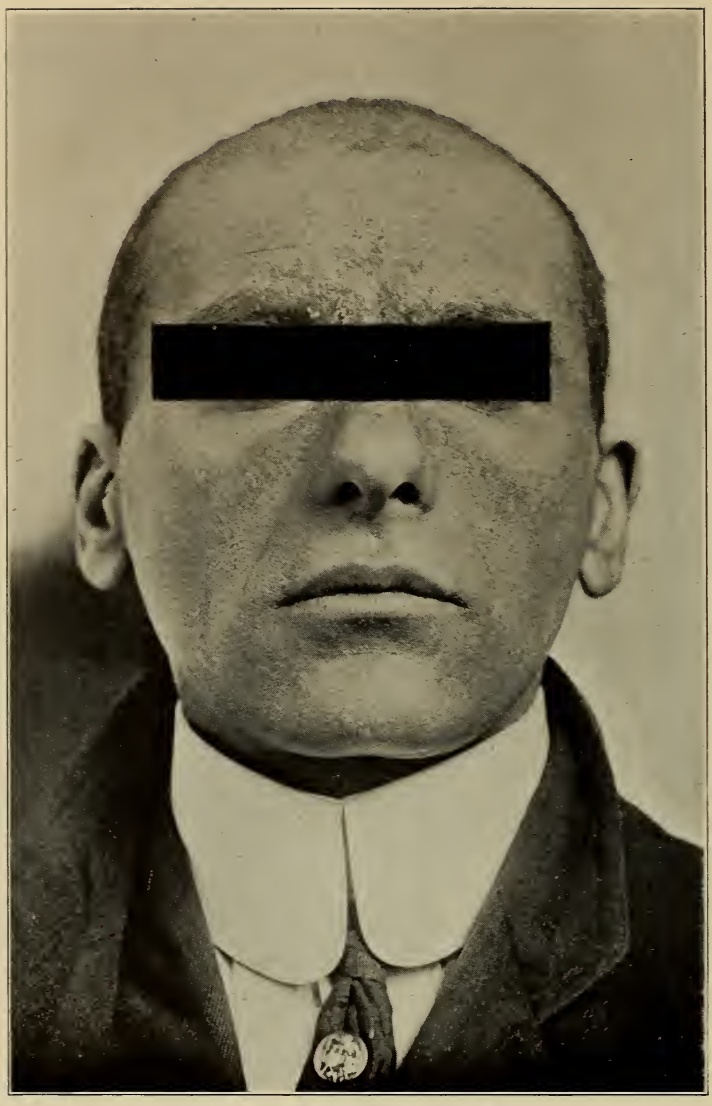

FIG. 25. SEBORRHOEA, SCALP

Seborrhoea includes a group of conditions (Fig. 27) featured by inflammatory lesions. These are small or large, flat papules or patches which are yellow or brownish, and covered by greasy white, yellow, or grey scales. They favor the flexor surfaces, chest interscapular regions, back, face and scalp. This picture represents the last form. The lesions individually resemble psoriasis, save for the greasiness of the scales, but the disease differs from psoriasis as to location, the ease with which it leads to vesicular dermatitis, and the frequency with which it leads to baldness.

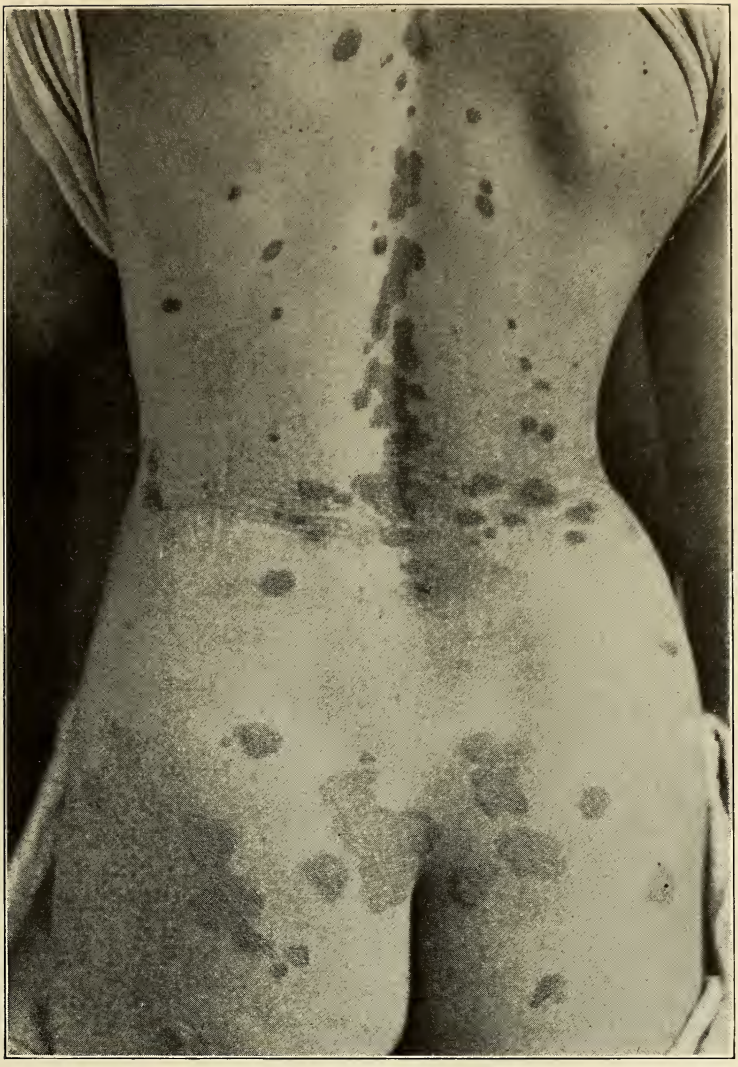

FIG. 26. SEBORRHOEA, BODY

This picture is typical. The patches are small or large, and vary greatly in outline. The scales are not conspicuous in this patient, but the localization is typical. (See note, Fig. 25.)

CHAPTER XII

THE ERYTHRODERMAS OR EXFOLIATIVE DERMATITIS

Here again, as in all instances in which it is endeavored to group dermatoses, many factors enter into conflict. Regarded from one standpoint, the erythrodermas, which are nothing but diffuse, inflammatory, red, desquamating diseases of the skin, may be divided into two groups, primary and secondary. The primary, which belong together only because of the circumstance that they arise from no forerunning cutaneous condition, are dermatitis exfoliativa of Wilson-Brocq, pityriasis rubra of Hebra, dermatitis exfoliativa neonatorum of Ritter, erythroderma desquamativa neonatorum of Leiner, erythroderma ichthyosiforme congenita of Brocq, and dermatitis exfoliativa epidemica of Savill. The secondary varieties are those which arise from any disseminated maculopapular disease which under certain conditions becomes universal, and which when universal loses its identity so that its true nature cannot be recognized. It remains unrecognizable until the process reassumes its original form. Psoriasis, scaling dermatitis, lichen planus and lichen acuminatus are capable of this.

Regarded from another viewpoint, the conditions may be classified as characteristic of the newborn and of the adult. The former comprise Ritter's and Leiner's diseases and Brocq's congenital ichthyosiform erythroderma. All the others belong to the adult group.

Etiologically, so far as anything is known at all, all of the conditions are individual, and only the infantile forms have a common bond. This tie is a loose one and consists simply of the fact that the infant human being is attacked. There is no doubt whatever that these conditions again are examples of cutaneous reactions, for widely varied causes may excite similar scaling processes, and it is equally likely that the same cause may provoke desquamative processes differing in appearance. With these facts clearly in mind and fully appreciating that the essentially unrelated entities are assembled in one chapter because of accidents of objective similarity and ignorance of their ultimate causation, we may proceed to a study of their clinical features.

DERMATITIS EXFOLIATIVA

(Wilson-Brocq)

This is a very rare condition. It starts as bright red erythematous patches covered with coarse scales resembling pie crust. Rapidly the entire body is involved, no area being spared. The hair grows brittle and lustreless and may fall. The nails become thick and dull and may also fall out. Usually the process is dry, but it may weep. Occasionally mild systemic symptoms inaugurate the disease. When in full bloom the malady has a distinctive appearance. The entire body is bright red and covered with coarse, large scales which peel back at their edges. There are often slightly enlarged glands, but the general health suffers only slightly. Periods of remission, alternating with recrudescences characterize the process and usually, even after lasting for many decades, the patient recovers, although a few deaths have occurred. Quinine, by mouth or even intravenously administered is of great value, and such drugs as pilocarpine, aspirin and the like have been used with success. Locally, simply salves and oils are to be employed. The etiology of the disease is totally unknown.

PITYRIASIS RUBRA OF HEBRA

Synonyms. Dermatitis Exfoliativa; Lymphadenitic Erythroderma.

Definition. Pityriasis rubra [1] is a rare, chronic, diffuse disease leading to atrophy of the skin after months or years of illness. It ends in death.

Symptoms. Usually the onset is gradual and takes place as desquamating erythema in the knee and elbow flexures. From these sites it extends until the entire body is covered by red, uninfiltrated, thin integument from which fall an abundance of scales, usually fine, but sometimes coarse. Atrophy occurs and the skin stretches, becoming tense and thin so that over bony prominences and in body folds, it cracks or ulcerates. The lymphatic glands become huge; the hair and nails after growing dry and dull fall out, the patient emaciates, weakens, and atrophy of the facial skin causes ectropion of the eyelids. Thus, conjunctivitis develops. Moderate or severe pruritus is present, and there is regular fever, usually highest at night, with a rapid pulse, sweats, diarrhoea, and often chills, stupor and rigors. Albuminuria and icterus are also frequently encountered.

Course. The illness starts as described, involves the entire body in from a few weeks to two years, runs a chronic, progressive course with the complications enumerated above, and after a year or several

[1] This must not be confused with Pityriasis rubra pilaris which is synonymous with lichen acuminatus (Chapter XIII).

years ends fatally. Death is determined by one of the complications mentioned, or asthenia, pneumonia, bronchitis, albuminuria, pulmonary edema, or some intercurrent infection. Pulmonary or generalized tuberculosis are usually discovered at autopsy.

Differential Diagnosis. Dermatitis exfoliativa (Wilson-Brocq), premycotic erythroderma and the secondary erythrodermas must be differentiated from Hebra's pityriasis rubra. The great prostration in the last named disease, the glandular enlargements, fine scales, atrophic skin, progressive and unremitting course, and the consistent decline of the patient are characteristic. Wilson's form has periods of remission and usually ends in recovery; the scales are coarser, the patient is in better general health and the skin does not atrophy. In premycotic and preleucemic erythroderma, the diagnosis cannot usually be made, save in retrospect when the patient has mycosis or leucemia. This subject will be discussed in detail later on (Chapter XXVI). The secondary forms may be recognized by their milder course, remissions during which lesions typical of the primary disease appear somewhere on the body, and a greater degree of infiltration. Lipmann-Wolf in a discussion of this subject at the Berlin Dermatological Society (May 14th, 1912) said that the diagnosis of pityriasis rubra of Hebra could not be made during life, that death was the pathognomonic feature of the condition.

Etiology and Pathogenesis. Very little is known of the cause of the malady. *Lipmann-Wolf* on the occasion mentioned above, quoted *Saalfeld's* view that it was not a disease *sui generis,* but a cutaneous reaction, an attitude in which *Brocq* concurs. *Jadassohn* pointed out its relation to tuberculosis. A great deal of evidence to this effect exists and undoubtedly, in at least one form of the disease, this factor is causative. Hence, pityriasis rubra may be included in the tuberculides (Chapter XXV). In a typical case I saw in consultation and had the opportunity to study, the patient had carious teeth, pyorrhoea and apical abscesses. The usual prognosis was made, but after his teeth had been extracted he recovered completely and, at the present writing, precisely seven years after the grave prognosis had been made, the patient is perfectly well and attending to his affairs, following an illness which had grown progressively worse for two years.[1]

Treatment. This is purely symptomatic and should be con-

[1] In another instance the theory involved in the first case was applied without success.

ducted along the lines indicated in discussing Wilson's disease. Tuberculosis should be considered and, if present, it naturally furnishes the general therapeutic indications.

Prognosis. The prognosis is grave.

SAVILL'S DISEASE

This disease is also known as dermatitis exfoliativa epidemica. An epidemic skin disease occurred in London in 1891 where several hundred cases were reported. Two types were described; one corresponding to a universal, exfoliating and weeping dermatitis, the other to pityriasis rubra of Hebra. The onset was acute without prodromata, but accompanied by vomiting and diarrhoea, and in some instances by pharyngitis. The head and upper extremities were favored, although other sites were involved at the outset, and gradually the rest of the body became affected. In the young, the cervical glands were enlarged. *Savill* described a papulo-erythematous stage, lasting up to eight days. The papules were shotty, single or grouped in circles. Then an exudative stage appeared lasting up to eight weeks, in which there were scaling, vesiculation and diffuse erythema. Finally, a period of defervescence closely simulating pityriasis rubra of Hebra appeared. The cause has never been determined. The mortality reached as high as thirteen percent.

DERMATITIS EXFOLIATIVA NEONATORUM (Ritter)

This disease is also known as Dermatitis erysipelatosa. It was first described in 1878 by *Ritter von Rittershain.* It begins about the second week of infancy, starting as an erysipelatous rash about the mouth, whence it extends to the face, neck and entire body. Crusts, scales and scabs form, and the desquamating areas are uneven, red and greasy. The trunk is more involved than the extremities and the worst area is the scalp. The buccal mucosa is red and exfoliates. Fissures form about the lips and anus, and at first the infant gains, but in from ten days to several months about half of the patients die, the others recovering entirely. Death is caused by an intercurrent disease such as pneumonia, or chronic indigestion with marasmus.

ERYTHRODERMA DESQUAMATIVUM NEONATORUM (Leiner)

This disease was first described in Leiner in 1907. The ensuing is an abstract of the original description. At first the disease involves the scalp extending to the face and the rest of the body as an exfoliating, crusted, fatty group of lesions which are most characteristic on the forehead, or as slightly scaling, circular lesions at the same site. The scales are thickest on the brows, thinnest on the cheeks; the eyelids are red, their margin thick, the lashes sparse, and excoriations appear on the involved integument. The body is red, scaling, and the skin is thickened, dry, shiny and even velvety. In places it atrophies or becomes ragged. Some-

times the scales are imbricated. Desquamation appears on the hands and feet, the nails are disturbed in their growth and sometimes fall out, and a greasy exudation arises in the body folds. The mucosa remains normal.

Leiner emphasizes the fact that dyspepsia accompanies the picture, and the disease occurs almost exclusively in breast fed babies. Its etiology is little known; some regard it as pemphigus; others as an infantile form of pityriasis rubra of Hebra; some think it due to breast milk, others to an infection. *Brandweiner* regards it as baby seborrhoea. Death is due to an intercurrent disease or marasmus, and most of those affected die.

ERYTHRODERMA ICHTHYOSIFORME CONGENITUM
(Brocq)

This is a rare scaling erythematous disease presenting some features of exfoliative dermatitis and some of ichthyosis. It is not yet conceded to be a definite entity.

Besides these conditions must be mentioned a disease vaguely related to the dermatoses included in this chapter. It is a generalized foliaceous erythema known as erythema scarlatiniforme desquamativum recidevans of Feréol and Besnier. It is also called scarlatinoid erythema, scarlatinoïde, roseola scarlatiniforme, and dermatite exfoliante aïgue benigne by Brocq. It begins as a generalized erythematous exfoliation. Its onset is sudden, accompanied by headaches, chilliness, fever (100°–102°) and the rash involves the body folds, extremities, and gradually the whole body, sometimes including the head. It resembles scarlet, measles and German measles, and after the erythematous stage fine scaling sets in. There is slight or no sore throat, no Flindt's spots, no glandular enlargement, and recurrences are common. These facts connected with its uniform benignity differentiate it from the commoner acute exanthemata.

CHAPTER XIII

THE LICHENS AND LICHENIFICATION

This group of diseases is a comprehensive one, and includes many conditions the causal identity or even close relationship of which is not established. The confusion is historic and originally arose from a diffuse and unsound application of the purely figurative word lichen. Botanically, a lichen is a dry, mossy, scaling growth found on stones, stumps of wood and the like, and since many skin diseases roughly resemble this plant, the purely fanciful designation was adopted in medicine. Many diseases, not called lichen, show this resemblance much more than others to which the term is applied. Psoriasis, scaling dermatitis, pityriasis versicolor all do so infinitely more than lichen planus. In the technical manner of employing the term sanctioned to-day, a lichen has acquired the significance of a rather raised infiltration composed of single or coalesced papules which scale, and lichenification is the name of the pathological process by means of which lichens are produced. Lichenification also has become synonymous with the word thickening, and lichenified with thickened.

It would be unprofitable, not to say confusing to those uninterested in dermatological dialectics to review the history of the name, and the literary and clinical evolution of the diseases in this group as we to-day conceive them. They all have common points of resemblance. They are thickened, impliable, scaling, more or less red, buff, or violaceous lesions which itch, and are composed of small papules single or grouped, follicular or non-follicular, which tend to coalesce into fairly circumscribed, scaling patches of rather distinct configuration. These patches are called patches of lichen, lichenification, or lichenified plaques. In the various diseases to be described the primary papules, however, have distinctive features, and the different conditions have peculiarities of site, appearance and course. When the ensuing matter has been thoughtfully studied, it will become clear that the complexities of the question are neither so numerous nor insuperable as they appear at first.

Older authors, as *Willan,* described five types of lichens: lichen

simplex, later called lichen simplex acutus, or prurigo mitis; lichen pilaris, now known as perifollicular seborrhoea; lichen lividus, a purpura; lichen tropicus, now called sudamina; and lichen urticatus, now called prurigo mitis, or mild papular urticaria. *Rayer* included lichen agrius, known now as prurigo ferox. These names are recorded because readers may encounter them elsewhere, and to emphasize the fact that they are not lichens in the modern sense. To-day we might well divide the group as follows:

I. True Lichen
   1. Lichen planus (Wilson).
   2. Lichen ruber acuminatus of Hebra, or pityriasis rubra pilaris of Deverige (included out of respect for tradition).

II. False Lichens.
   1. Follicular
      a. Lichen pilaris (Bazin), or keratosis pilaris simplex (Chapter XV).
      b. Folliculitis rubra (Wilson), probably Unna's ulerythema ophryogenes (Chapter XV).
      c. Lichen tropicus (prickly heat).
      d. Lichen spinulosus
   2. Toxic lichens, lichen agrius or prurigo, lichen urticatus or infantile chronic urticaria.
   3. Specific lichen
      a. Lichen syphiliticus (see syphilis, Chapter XLII).
      b. Lichen scrofulosorum (see tuberculides, Chapter XXV).
      c. Lichen nitidus (probably tubercular).

III. Lichenification.
   1. Primary-lichen simplex chronicus (Vidal) or neurodermite (Brocq).
   2. Secondary-lichenification (Brocq) lichenization (Besnier), found in itching dermatoses.

Of these, lichen planus, lichen acuminatus, lichenification and lichen chronicus simplex will be studied in this chapter, for they are the sole surviving lichens in which the term is more or less justifiable. Lichen nitidus will be here mentioned because its true place is not yet definitely known. Lichen spinulosus somewhat suggests lichen acuminatus, but is actually more like keratosis pilaris (Chapter XV). In a restricted sense there is but one true lichen, lichen planus, but this point need not be too narrowly emphasized. Thus, for practical purposes the clinical student may regard the lichens as:

1. Lichen planus (one form of the so-called lichen ruber)
2. Lichen acuminatus (another form of the so-called lichen ruber)
3. Lichenification
   a. Primary
   b. Secondary
4. Lichen nitidus

But one further condition must be mentioned before we begin our clinical survey, and that is lichen ruber. A classical battle once was waged about the relation of lichen planus to lichen acuminatus. It had been thought that the two latter were different phases of a single process, and that lichen ruber included both. The only thing to be remembered is that the term lichen ruber is obsolete, although it was for a time retained in the designation lichen ruber planus.

LICHEN PLANUS (FIG. 27)

Synonyms. Lichen ruber planus (obsolete).

Definition. Lichen planus is an infrequent, though not rare, itching papular eruption, involving the skin and buccal musoca, inclined to be chronic, often acute, and capable of assuming many forms. Its elementary lesions tend to become confluent forming plaques, which scale, and its color varies from waxy buff to purple.

Symptoms. As a rule the disease appears on the front of the wrists or above the ankles, as an eruption of pinpoint, waxy, flesh-colored or pink papules which itch. The papules are polygonal (Fig. 27) or angular, have steep sides, show a central tiny depression or umbilication, and are covered by a firmly attached small, white scale. Although many papules remain discrete throughout, an equal number may become confluent forming groups of lesions three to five millimeters broad which are elevated, red to violaceous or purple in color, and covered by thin, adherent, white scales. Larger patches or plaques are usually purple, this shade shining through a white or gray film partly produced by the desquamation. The sides of the plaques are steep, the surface is furrowed by minute intersecting lines, and the scales, as mentioned above, adhere firmly. The buccal mucosa (Fig. 28) is nearly always involved. On the sides of the mouth, tongue and lips are seen tiny snow-white papules corresponding to those on the skin, or leucoplakial sheets, corresponding to the cutaneous plaques like which they, too, are furrowed. Almost the entire body may be included in the process, but the neck, face and scalp rarely are, and the palms and soles never. The sites of election

are the front of the wrists, legs above the ankles, buccal mucosa, glans penis and the greater labia.

Itching is frequently intense. There is rarely vesicle formation except after the use of arsenic which is perhaps the cause of the so-called pemphigoid lichen. Lichen papules may arrange themselves in rings (Fig. 29), lines, plaques, or in some combination of these figures, and they may be large, small, or obtuse. As a rule, no general symptoms accompany the outbreak, but fever has at times been noted, and very rarely high fever and prostration. Ordinarily, but not always, the distribution is symmetrical; at times the lesions cover the entire trunk and extremeties, and not infrequently there appears an acute general outbreak of minute papules, which rapidly assume the appearance of those seen in the form that develops more slowly. It is essentially a disease of adult life, and most exceptional in children.

Involution takes place with hyperpigmentation, and sometimes depigmentation at the sites of the vanishing papules. The discoloration gradually fades, the normal hue of the skin finally returning.

Course. The onset usually is gradual, but sometimes acute. Months or years may be consumed in its course, but nearly all of the cases end in recovery. Some are inveterate, however, and the buccal lesions are very refractory, although in a great many instances even these disappear. Recurrences are exceptional.

Varieties. There are many varieties of lichen planus, but these are chiefly due to accidents of arrangement, distribution, and the like.

I. Common lichen; Lichen planus (Wilson) (Fig. 27)
II. Hypertrophic Forms
    1. Lichen planus hypertrophicus (Vidal and Leloir) (Fig. 30)
    2. Lichen planus verrucosus
    3. Lichen planus obtusus
III. Atrophic Forms
    1. Lichen planus sclerosus et atrophicus (Hallopeau)
    2. Lichen planus morphoeicus (Crocker)
    3. Lichen albus (von Zumbusch)

    These are identical.
IV. Lichen Pemphigoides
V. Forms varying as to distribution, arrangement, *et cetera*
    1. Lichen of the mucosa (Fig. 26)
    2. Lichen annularis

3. Lichen moniliformis
4. Lichen striatus or linearis
5. Lichen planus erythematosus

Lichen Planus Hypertrophicus (Fig. 30) usually occurs on the lower, sometimes the upper extremities, in irregular strips or oval patches, or irregular reddish-brown or purple plaques. Horny excrescences and scales develop. The itching is severe.

Lichen Planus Verrucosus is a further exaggeration of the preceding, in which the excrescences become warty.

In *Lichen Obtusus* the papules are larger than in ordinary lichen, and flat or convex in outline.

The three atrophic forms, *Lichen Sclerosus, Morphoeicus* and *Albus,* consist of white papules, or ordinary papules surrounding a clear white, atrophic center. The former are outlined by a violaceous or red areola. The white papules, even when coalescent, do not lose their individual shape entirely. Plugs arise from the depressions or umbilications. They often spring from the sweat pores. Usually the trunk is affected.

Lichen Pemphigoides is extremely rare. Bullae arise on the patches or plaques.

Lichen Annularis (Fig. 29) is formed by the papules grouping themselves so as to form a ring, or more rarely by extending peripherally while they heal within. This form is commonest on the glans penis.

Lichen Moniliformis is rare. It consists of papules arranged in the manner of beads in a necklace.

Lichen Striatus or *Linearis* consists of a linear arrangement of the papules.

Lichen Erythematosus simply indicates that the papules are extremely red.

Differential Diagnosis. In all cases the diagnosis rests upon the appearance of the lesions and their distribution. The papules are polygonal, steep and umbilicated, covered by an adherent scale; they are waxy buff or pink, reddish or violaceous. They favor the front of the wrists, the legs above the shoetops, the buccal mucosa and the glans, and itch more or less. In the rarer varieties concomitant typical lesions may be found indicating the true diagnosis. The diseases to be differentiated are psoriasis, parapsoriasis, vesicular dermatitis, lichen syphiliticus, lichen scrofulosorum, lichen nitidus and lichen simplex. Lichen sclerosus must be distinguished from that variety of scleroderma or morphoea (Fig. 40) known as white spot disease, and idiopathic macular atrophy, while buccal lichen must be differentiated from simple and luetic leucoplakia (Fig. 74). Psoriasis favors the extensor surfaces and scalp, which is never the case in lichen, while the latter is often, if not always, in the mouth, and the former never. Removal of the scales in psoriasis causes

punctate bleeding; the scales are silvery and imbricated. None of these features exist in lichen. Parapsoriasis does not itch, is not infiltrated, and never involves the mouth. Indeed only one variety of this disease, namely, pityriasis lichenoides chronica, or guttate parapsoriasis might cause confusion. But the scales are less adherent than in lichen planus, the lesions are lenticular rather than polygonal, and never are waxy or violaceous. Vesicular dermatitis and sudamina from a distance resemble fresh disseminated lichen, because of the glistening yellowish to pink color. On close inspection the lesions are found to be vesicles and not papules. Lichen syphiliticus, or the small, lenticular papular syphiloderm, closely resembles lichen planus in all respects. Lichen syphiliticus is one of the few specific eruptions that may itch. This is more common, however, in negroes, and furnishes a further point of confusion with lichen planus. The syphilitic variety, however, is usually found to be follicular, which lichen is not, and other features of syphilis are present — enlarged glands, mucous patches, and the Wessermann reaction. Lichen scrofulosorum is a rare disease, most often seen in children at an age earlier than lichen planus is likely to occur. The lesions themselves are similar in the two conditions, but those of the scrofulous variety are grouped in circular patches and rarely coalesce. The sites involved are primarily the sides of the trunk, buttocks, and upper part of the thighs, and not seldom evidences of general tuberculosis are present. Lichen nitidus is extremely rare. The mere fact that among other sites it is found upon the palms distinguishes it from lichen planus. Lichen simplex or neurodermatitis has favored sites over the vertebra prominens, in the elbow and knee flexures, and over the small of the back. It may also occur elsewhere and simulates only the plaques of lichen planus. The color of the two differs, that of lichen simplex being redder, and the scales in this disease larger; excoriations often are present, and the margins are not so sharp, often being studded with small papules at their periphery. Lichenification is differentiated from lichen planus by an appearance closely resembling that of lichen simplex, and by the further fact that it exists in combination with some other itching disease to which it is a sequel. Lichen planus must also be distinguished from lichen acuminatus. This will be discussed below.

Etiology and Pathogenesis. Although nothing is really known of the etiology of lichen planus inferential evidence exists that there are two groups of causative factors,— nervous and infectious. In addition to this a plethoric literature, purporting to prove the toxemic,

reflex and local infectious nature of the malady, precisely as in all other illnesses of unknown origin, need be mentioned only to be dismissed. The reasons for dwelling upon the possibility of its relation to the central nervous sytem are that it occurs prevailingly in the neurotic, or after mental, psychic or emotional shock. This is more than fanciful as significant instances occur in every dermatologist's experience. The reasons for seriously regarding infection as the possible cause may be summed up in the fact that arsenic and mercury cure it, and the time has long since passed when the efficacy of these medicaments could be explained on the ground that they are alteratives. Whether the pathogenic agent is a bacterium or protozoön no one knows. The response of the disease to the two metals mentioned speaks for the second assumption. *Hazen,* in discussing a paper by *Lieberthal,* announced that he had found a spirochaete, the pathogenic nature of which he hinted at. What reconciliation there may be between the nervous and infectious theories it is hard to understand, unless the former indicates that the physiology of the host is so altered as to make him susceptible to the specific undiscovered agent in question.

Treatment. Of all internal medicaments employed the only two of any value are arsenic and mercury. Arsenic is given in increasing doses, whether Fowler's solution, arsenic trioxide, the cacodylates, or Asiatic pills are preferred (Chapter XI. Treatment of psoriasis). Mercury may be administered by mouth or injection as in syphilis. In my experience mercury is the more efficacious drug, and the protiodide is its must useful salt. At first pills (Gr. $\frac{1}{8}$ plus, t. i. d. p. c.) are given, and the dose is increased daily until the lesions begin to fade, whereupon internal treatment is no longer necessary.

Two indications are to be met as to the local treatment,— the itching, and the need to cause involution of the lesions. Although tars, salicylic acid and resorcin have been employed, it has seemed to me that they do not hasten the disappearance of the papules in disseminated lichen, and furthermore, they often irritate the skin, provoking erythematous and vesicular dermatitis. Thus, I have learned that a mentholated powder (two percent. of menthol in zinc stearate), used two or three times a day, or mentholated calamine and zinc lotion similarly employed control the pruritus. In addition to this, divided doses of X-Rays limit both the pruritus and duration of the disease, reducing the course to a maximum of three, instead of from six to twelve, months.

In hypertrophic and verrucous forms salicylic acid plaster (ten to twenty percent.), and two to four H doses of X-Rays, and the use of the Kromayer lamp work best, coupled with mercury internally. In atrophic lichen the process may be stopped with the internal medication already mentioned and with X-Rays, but the atrophy is permanent. Buccal lichen, if not responsive to mercury and arsenic, is intractable. After the lichen papules are gone a hyperpigmentation persists for months. Nothing hastens its disappearance and it is a characteristic feature of the disease.

Prognosis. As regards life, the prognosis is nearly always good, although isolated instances of fatal termination are on record. Ordinary disseminated lichen may persist for a year or even more when neglected. Otherwise, its cure may be accomplished within a maximum of four months. The atrophic form may be limited by therapy but the atrophy persists. Buccal and hypertrophic forms are difficult to cure, but often this desirable result is attainable. The other forms being merely aberrances of the common variety, respond readily to the usual treatment. The disseminated variety rarely recurs; the localized forms sometimes do.

LICHEN ACUMINATUS (FIG. 31)

Synonyms. Lichen ruber (Herba);[1] Lichen ruber acuminatus (Kaposi);[1] Pityriasis rubra pilaris (Devergie).

Definition. Pityriasis rubra pilaris is a chronic folliculitis with hyperkeratosis, the clinical features of which are conical, red, scaling papules containing a horny plug. The lesions are situated about the hair and sweat follicles in a more or less definite distribution. At times the process becomes generalized and it often is preceded by scaling at the sites subsequently to exhibit the papules.

Symptoms. At the start, gradual as a rule, though sometimes sudden, the patient shows moderate scaling on a slightly thickened, rough and somewhat erythematous base. Then pink, buff, red, violet or even purple papules arise, which are conical and in the centre of which there is a horny plug only slightly elevated above the volcano shaped papules. The lesions are from one to three millimeters broad and high. The hands, feet, face, scalp, eyebrows and body surface are affected, the order of frequency being indicated by the sequence just mentioned. Palmar (Fig. 31) and plantar hyperkeratosis develops, and the buccal mucosa is very rarely involved, the lesions here resembling those of lichen planus. By far the great majority

[1] Obsolete.

of cases are not extensive, the lesions remaining localized to certain places, the site of election being the back of the fingers. On the whole the extensor are rather more favored than the flexor aspects. The papules may remain discrete, but are often packed together. Plaques may form, or exfoliating patches, and later in the disease the lymph glands enlarge and marasmus supervenes.

To the touch the skin feels rough. When the face is involved ectropion of the lower lids develops; the conjunctivae are inflamed, becoming red and everted. Thus lachrymation and crusting of the lids arise. The patient is sensitive to cold, and the moderate but constant itching and tension of the skin become cumulatively more annoying as the resistance and general health decrease, leading to wasting of the body. There is not, however, a progressive augmentation of the severity of the disease. Periods of complete remission are usually encountered with ensuing relapses, each of which is more prolonged and severer than the preceding one. Often, however, permanent recovery from the attacks has been noted. In the severer forms the aspect of exfoliative dermatitis is assumed, and in many of the milder ones that of chronic dermatitis with weeping and crusting. The nails and hair grow dull and brittle and sometimes fall.

Course. The disease starts suddenly or gradually, remits, or gets uniformly and progressively worse and more extensive, and after lasting for many years with the general health relatively unaffected, the patient dies of a different ailment, or rarely of pityriasis rubra pilaris itself when marasmus has developed.

Differential Diagnosis. In generalized forms the confusion with pityriasis rubra of Hebra is such that the two conditions are indistinguishable, unless typical lesions of lichen acuminatus are found. In forms more or less localized the typical lesions, and particularly their localization on the dorsum of the fingers, facilitate the recognition of the disease. The scaling plaques resemble psoriasis, but the scales and other characteristics of the latter exclude the likelihood of error in diagnosis. Lichen spinulosus is a rare follicular hyperkeratosis in which conical papules containing a spine two to three millimeters long are seen. Keratosis pilaris is common, and restricted usually to the backs of the arms and fronts of the thighs. Lichen planus, when its papules are perifollicular, closely simulates lichen acuminatus, but typical umbilicated papules and the greater frequency of the former condition, serve to exclude diagnostic error. The two diseases rarely coexist.

Etiology and Pathogenesis. In *Kaposi's* early series of cases arsenic was so efficacious that the possibility of an infectious origin of the disease gained serious support. Nothing definite is known, however, substantiating any of the numerous hypotheses advanced to explain the pathogenesis of the malady.

Treatment. This corresponds to that of lichen planus, although the disease may scarcely be called curable.

Prognosis. As a rule the prognosis is good. Deaths, excepting in *Kaposi's* original cases, are rare.

Lichen Spinulosus (Devergie), also called keratosis follicularis spinosa and lichen pilaris (Crocker), is a rare condition chiefly seen in childhood, and involving the neck, buttocks, belly, thighs, popliteal spaces, and extensor aspects of the arms. The lesions are conical papules containing a filliform, projecting plug or spine, about two millimeters long. The cause of the disease is unknown. Salicylic acid and tar salves cure it. It is probably no lichen, but a form of keratosis pilaris.

Lichen Nitidus is a rare papular disease, occurring on the genitals, breasts, cubital fossa, palms, and abdomen. Clinically minute lichen planus papules are suggested, and microscopically giant cells and other features recall the picture of tuberculosis.

LICHEN CHRONICUS (SIMPLEX) OR BETTER PRIMARY LICHENIFICATION (FIG. 32)

Synonyms. Neurodermite (Brocq and Jacquet); Prurit Circonscrit, or Prurit avec lichenification (Brocq); Lichen Chronicus Circumscriptus (Vidal); Eczema Papulosum (Hebra).

Definition. Lichen chronicus circumscriptus is a circumscribed, itching, scaling, and more or less sharply circumscribed thickening of the skin, preceded by pruritus, and favoring certain definite localities and prone to recur.

Symptoms. After a more or less prolonged period of itching, during which time no objective changes are present, the skin becomes pink or red, the color gradually growing more vivid. Then, flat, somewhat shiny papules arise, which increase in number until they are compactly grouped into plaques crossed by transecting furrows which are formed by the contiguous margins of the papules. Thus, a mosaic is built. When fully developed the plaque is five to fifteen centimeters in its greatest diameter, and in shape is oval or irregular. The favorite sites are the nape of the neck, elbow and knee flexures (Fig. 32), small of the back, scrotum, vulva, lower portion of the outer surface of the legs, thighs, crural folds, palms and soles; but any part of the body is vulnerable. Single or

multiple patches, symmetrically or asymmetrically arranged, may be present.

A fully developed plaque consists of three zones; an innermost, scaling, furrowed, infiltrated area, a second scaling, papular area, and an external or erythematous ring not more than three millimeters broad. The lesions may itch intensely, grow warty, or become excoriated. Larger or diffiuse plaques of the same general nature may include great areas on the extremities, sides of the body, abdomen, upper part of the thorax, *et cetera*. These are almost always symmetrical.

Course. Developing as described above, the lesions may increase in number, disappear spontaneously, remain away or recur, or they may persist indefinitely.

Varieties. There are varieties determined by site, appearance, and accidents of development, but these are too many and of too little importance for enumeration.

Differential Diagnosis. But three conditions simulate lichen simplex — psoriasis, lichen planus and secondary lichenification. The silver scales of psoriasis, the absence of furrows, the less marked infiltration, preference for the extensor surfaces, involvement of the scalp, and usually other typical lesions of the disease point out the probability of psoriasis. Lichen planus, unless typical papules and mucous lesions are present, is indistinguishable from lichen simplex. Secondary lichenification closely simulates primary, but it arises as a sequela of some pre-existing dermatosis, and thus its recognition is always simple. This question will again be discussed below.

Etiology and Pathogenesis. Very little definite knowledge of the origin of the disease exists. It is commoner among women than men, is associated with all sorts of metabolic disturbances, and frequently develops after psychic or emotional strains. Every explanation has been furnished that it is customary to advance in lieu of conclusive evidence.

Treatment. This corresponds exactly to the treatment of chronic psoriasis (Chapter XI). The Kromayer lamp is of great value.

Prognosis. In general, the plaques are amenable to therapy. Recurrences are likely. When a high degree of infiltration is present, the patches may be intractable. The general health is never affected.

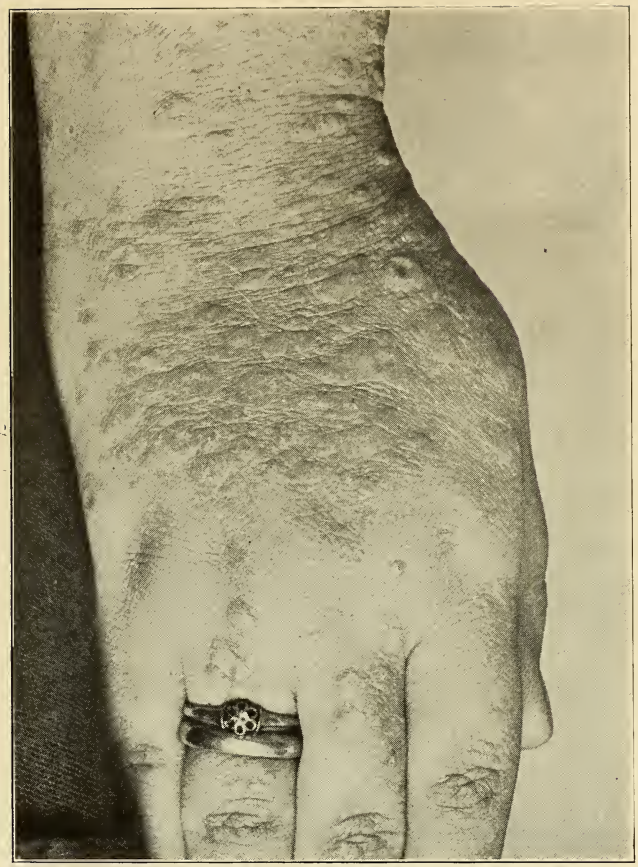

FIG. 27. LICHEN PLANUS

Examples are here shown of all types of lesions. The umbilications show distinctly, below the wrist and over the middle metacarpals. Near the rings are seen tiny fresh lesions. The lesions are elevated, polygonal and steep sided.

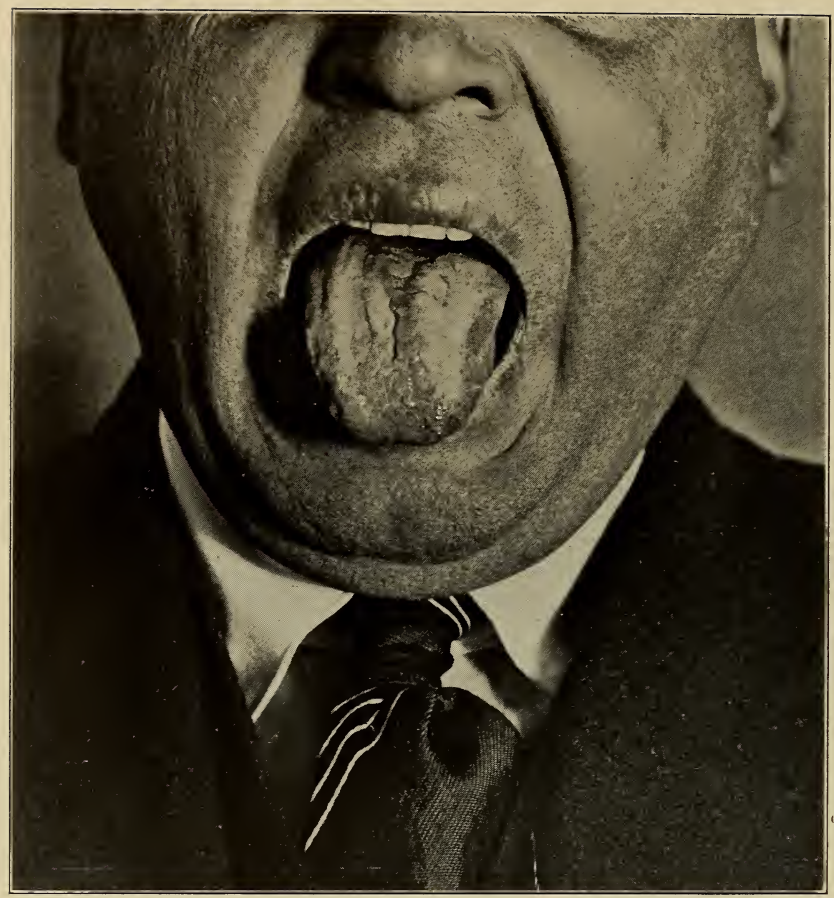

FIG. 28. LICHEN PLANUS OF THE MOUTH

Near the tip of the tongue are minute polygonal papules. Over the dorsum the lesions have coalesced into a plaque. There are papules and patches on the lips. All are shining and white. Syphilitic and non-specific leucoplacia are simulated, but in these conditions there are no distinct papules. Lupus erythematosus sometimes involves the lips, but its color is less snowy, and there is either more exudation or more atrophy than in lichen.

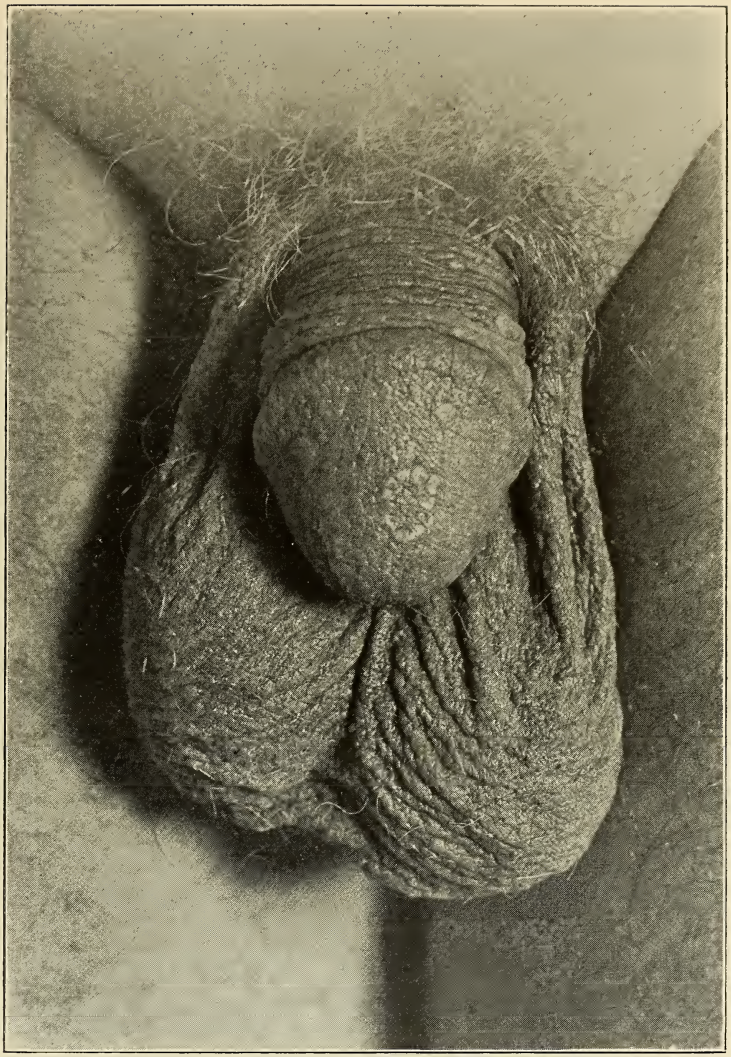

FIG. 29. LICHEN PLANUS ANNULARIS

This form may appear anywhere on the body, but is most frequently situated on the glans. A lavender ring of papules forms, some of which are umbilicated, and nearby lie isolated papules, as shown in the picture. The infiltration at times suggests that of syphilis, the annular forms of which are sometimes imitated so closely as to defy diagnosis, save that the specific forms do not itch, and are buff rather than purple.

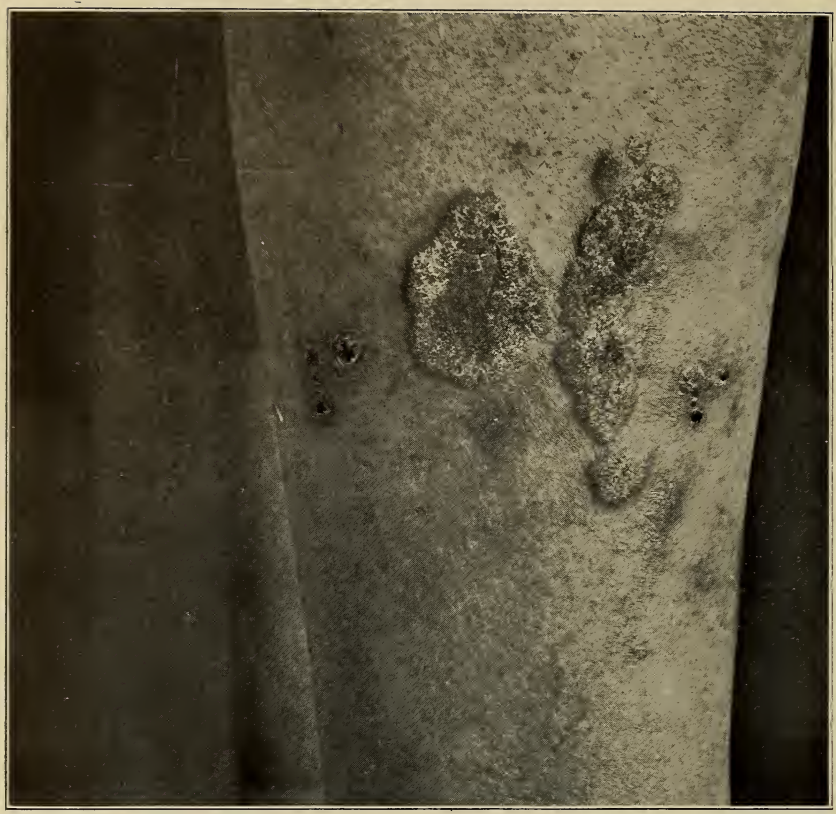

FIG. 30. LICHEN PLANUS, HYPERTROPHIC OR WARTY

This form may occur at other sites, but it favors the shins over the shoe-tops. It consists of warty, or at least raised, scaly, purple patches, composed of coalesced papules, the contiguous margins of which survive and remain separated by narrow linear furrows. Thus a curious cross-hatching arises, quite distinctive, and clearly indicated in the picture. Nearby there are often isolated papules. Lichen simplex, hypertrophic dermatitis, or thickened psoriasis may be simulated, and differentiation is sometimes impossible without discovery of pure lesions typical of the different diseases.

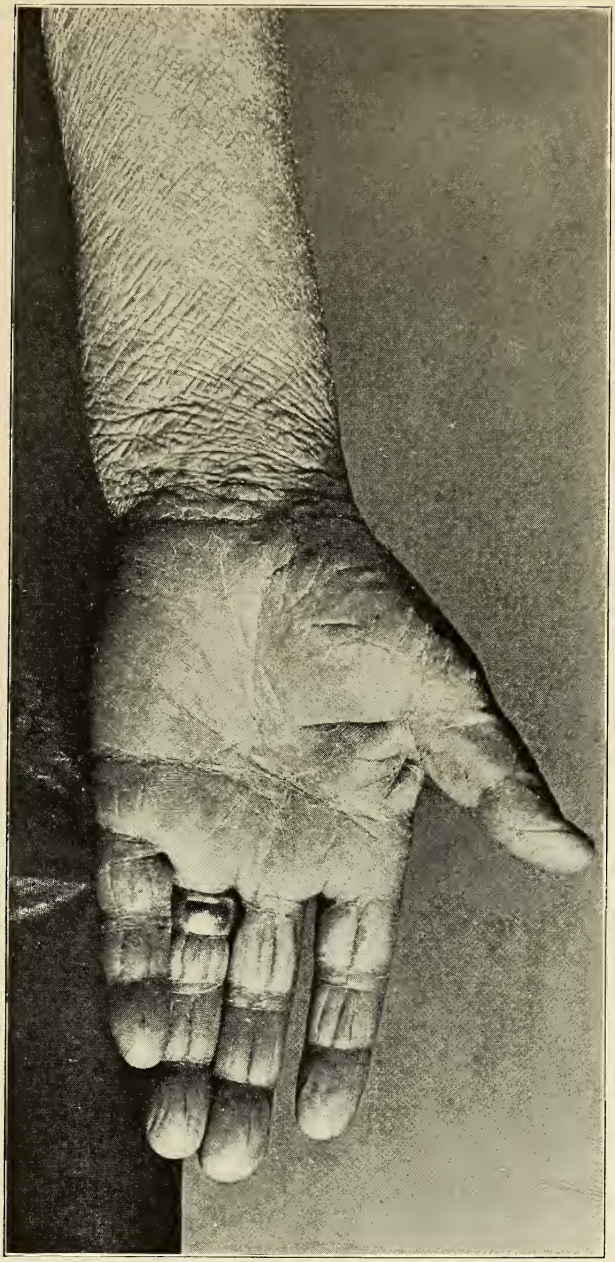

FIG. 31. LICHEN ACUMINATUS OR PITYRIASIS RUBRA PILARIS

Characteristic follicular papules are on the forearms. The wrist is lichenified, and the palm, tylotic. These features are distinctive.

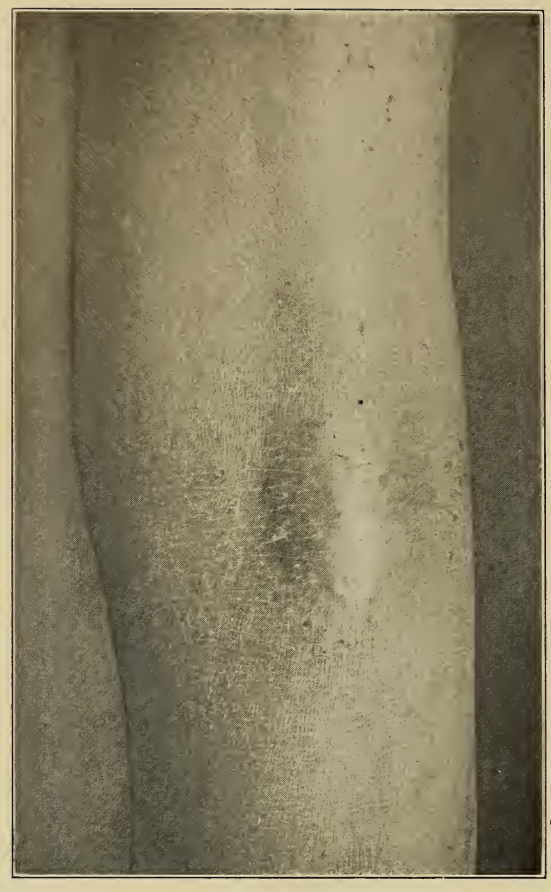

FIG. 32. NEURODERMITE

This condition is also called lichen simplex, primary lichenification or pruritis with lichenification. Its favorite sites are the popliteal and cubital bends, the small of the back and nape of the neck. It consists of thickening which is brown, buff or red and the area is traversed by the natural markings of the skin, exaggerated. Often scratched papules are studded over the affected surface.

SECONDARY LICHENIFICATION

This is a thickening of the skin arising in the course of itching dermatoses such as chronic dermatitis, prurigo, chronic urticaria and dermatitis herpetiformis. The appearance of the plaques is identical with that of those arising in primary lichenification. Their diagnosis rests upon their association with a preceding dermatosis. It is this form of lichenification which Hebra called papular eczema, because papular eczemas resemble it, and this was responsible for the confusion that has long existed in our conception of the relation of primary to secondary lichenification. The treatment and prognosis of the disease are determined by the nature of the underlying cutaneous affection.

GROUP IV. EPIDERMAL DYSTROPHIES AND HYPERKERATOSES

This group is complex to define. The epidermal dystrophies all possess the common attribute of peculiar epidermal inclusions originally regarded as pathogenic organisms, and which since have been found to be altered or perhaps degenerated rete cells incapable of complete keratinization, that is, of fully maturing. The process in question is termed dyskeratosis, and the cells in question are called dyskeratotic. At least one of these, the molluscum body of molluscum contagiosum, which is an infectious disease, must contain the pathogenic agent, although it has until now eluded identification. Verruca plana is also probably infectious, but presents no structure similar to that observed in molluscum, nor is the microscopic architecture of the lesion suggestive of any close kinship with the latter. On the contrary, its histology is like that of the common wart, or verruca vulgaris, namely an epithelial proliferation. It must be remembered though that molluscum is likewise due to such a proliferation, and one of the names of this disease is epithelioma molluscum.

The other dyskeratoses are psorospermosis, or Darier's disease, containing psorosperms which are only large degenerated epithelial cells, condyloma acuminatum containing the curious X-cell first noted by Unna; Bowen's precancerous dermatosis and Paget's disease of the nipple, both characterized by the presence of changed epidermal cells similar to those observed in psorospermosis.

The hyperkeratoses are noteworthy for the fact that without marked inflammation in the cutis the epidermis proliferates either within or without the follicles or both. A great number of clinical pictures results from this process. Whether the disease is congenital or acquired, an anomaly or the result of trauma, a familial or merely congenital disease, or one caused by some internal disturbance, the salient clinical feature is a hyperkeratotic plug, scale, or a combination of these; the salient histological feature is a thickening of the epidermis mainly due to hyperplasia of the corneous layer.

CHAPTER XIV

EPIDERMAL DYSTROPHIES; DYSKERATOSES

The diseases of this class are molluscum contagiosum which is infectious, psorospermosis, condyloma acuminatum, Bowen's precancerous dermatosis and Paget's disease. Verruca plana may perhaps belong here too, but since its only claim to such classification is its probable infectiousness, and since microscopically it resembles the ordinary wart, it will be described with the benign epithelial neoplasms. Molluscum contagiosum and condyloma acuminatum are common diseases; the others are comparatively infrequent.

MOLLUSCUM CONTAGIOSUM (FIG. 33)

Synonyms. Epithelioma contagiosum, Epithelioma molluscum. (There are other synonyms which have fallen into disuse and hence will be omitted.)

Definition. Molluscum contagiosum is a small cutaneous tumor, evidently infectious, not serious, incapable of spontaneous involution and easily removed. The tumors may be single or multiple and are at times numerous.

Symptoms. The tumors average the size of a pea, are pink, flesh-colored or whitish, rounded, sessile, but often slightly constricted at the base, and are pierced by a central aperture (Fig. 33). When compressed, a glistening waxy mass is extruded. The tumors may occur anywhere on the body, but the face, eyelids, nose, scrotum, labia, breasts and backs of the hands are the favored sites. At times infection takes place converting the mass into an abscess. There are no subjective symptoms.

Course. The lesions develop suddenly or gradually, and one lesion may persist for years, or they may become countless in number and vary in size. Extremely large ones, the size of an egg in fact, have been reported, but this is exceptional.

Pathology.[1] Histologically the lesions suggest the structure of a pomegranate. They are septate and consist of epidermal wedges containing rounded, enlarged structures, the molluscum bodies,

[1] This is of diagnostic importance.

which fit into the segments as do the seeds in the above mentioned fruit. The picture is pathognomonic.

Differential Diagnosis. Warts resemble molluscum contagiosum but contain no waxy mass. An infected molluscum looks like a furuncle or suppurating sebaceous cyst, and often cannot be recognized unless the characteristic waxy contents are found, or a microscopic study of the lesion is made. Eyelid molluscum often resembles the ordinary stye and a diagnosis is reached only as outlined in the previous sentence. To confuse molluscum with variola, or when it is on the genitalia with syphilitic chancre, is virtually impossible, and although the average text-book discusses these points, the matter is too absurd for consideration.

Etiology and Pathogenesis. The facts that a mother's breast may be infected by a nursing baby suffering with molluscum, and experimental evidence indicating its transmissibility, prove its infectious origin. The micro-organism has not yet been isolated, although Wile has isolated a filtrable virus which he thinks produces the local changes in the rete causing the molluscum bodies.

Treatment. The only rational treatment is the removal of the lesions by expression, with or without previous incision, curettage or puncture. The method employed is determined by the site, size and number of the lesions.

Prognosis. The disease is always curable and relapses are rare.

PSOROSPERMOSIS (FIG. 34)

Synonyms. Darier's disease; Psorospermosis follicularis vegetans; Keratosis follicularis; Keratosis vegetans.

Definition. Psorospermosis is a rare, probably familial disease the characteristic clinical features of which are follicular plugs and perifollicular papules, red, brown, or purple in color, fairly definitely localized, capable of proliferating and becoming crusted, ulcerated or vegetative. The anatomic structure is distinctive, indicating that the malady depends upon a disturbance of the normal maturing of the epidermis.

Symptoms. The lesions first appear on the head and face. Then the extremities, front of the thorax, groins, genitalia and flanks are involved. Punctate, follicular flesh-colored papules are the earliest lesions observed. These grow, becoming red, brown or purple and develop into crusted, ulcerating or papillomatous masses which in the body folds are macerated and may emit an offensive odor. The distribution of the efflorescences on the face is fairly

typical, the scalp, temples and nasolabial folds being first involved. As the papules grow the plugs they contain darken and look something like a blackhead, but are hard, not fatty, and difficult to express from the follicular openings. A hyperkeratosis arises on the palms, soles and backs of the hands, depending primarily upon the papules. Under the nails there is epidermal proliferation.

Course. Beginning gradually in adult life the disease progresses steadily, does not diminish the general health, and except for slight itching, causes no annoyance. *Wende* reported a unique case in which epithelioma developed.

Differential Diagnosis. Keratosis follicularis contagiosa of Brooke and acanthosis nigricans almost duplicate the clinical appearance of psorospermosis. The history, in the former, and the presence of a visceral cancer in the latter, as will be further emphasized below, exclude the likelihood of diagnostic error. Lichen acuminatus in its earlier stages resembles psorospermosis, but it never vegetates. A microscopic study is always of aid in recognizing the disease, since the structure of the lesions is characteristic. A follicular plug of corneous cells, and the presence of enlarged, round bodies, the corps rondes, or psorosperms, with the lack of marked alteration elsewhere, are distinctive.

Etiology and Pathogenesis. Nothing is known of the cause of the malady save that it frequently appears in many generations of the same family. The infectious theory of its origin has been abandoned, through lack of substantiation, and the psorosperms have long since ceased to be considered pathogenic organisms.

Treatment. Salicylic acid, salves and pastes and Roentgen therapy have caused amelioration, but no cures of the disease.

Prognosis. The disease is harmless, but incurable.

CONDYLOMA ACUMINATUM (FIG. 35)

Synonyms. Verruca Acuminata; Venereal Wart; Fig-wart; German, Spitzencondylom; French, Crêtes de Coq.

Definition. Acuminate condylomas are papillary excrescences developing upon the genital mucosa and contiguous surfaces. They may be single or multiple, or so numerous as to be compactly crowded. Secretion with crusting may form, and the entire mass may ulcerate and become fetid. The glans, prepuce, introitus and anus are the most commonly affected sites. The lesions are bright red on the mucous membrane and soft to the touch. When the neighboring skin is affected, the color is paler and the consistency

firmer. They resemble filiform projections, or are shaped like a cock's comb, or are delicately tufted.

Course. They rarely disappear spontaneously, but start gradually or suddenly and increase in number.

Diagnosis. When single they cannot be differentiated from papillomatous warts. When multiple their appearance is distinctive. Histologically, the epidermal cells are found to be large and have a clear space where the cytoplasm is degenerated. The nuclei are crescent shaped and excentric. These are the so-called X-Cells of *Unna* and were originally regarded as pathognomonic of the disease. Although this is not entirely true, there is no condition in which they are so numerous as in condyloma acuminatum, and this fact may serve as a diagnostic aid.

Etiology and Pathogenesis. They are precipitated by irritating secretions, leucorrhoea, balanitis, gonorrhoeal pus, discharges incident to pregnacy, *et cetera,* and are infectious, although the pathogenic organism is still unkonwn. They are not gonorrhoeal in origin as was once supposed. Gonorrhoeal discharge is but one of the elements favoring their development. They are auto-inoculable but not transmissible from one individual to another.

Treatment. They must be removed mechanically, either by the curette or high frequency spark. As the parts affected are highly sensitive, local or general anesthesia is necessary, according to the site and extent of the lesions. After their removal, antiseptic dusting powders must be used to prevent infection, or if the surface treated is extensive, large, antiseptic dressings must be applied. When the disease occurs in gravidae there is no sense in starting treatment until after the confinement, as recurrences are inevitable. It is necessary, however, in order to minimize the danger of puerperal infection, to treat the parts with mild antiseptics, and to cleanse them even more thoroughly than usual at the time of the delivery. At times involution may be caused by powdering with equal parts of alum and calomel.

Prognosis. The condition is invariably curable.

Verruca Plana Juvenilis (Fig. 36) are flat warts mentioned here because they share with molluscum and pointed condylomas the characteristics of infectiousness. They are, however, not dyskeratoses, but hyperkeratoses, and will be described in detail with the benign epithelial neoplasms. (Chapter XXVII.) They too, according to Wile, are due to a filtrable virus.

Bowen's Precancerous Dermatosis (Fig. 37) is a dyskeratotic condition closely related to the senile wart. It is included here to complete the classification of dyskeratoses, for microscopically there are corps rondes as in

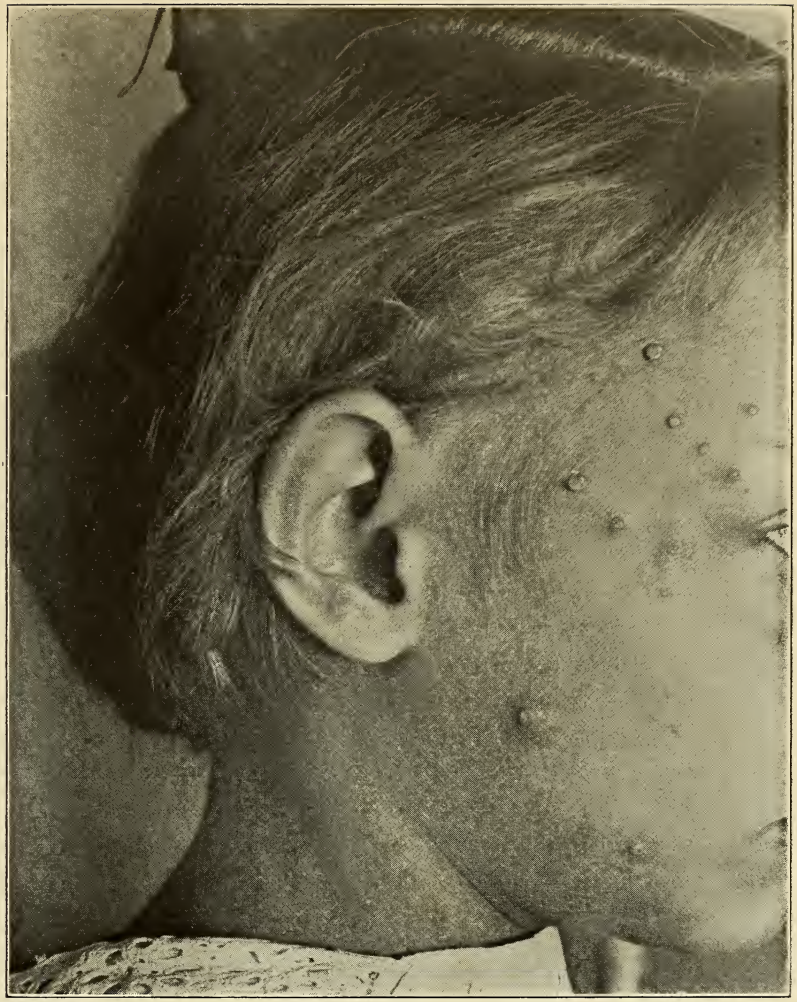

FIG. 33. MOLLUSCUM CONTAGIOSUM

In this disease, small yellow, pink or red papules or nodules arise, mainly on the face but also elsewhere. A small pore or depression is found in the center; and on pressure, a white, waxy glistening mass may be forced out, which microscopically shows molluscum bodies. Clinically, the disease can scarcely be confused with anything else.

FIG. 34. KERATOSIS FOLLICULARIS CONTAGIOSA

This disease is called Darier's disease and psorospermosis, a term now obsolete. This is a classical picture. The lesions are small, brown, follicular, conical papules which first appear in the body folds and then become generalized, forming large patches covering great areas. Only acanthosis nigricans, a still rarer disease, is remotely simulated. The latter is associated with visceral malignancy. Darier's disease has a fairly characteristic histology, peculiar rounded bodies being present in the epidermis near the follicles.

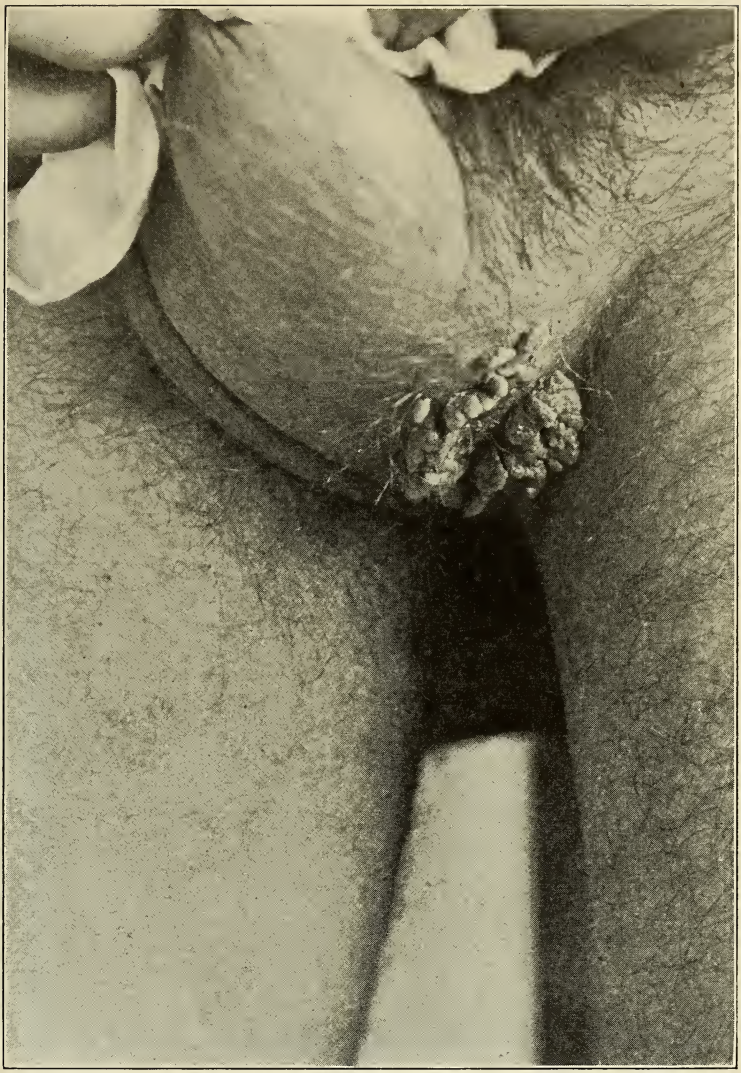

FIG. 35. CONDYLOMA ACUMINATUM OR VERUCCA ACUMINATA

This is a papilloma of the genitalia, and may occur anywhere on these organs. The lesions are pointed, fleshy, red, brown or purplish, pointed papillomata.

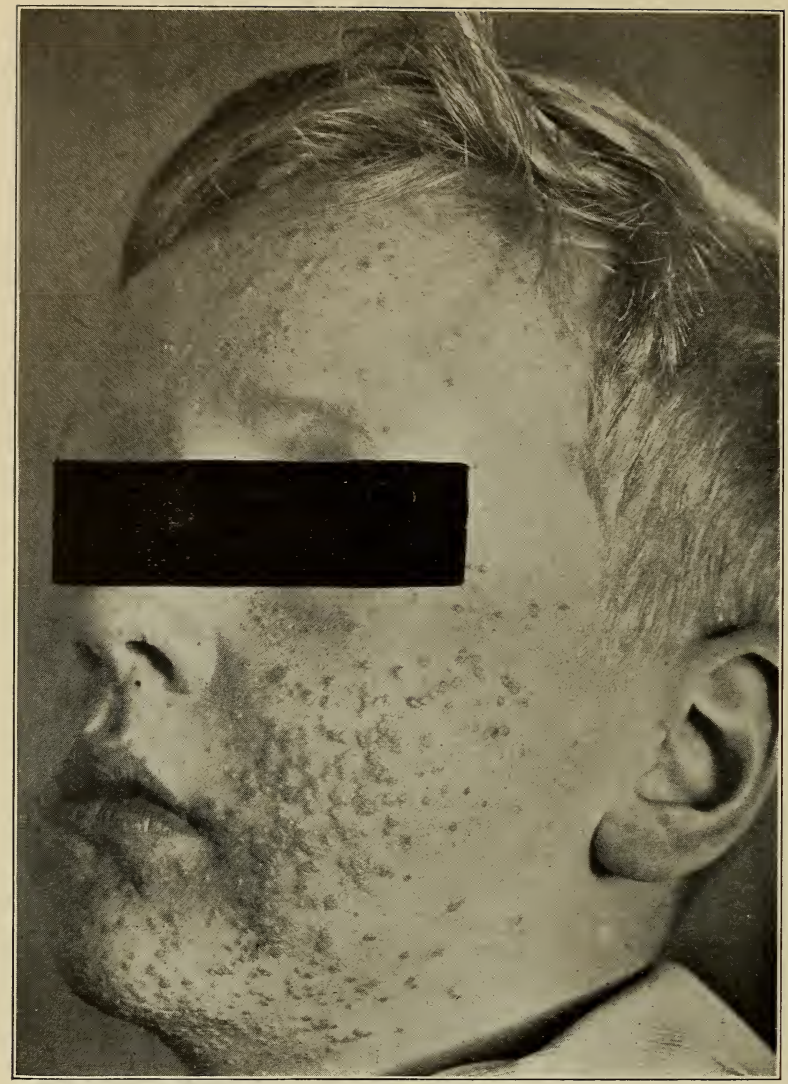

FIG. 36. VERUCCA PLANA JUVENILIS

The face and back of the hands are covered with minute waxy, yellowish, glistening papules. In this patient, only the facial lesions are shown. There is no inflammation, but from a distance at times, milia-crystallina, or early lichen are suggested. The former is excluded by puncturing the lesions. In milia fluid would exude. The latter is excluded by the rarity of lichen in children, and the great rarity, at any age, of facial lichen; to say nothing of other evidence of the latter such as itching, mucus, involvement, etc.

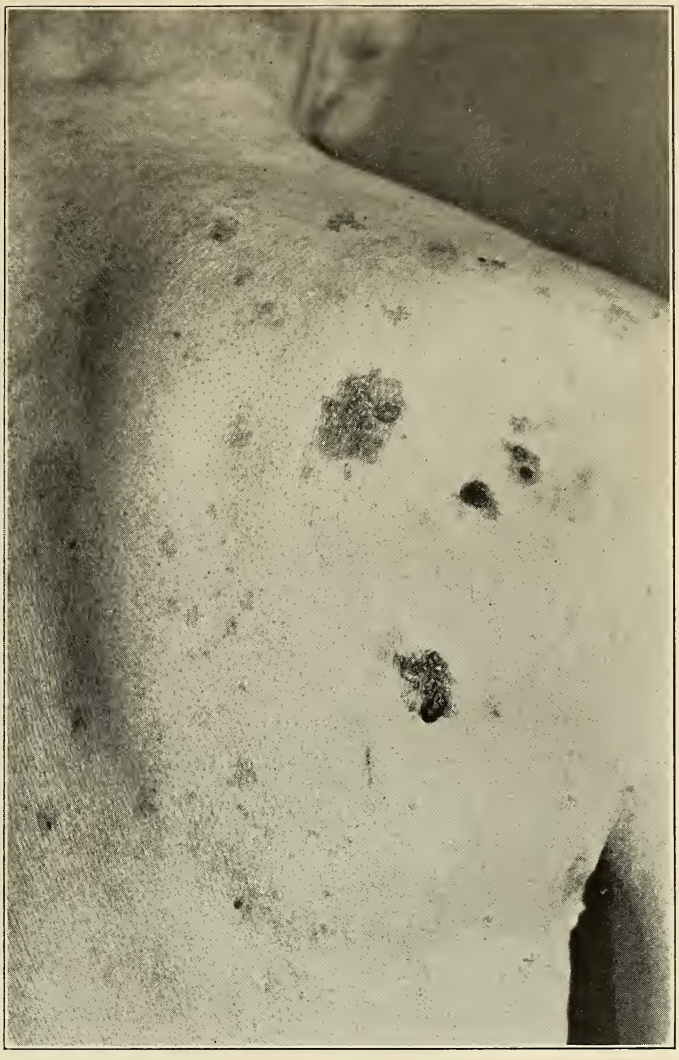

FIG. 37. KERATOSIS SENILIS, OR SENILE OR SEBORRHOEAL WART

This is found on the face, back or chest of the elderly or aged. The lesions are flat or slightly raised, circular, oval or somewhat irregular in outline, and covered by a scale that is yellow, buff or gray, brown or black, and hard, rough and dry. At times such lesions give rise to epitheliomata and are closely related to Bowen's precancerous dermatosis. When grouped, they may resemble tuberous gummata and the Wassermann test or a histological examination may be required to make the diagnosis.

psorospermosis. It will be described in detail in connection with senile hyperkeratoses and verrucae (Chapter XXIX), and its relation to epithelioma will be discussed in this connection and in the chapter on cutaneous cancer.

Paget's Disease, both mammary and extramammary, also contains psorosperms, but the disease will be referred to more completely in the discussion of the classes of epithelioma. (Chapter XXIX.)

CHAPTER XV

HYPERKERATOSES

The hyperkeratoses are varied in their appearance and causation. Their common bond lies in the fact that they all possess a thickened stratum corneum. They may be classified as follows:

Hereditary:	Keratosis palmaris et plantaris (Mal de Meleda)
Congenital:	Keratosis exfoliativa congenita Ichthyosis (probably a Naevus — Chapter XXXII)
Congenital Gaining Expression in Adult Life:	Keratosis pilaris
Pure Follicular Hyperkeratoses:	Lichen spinulosus (Chapter XIII.) Ulerythema ophryogenes Lupus Erythematosus (possibly a tuberculide — Chapter XXV)
Infectious:	Keratosis follicularis contagiosa
Metabolic:	Acanthosis nigricans
Traumatic:	Callus Clavus
Regressive:	Keratosis senilis (see Seborrhoea — Chapter XI and Epithelioma — Chapter XXVIII)
Unknown Origin:	Cornu cutaneum (see Epithelioma — Chapter XXVIII) Porokeratosis.

Clinically, these diseases are characterized by a thickening of the corneous layer causing either sessile, solid elevations as in corns,

calluses, horns and palmar keratoses; follicular hyperkeratoses with plugs as in keratosis pilaris, lichen spinulosus, ulerythema ophryogenes, lupus erythematosus, keratosis follicularis contagiosa and acanthosis nigricans; or scaling as in ichthyosis, keratosis exfoliativa congenita, lupus erythematosus, and porokeratosis; atrophy as in keratosis pilaris, ulerythema ophryogenes and lupus erythematosus; or some combination of these features as is obvious from the above, and as will be further indicated in the ensuing. The majority of these diseases cause no subjective symptoms. Many of the diseases are rare; those of practical significance are ichthyosis, keratosis pilaris, lupus erythematosus, callus, clavus, keratosis senilis, cornu cutaneum. The less usual ones will receive scant mention and this only for the sake of completeness. Lichen spinulosus has already been described (Chapter XIII).

ICHTHYOSIS

Synonyms. Fishskin Disease.

Definition. Ichthyosis is a congenital skin anomaly characterized by dryness, harshness and scaling. There are three commoner types of the disease, ichthyosis simplex, hystrix, congenita, and two rare forms, ichthyosis follicularis and erythroderma congenitalis ichthyosiforme.

Symptoms. Ichthyosis simplex is the mildest form. The skin is rough, dry, impliable, covered with furfuraceous scales. The desquamation is fine or flaky, and the extensor surfaces of the extremities may be involved alone, or the entire body may participate in the process. The color ranges from the normal flesh tones to a dirty gray or brown. Although the disease is congenital, its first manifestations appear at the seventh or eighth week and increases in severity with years. During the summer it often improves or disappears entirely, getting worse in winter. Xerosis is another name for this mild type, which may persist throughout life, and grow gradually worse constituting a so-called xeroderma, which must not be confused with xeroderma pigmentosum. Severer forms closely resemble reptilian skins and are called sauriasis. In the fish skin and reptilian or alligator skin forms the scales look like those seen on the animals in question and are separated from each other by depressions.

Ichthyosis hystrix is a form in which are present scales and patches with spinous projections arising from papules or verrucoid areas. In this form the lesions occur in circumscribed islands,

linear, plaque-shaped or irregularly outlined. They closely resemble hyperkeratotic naevi which they probably are. This view is widely accepted and appears logical if the question of naevi is approached with breadth of view.

Ichthyosis congenita is rare. It is also called keratosis universalis congenita. There are two varieties; one always fatal, the other often mild. The former is peculiar in that the fetus appears as if covered by a coat of mail. The face looks mummified, and the digits resemble claws rather than fingers and toes. The milder form presents a papyrus-like skin which after a few days splits into large scales, under which there is a normal integument. These cases recover, or develop a mild form of ichthyosis which persists.

Ichthyosis follicularis is rare. There are only three cases reported. *Erythrodermie congenitale ichthyosiforme* of Brocq is a congenital malady resembling ichthyosis, but the skin is red as though inflamed. It is usually seen only about the face, neck, body folds, flexor surfaces; associated seborrhoea of the scalp is usually observed and the general health remains good.

Course. Simple ichthyosis appears two months after birth, improves in the summer, is augmented in the winter, and either stays unchanged throughout life, or gets worse at puberty, remaining unchanged after this period. Ichthyosis hystrix, follicularis and congenital ichthyosiform erythroderma never change. Only one form, as noted above, is fatal, or the afflicted individual is born dead, and this is the malignant type of congenital ichthyosis.

Varieties. These have been enumerated in the description of the disease.

Differential Diagnosis. Except in ichthyosis hystrix, which those who exclude it from naevi would have to differentiate from the latter, the picture is so characteristic that no other condition is to be considered.

Etiology and Pathogenesis. Barring the certainty of its being a congenital and in some cases an hereditary anomaly, nothing is known of its causation.

Treatment. Oils, fats, and X-Rays are employed and occasionally a cure is effected. Salicylic acid may be incorporated into the oils up to five percent. and daily inunctions given. This simply serves to increase the pliability of the skin. Eucerin and cold cream are valuable, and divided doses of X-Rays are particularly to be recommended. Ichthyosis hystrix responds best to carbon dioxide snow, massive doses of X-Rays and it is probable that radium will prove to be of great value.

Prognosis. The disease is rarely curable and equally rarely harmful.

KERATOSIS PILARIS

Synonyms. Keratosis suprafollicularis (Unna); Lichen pilaris; Pityriasis pilaris. Probably Unna's designation is the most accurate as the chief anatomical changes are found high in the follicle.

Definition. Keratosis pilaris is a hyperkeratosis causing the formation of a horny plug in the upper third of the pilosebaceous follicle. About the affected ducts a red or white papule arises.

Symptoms. The disease is common and consists of perifollicular papules surmounted by horny plugs filling the mouths of the affected organs. The plugs are short, white, gray, or dark, often contain an atrophied hair which may be curled up or project slightly, and the sites usually affected are the extensor aspects of the limbs, the thighs, and even the shoulders. Occasionally, the face is involved. The papules are white or red, and the involved skin feels rough. The depth of the red varies from pink to purple, according to the degree of vascular dilatation present.

Course. The disease sometimes appears in childhood, more frequently at puberty, and thereafter persists throughout life, tending, however, to decrease after the third decade. In disappearing, it leaves atrophic spots causing a slight wrinkling of the skin at the site of the former papules.

Varieties. Keratosis pilaris alba and rubra are the only two known types.

Differential Diagnosis. Only lichen acuminatus and lichen spinulosus resemble keratosis pilaris. The history, onset, commonness of the disease, its typical localization and absence from the fingers, differentiate it from lichen acuminatus. From lichen spinulosus it is distinguished by the lack of spines.

Etiology and Pathogenesis. Puberty is one element in its causation. This brings it into direct connection with changes in the endocrinous glands. By many it is regarded as a form of ichthyosis. It is so common — in fact, few individuals fail to present a mild form of it — that it must be hereditary in the broadest sense. Uncleanliness is regarded as a cause. This is absurd, although frequent bathing reduces the intensity of the symptoms by removing the plugs.

Treatment. This corresponds to that of ichthyosis.

Prognosis. The disease is harmless but in general incurable, although its extreme manifestations may be ameliorated.

Lupus Erythematosus. This disease will be taken up in detail in Chapter XXV. One of its features, however, the presence of horny plugs at the bottom of its scales, indicates a certain relationship to the hyperkeratoses. It is only its possible tuberculous origin, and the fact that it appears in non-keratotic forms, that make it advisable not to include it too arbitrarily in the hyperkeratoses.

CALLUS

Synonyms. Callositas; Tylosis; Keratoma; German, Schwiele.

Definition. Callosities are circumscribed, thick, horny patches of epidermis, usually due to pressure, and found as a rule on the palms and soles.

Symptoms. The appearance of these lesions is so well-known that a description is unnecessary. Unless they become infected, or unless the pressure which provokes them is not properly relieved from time to time, they do not hurt.

Course. While the exciting condition obtains they persist.

Diagnosis. They must be differentiated from keratosis palmaris et plantaris hereditaria and arsenical keratoma. This is always easy if a careful history is taken.

Etiology and Pathogenesis. The lesions result from the pressure of ill-fitting shoes, various occupations, such as metal working, the playing of certain instruments, notably the banjo and harp, and even the piano. Thus, the site of a callosity often indicates the nature of the patient's work.

Treatment. Applications of salicylic acid plaster, ten to twenty-five percent., or salicylic acid in collodion are the best methods of treatment. Surgical removal, or the use of the high frequency spark, is often indicated. The only rational therapy is the elimination of the exciting cause. The Roentgen rays are often of great value.

CLAVUS

Otherwise known as Corn.

It is unnecessary to enter into details concerning this common affection.

KERATOSIS SENILIS (FIG. 37)

In the aged the face, chest and back become covered with papules possessing a hyperkeratotic scale, gray or brown in color. These lesions vary in size up to that of a dime and are often one-eighth inch high. They are greasy, look like flat warts, and are often called senile warts. Many authors include them among precan-

cerous dermatoses, a point which will be discussed in the etiology of epithelioma. (Chapter XXIX.) They are readily cured by sulphur salves, and five to ten percent. salicylic acid plaster. They occur anywhere that seborrhoea occurs and are distinctly related to senility.

CORNU CUTANEUM

Cutaneous horn, is a rare hyperkeratosis which may be small or large, and roughly simulates horns as seen in lower animals. The growth may be straight, conical, twisted, or irregular in shape or size. Usually the lesions are single, rarely multiple and their favorite sites are the scalp, brow, temples, nose, penis, and finally anywhere else on the body or face. Their cause is similar to that of senile keratoma, but they often spring from scars of lupus vulgaris, burns, or cicatrices resulting from strong acids and alkalies. At times epithelioma develops at the base. The treatment consists of excision, and if epithelioma is present, of massive doses of X-Rays to the scar resulting from the operation.

KERATOMA PALMARIS ET PLANTARIS

Synonyms. Keratoderma palmaris et plantaris; Congenital palmar and plantar keratosis; ichthyosis or tylosis of the palms and soles; Mal de Meleda.

Definition. The disease is a familial, often hereditary callosity of the palms and soles, one form of which is indigenous to the Dalmatian Island of Meleda.

Symptoms. The palms and soles are thickened as to their epiderm. They are thus covered by a horny plate which is smooth yellow, unless it becomes soiled by work, when the surface is gray, brown or black, pitted, fissured or verrucous. At the margin of the lesion the normal skin appears abruptly.

Course. The disease is permanent.

Varieties. There are two varieties, the acquired and hereditary which may be congenital, or appear long after birth. Mal de Meleda belongs to the second group.

Differential Diagnosis. The true palmar and plantar keratoma must be differentiated from the hyperkeratosis developing at these sites in chronic dermatitis, syphilis, lichen planus and acuminatus, arsenical keratosis, callus, and rarely in psoriasis. Other evidences of the first four diseases clear up the differential points involved in their recognition. The history is the determining factor in recognizing arsenical keratosis. Trauma and occupation are the determining factors in diagnosticating callosities. Palmar and plantar psoriasis are so rare that it is useless to dwell upon it.

Treatment. In the acquired forms the removal of the predisposing cause

will often produce healing as in callosities. The familial varieties are incurable. X-rays may be tried and often produce improvement.

Prognosis. This depends upon the cause.

Keratosis Follicularis Contagiosa (Brooke).

This rare condition arises in children, on the neck, face, shoulders, and extensors of the upper extremities. It somewhat suggests psorospermosis, is infectious, and easily cured by mercurial antiseptics, particularly the iodine salts applied locally.

Porokeratosis of *Mibelli* and *Respighi* is very rare. Its main characteristic is a prismatic wall encircling a normal, atrophied or hypertrophied basin. From the inner aspect of the wall a fine scale projects toward the basin, and this scale resembles a collar. The summit of the wall is depressed into a gutter. The disease cannot be cured. It affects the extremities, particularly the hands and feet, more than any other part of the body.

Acanthosis Nigricans, keratosis nigricans, or dystrophie papillaire et pigmentaire of the French, was first described by *Sigmund Pollitzer.* The disease is rare, occurs in the body folds, chiefly the axillae, groins, inner sides of the thighs, bends of the elbows and knees, on the belly, forearms, perineal region, face, umbilicus, scrotum and about the anus, although the entire skin may be covered. The color of the lesions varies from yellow or brown to black, and tubercles, papillomas, or vegetating masses are seen. The tumors are sessile or pedunculated, and frequently all sorts of warty or nevoid lesions co-exist. An adult variety is known associated with visceral malignancy, and a second variety of which the causation is not understood. The therapy is usually ineffective and the prognosis always grave, although some cases improve when the visceral tumor is removed. Local therapy with oils, salves and plasters is of slight benefit.

Keratosis Exfoliative Congenita. This is a rare exfoliative congenital dermatosis, probably related to ichthyosis and allied with the still rarer ichthyosis rubra of Rasch. The condition is so uncommon that, for practical purposes, it requires scant mention.

GROUP V. DYSTROPHIES OF THE SKIN AS A WHOLE

This class of diseases includes four sub-groups, hypertrophies of the connective tissue, atrophies of the connective tissue, pigmentary diseases, and finally, degenerations. Save for the general fact that all of the conditions here included are basically alterations in the collagen or elastic tissue, if not both, there is no striking relationship among the groups. Collagenous and elastic tissues, the blood and lymph vessels therein imbedded, the pilosebaceous and sweat structures, and the arrector muscles, form a background for a great variety of changes, and respond variously to diverse pathogenic forces.

CHAPTER XVI

HYPERTROPHIES OF THE CONNECTIVE TISSUE

Three common and six rare diseases constitute this series, and another condition, paraffin prosthesis, due to the presence of a foreign body exciting a fibrosis, must here be mentioned. The pathology of the last named is determined by the attempt of the body to guard itself against an alien substance. The common conditions are the scar, keloid and elephantiasis; the rarer ones are sclerema neonatorum, edema neonatorum, hereditary edema of the legs, acromegaly, myxedema and cutis verticis gyrata.

SCAR

Synonyms. Cicatrix; German, Narbe; French, cicatrice.

Definition. A scar is the result of the reparative process following an injury to the skin involving at least the papillary body, if not deeper levels. It is composed of fibrous connective tissue.

Symptoms. Scars may be normally, excessively, or subnormally sensitive, or even insensible. Pain may be spontaneous, or elicited only by touch. To the eye the lesions may be flush with the skin, depressed, raised, or have a raised margin and a depressed centre. Some cicatrices are markedly raised, and these are known as hypertrophic or keloid scars. In shape and size they vary according to the determining injury, and their depth depends upon whether

the papillary body or still deeper levels have been destroyed. Their color varies; fresh scars are red or blue because they are rich in vessels; older ones tend to be white. Some have the normal flesh tint, others are depigmented, others are pigmented, and still others have white centres and brown rims. Usually they are movable; occasionally fixed to the deeper tissues.

Scars resulting from syphilis tend to be white within and brown at the margin; they are crinkly and thin, reniform, fan-shaped, circular or oval, single or grouped according to the lesions producing them. Scars resulting from tuberculosis, particularly those following lupus vulgaris, are white, redundant, corded and irregular in outline. Scrofula produces cicatrices fixed to the deeper tissues. Zoster causes grouped, small, pitted cicatrices at the site of distribution of the original eruption. Variola, varicella, acne varioliformis and the tuberculides produce small pits, no larger than a bean with deep, steep sides and a white base. Morphine addicts have similar scars from infected hypodermatic injections. The only free areas are those inaccessible to the patient's hands. This fact makes the picture characteristic. Ecthyma and vaccination form white, pliable cicatrices, often cribriform because they are so superficial that the follicles are not destroyed. Acne forms irregular, small pitted scars. Cicatrices due to trauma, surgery, acids, caustics, burns, *et cetera,* vary in size, shape, contour and color and other attributes, according to the nature of the injury. The possibilities are so numerous and the pictures presented so well-known that further description is unnecessary.

Varieties. Scars are atrophic, hypertrophic, pigmented or depigmented, as mentioned above.

Differential Diagnosis. There is no difficulty in recognizing cicatrices.

Etiology and Pathogenesis. This is obvious.

Treatment. Only excision, either with or without skin grafting, is of avail.

Prognosis. Scars are permanent.

KELOID (FIG. 38)

Synonyms. Kelis; Cheloid.

Definition. A keloid is a connective tissue hypertrophy, irregular or regular in shape, usually presenting dentritic or fan-like proliferations, and in its true form arising spontaneously. It is more common among negroes than whites.

Symptoms. Keloids start as nodules which progress becoming ribbon or strand-like, fan-shaped, arborescent, or irregular in outline. They are hard, raised, pink or red, if vascular; white or brown, according to their pigment content, and they may or may not be painful or tender. Any part of the body may be affected, but the chest, face and neck most frequently are, and of these chiefly the first, over the sternum. The lesions may be single or multiple and in the latter eventuality a dozen or more may be present.

Course. The disease usually begins in adult life and develops slowly, progression being gradual but inexorable.

Varieties. There are two varieties, the true or spontaneous keloids which are fibromata (Chapter XXX), and the false or spurious which are hypertrophic cicatrices (see above), resulting from injury or disease.

Differential Diagnosis. The appearance is typical and the diagnosis is made upon this point.

Etiology and Pathogenesis. This is unknown, but race or heredity must have some bearing on their causation, since the disease is so much commoner among negroes than among other people. Probably true keloids are fibromata.

Treatment. Fibrolysin and thiosinamin have been used, on the whole without favorable results. Roentgen (four Holzknecht units repeated once or twice at intervals of a month) and radium therapy are successful very often. Excision, cauterization, *et cetera,* are useless, as recurrences almost invariably take place.

Prognosis. On the whole the prognosis is bad because of the tendency toward recurrence even though therapy may be successful with reference to given lesions.

ELEPHANTIASIS

Synonyms. Elephant, Barbados or Cochin Leg; Pachydermia; Elephantiasis Arabum; Hypersarcosis.

Definition. Elephantiasis is a chronic cutaneous and subcutaneous hypertrophy and hyperplasia, usually secondary to obstruction of the lymphatics.

Symptoms. The organs most frequently diseased are the lower extremities, genitalia, upper extremities, lips, ears, or other parts of the face. Any part of the body, however, may be affected. At first the skin is tense, white and thickened; then these characteristics become augmented, and gradually warty, scaling, pigmentation, with atrophy or hypertrophy of the skin organs, develop. The hair and

nails become brittle and discolored, or fall off. There is pitting on pressure and often painful lymphangitis exists. In later stages secondary dermatitis, ichthyotic changes, marked verrucosities, fissures, furrows, deep pigmentation appear and secretions and detritus accumulate. When extreme the enlargements are huge, the leg becoming elephantine, the genitalia enormous and pendulous. Deep fissures and lymphorrhoea ensue. Occasionally itching, burning or pain are present, and genital involvement is accompanied by epididymitis, hydrocele, and hernia. There may be reflex nausea and vomiting.

Course. The disease usually follows on inflammation or infection which produces lymphatic obstruction; such as filariasis, erysipelas, cellulitis, lymphadenitis, or the removal of lymph glands. Recurrences of the local inflammations, often accompanied by fever, increase the degree of the pathological process, intensifying the anatomical changes until the picture above described is produced.

Varieties. The clinical varieties have been indicated in the description. They are telangiectatic or naevoid elephantiasis, and the lymph scrotum.

Elephantiasis telangiectoides, telangiectatic lymphangitis, or naevoid elephantiasis is congenital and often associated with a naevus or hemangioma. The lesions are usually visible, but at times may be detected only by palpation, the overlying skin being raised though otherwise normal. The lips and cheeks are thus affected with perhaps more frequency than any other part of the body (Chapter XXXII).

Lymph scrotum is also called nevoid elephantiasis or varix lymphaticus. It starts with fever, swelling, redness and vesiculation of the scrotum. The vesicles burst and lymph wells up. A constant increase in all the symptoms takes place.

Differential Diagnosis. The condition is easy to recognize, but it is important to determine the underlying cause. (See Etiology.)

Etiology and Pathogenesis. Filaria are the cause of most of the tropical forms. The other varieties, excepting the congenital naevoid type, are due to mechanical obstruction of the lymphatics, following neoplasms, scars, pregnancy, ulcers, erysipelas, chronic dermatitis, syphilis, tuberculosis, lymphadenectomies, and diseases of deeper tissues such as osteomyelitis. These factors, either by pressure or inflammation, stop the lymph circulation and thus, through lymphedema and consequent over-nutrition, provoke hyperplasia and

hypertrophy of the tissues with such secondary changes as the formation of warty proliferations, lymphangiomata and the like.

Treatment. Aside from prophylaxis as regards filariasis, only surgery offers any hope in elephantiasis. In mild cases elastic bandages are useful. Scars must be excised if it is thought that these, by pressure, produce the disease. The local skin modifications secondary to elephantiasis must be treated symptomatically according to their nature. This, however, is purely palliative.

Prognosis. The prognosis is always poor as regards overcoming the condition, unless the underlying cause is curable.

The rarer conditions are numerous, but so unusual as to require but the briefest mention. They all exhibit a hyperplasia of the connective tissue, or an alteration thereof, a chemical disturbance leading to its volumetric increase. Etiologically the conditions are perhaps unrelated. Two, sclerema and edema neonatorum are congenital; one, Milroy's disease, is hereditary; two, acromegaly and myxedema, are due to a disturbance of the endocrinous glands, and one, cutis verticis gyrata, is still unexplained as to origin. In addition, dermatolysis must be here included, largely because it appears to be either a connective tissue or elastic tissue disease, and although it is not actually a hypertrophy, it has some features suggesting association with this process.

Sclerema Neonatorum, also called *Scleroderma Neonatorum,* occurs some time between birth and the tenth day of life. The lower extremities turn white, feel impliable, and the disturbance progresses to the lumbar area creeping upwards, and finally, if the child lives long enough, includes the entire skin. The integument feels as if it were half frozen, the joints are immobile, the mouth can be opened with difficulty, the general temperature is subnormal and the pulse is slow. Prematurely born and weak infants present the condition in a primary form, but it arises also secondarily as a result of persistent diarrhoea.[1] The treatment consists of maintaining the body temperature by applying heat externally, but usually the patient dies.

Edema or Scleredema Neonatorum also arises in premature, feeble babies, starting as an edema which pits on pressure and first appears on the lower extremities. Recovery is possible but rare, and the edema progresses, the child growing drowsy and finally comatose. Death results from asthenia or secondary infection. The condition clinically differs from sclerema only in that the skin in the former does not pit on pressure; and pathologically by virtue of the fact that sclerema exhibits fatty acid crystals and scleredema the ordinary minute anatomy of edema. Nearly all of the afflicted die.

[1] Probably this is primary also, the diarrhoea and the skin condition both being due to the same underlying cause.

Milroy's Disease, or hereditary edema of the legs, is hereditary and familial. The legs start to increase in size shortly after birth, and become large but not shapeless as in elephantiasis. At times, the involvement extends above the knees. None of the characteristics of sclerema or scleredema are present, and the patients usually complete the normal span of life.

Acromegaly is due to a hypertrophy of or neoplasm in the pituitary body. Aside from the well-known evidences of gigantism, the skin and mucous membranes show alterations such as hyperpigmentation, scleroderma, hyperhidrosis, hirsutes and hypertrophic scars. The patient is often adipose and the nails are sometimes distorted, flattened or grooved.

Myxedema, also called thyroid cachexia, cachexia strumipriva, or cretinoid edema, may be acute or chronic and appear either in infancy or adult life. The latter form is oftener encountered in females than males at or about the time of the menopause. The patient is sluggish, the skin translucent, thick, waxy, yellowish and dry, or dry and scaly. Over the malars there is usually a flush, the eyelids look puffy, and because of swelling the nose is thick, the eyes dull, and the cheeks covered by thickened skin. The integument elsewhere, particularly over the extremities, wrinkles. About the neck there is an increase in fat causing a cushion-like swelling. Alopecia develops, surviving hairs being lustreless, dry and brittle. The nails furrow, hyperpigmentation appears even to a deep bronzing, the general development is retarded and mentality is of a low order. Thyroid insufficiency is the cause. The diagnosis is simple and the treatment consists of thyroid administration which in many cases produces a cure. In some instances thyroid transplantation from sheep has produced a permanent cure, thus obviating the necessity of constant internal administration of the gland substance.

Cutis Verticis Gyrata is extremely rare. It involves the scalp, so furrowing the latter that it simulates a convoluted surface.

Dermatolysis includes two types of disturbances. The first is characterized by masses of pendulous integument almost anywhere on the body, some of which suggest fibromata. The second, also known as cutis laxa, elastic skin, or hyperplastic skin, is a condition in which the integument, as in a puppy or kitten, may be picked up in folds. Upon release, the stretched skin springs back into place.

Paraffin prosthesis is essentially a connective tissue hypertrophy, but it may be regarded as a foreign bodied tumor. It arises about masses of paraffin injected for cosmetic or legitimate surgical purposes, but which in susceptible people stimulates connective tissue hyperplasia and inflammation. The site injected and immediate neighborhood presents the lesion, the size of which is determined by the amount of paraffin introduced. A tumor develops which clinically may resemble a keloid, fibroma, leukemic infiltrations, sarcoma, or sarcoid. It is hard, white, red, purplish or brown in color. It

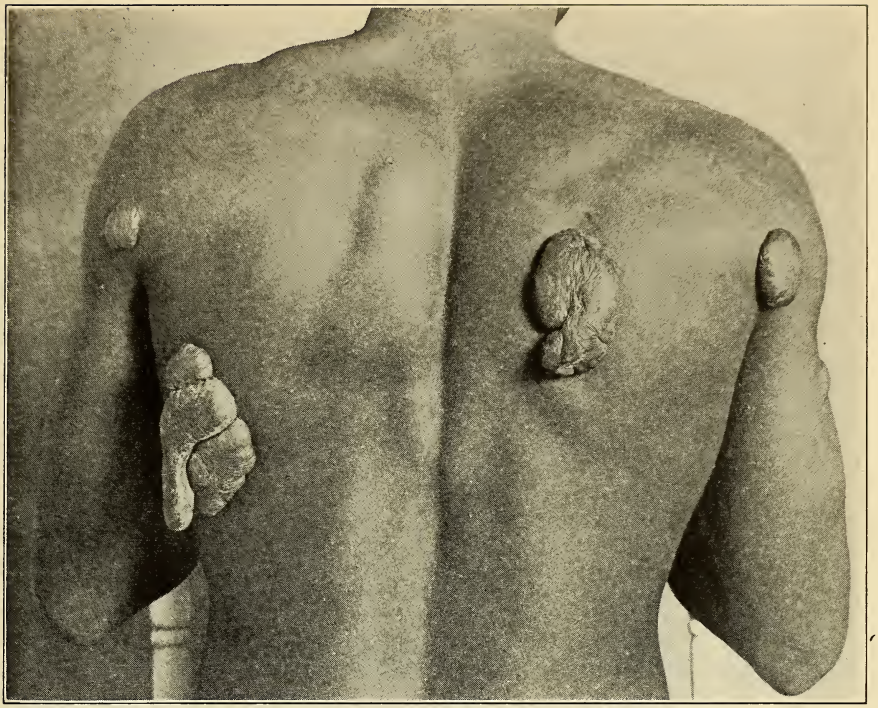

Fig. 38. KELOID

Keloids are spontaneous or secondary, the latter being closely allied to, if not identical with hypertrophic scars. Negroes present the lesions more commonly than whites. They are pink, red, yellow or flesh-colored, often dendrite, recur when excised, and structurally are identical with fibromata. Their commonest site is the thorax or near it.

feels rough and hard and is immovable in the skin, but moves with the skin over the tissues below. Cures cannot easily be effected. Small tumors may be excised. This is not possible, however, when the involvement is extensive. All other therapeutic measures are useless.

CHAPTER XVII

ATROPHIES OF THE CONNECTIVE TISSUE

Atrophies of the skin itself are usually due to a disturbance of the collagen leading to its ultimate thinning. Very frequently the elastic tissue also degenerates. From the clinical standpoint all diseases should be here included which terminate with thinning of the skin, except atrophic scars, and the atrophic stages of the erythrodermas and dermatitides. In the last mentioned the atrophy is not essential, nor frequently enough a part of the picture to justify its consideration under the atrophodermas. The commoner forms are:

A. The Scleroderma Group
    1. Scleroderma (Fig. 39)
    2. Morphoea (Guttate) (Fig. 40)
    3. Sclerodactyly
    4. Hemiatrophia facialis
    5. Ainhum
B. Idiopathic Atrophy Allied with Scleroderma
    1. Diffuse Idiopathic Atrophy
    2. Acrodermatitis Atrophicans
C. Macular Idiopathic Atrophy or Atrophia Maculosa et Striata
D. Senile or Regressive Atrophies
    1. Senile Atrophy and Atrophy Due to External Causes
    2. Kraurosis
    3. Striae et Maculae Distensae
E. Rare Forms
    1. Glossy Skin (possibly related to Scleroderma — A. 1)
    2. Multiple Benign Tumor-like Growths of the Skin (possibly related to Atrophia Maculosa — C).

SCLERODERMA (FIG. 39)

Synonyms. Hide-bound; Dermatosclerosis; Scleriasis; also Adult Sclerema; German, Sclerodermie; Hautsclerem; French, Sclérodermie.

Definition. Scleroderma is an induration of the skin followed by an atrophy. It is a chronic disease beginning insidiously, extending gradually, and leading to atrophy so slowly that a prolonged period is consumed in the entire evolution.

Symptoms. The disease begins with a thickening or even edema of the skin. The integument is firm, cannot be pinched or raised, and is dusky red or violaceous in color, resembling in consistency a mature erysipelas. Pain, burning, itching, parasthesia, neuralgias, malaise and even mild fever have been noted at this stage, which is known as the first, indurative or edematous period. Beginning at a given site it becomes diffuse, including at times the entire body; or, it may even start extensively. It progresses, and the moving margin is elevated, hard, and in sharp contrast to the neighboring normal areas. As already mentioned it looks like erysipelas, but feels waxy rather than brawny, and when edamatous may be pitted by pressure. But it differs from erysipelas in that the red surface is cool rather than hot.

After a certain period, if atrophy sets in, and it usually does, the skin grows thin, may remain red but often gets white, is cool, glossy, tense, and cracks or ulcerates over bony prominences. Often, in extreme cases, the entire body is encased in a stiff integument, limiting or entirely preventing the function of the joints, converting the face into a rigid mask, the mouth into an immobile orifice, and the hands into talons by retracting the fingers. This extensive type is, on the whole, uncommon. Frequently the disease remains limited to the legs, or hands, or face. Often the larynx is involved, and the voice is pitched to a metallic treble, or a mere whisper. The patient feels cold, the skin in places loses its gloss, scales, ulcerates, or becomes fissured and dry, and intercurrent metabolic or infectious disturbances cause death.

Course. There are acute or chronic, generalized and symmetrical, or asymmetrical localized varieties, determined by the course. The first two depend entirely upon the speed with which the disease evolves, and the course may be one extending over months or many years. The generalized form starts as such, or from a local primary area, and the localized types may remain so or become general. Arrest of the process is possible at any stage, and occasionally complete involution takes place.

Varieties. Besides the varieties determined by course and extent, as stated above, there are circumscribed scleroderma (morphoea or Addison's keloid) (Fig. 40), hemiatrophia facialis and ainhum.

The last two are rare. Circumscribed scleroderma takes the form of islands of disease, which are discrete, raised, circumscribed, and shaped as dots, lines or bands. This variety has a prodromal stage corresponding to the first period of the general type, followed by an atrophic stage. At first the lesions are pink or violaceous macules developing slowly or rapidly, and surrounded by a delicate violaceous ring. Later the lesions become yellow, buff, or white, the periphery remaining violaceous; and finally, atrophy, sometimes cicatricial in character, develops. The lesions may be single or numerous, symmetrical or not, flat or rough, ribbon-shaped, irregular, oval, or of any other form. Sometimes pigment surrounds the islands. As older lesions atrophy often new ones develop, the entire process covering many years.

A type developing on the legs in the atrophic stage is scaly, covered by patches of dermatitis, and the parchment-like skin is so transparent that the veins, which are often dilated, shine through and can be felt to be lying in a groove of hard, connective tissue. This form rarely extends above the knees, but is associated with acrodermatitis atrophicans of the thighs, and often other patches of circumscribed scleroderma are found elsewhere on the body.

Guttate Morphoea is possibly a variety of scleroderma, but it also may be a variety of sclerotic lichen, lichen albus, or lichen atrophicus. Hence, it is wiser not to describe it directly under scleroderma, but as a related condition. It is also called white spot disease and its most striking feature is its dead snowy or chalky whiteness. The lesions are usually seen on the chest, but may occur anywhere, are white, punctate, multiple, grouped, finally atrophic, and surrounded by a narrow brown, pink, red, or violaceous margin. They may be associated either with other lesions of scleroderma, or with lichen. Microscopically, too, they conform with either of these diseases.

Sclerodactylia is probably sclerodermatous, and it affects the feet more than the hands. It starts in childhood, and beginning with the smaller members, it gradually includes the entire foot or hand, legs or forearms, or all four extremities. It looks and acts like scleroderma, but at first may be hard to differentiate from Raynaud's disease, acro-asphyxia, perniones, or thrombophlebitis obliterans, particularly when necrosis or ulcerations develop. It is often associated with scleroderma elsewhere.

Hemiatrophia Facialis is scleroderma of one side of the face, which involves the deeper tissues. It is rare and usually accompanied by other evidences of scleroderma.

Ainhum is a rare disease restricted almost entirely to Ethiopians in their native surroundings. It is characterized by a constricting sclerodermatous band surrounding the base of the little toe. This acts as a permanent tourniquet, causing all the mechanical changes such an appliance would produce, and ultimately leading to spontaneous amputation.

Differential Diagnosis. The various forms of scleroderma are so characteristic that confusion with any other condition is almost impossible.

Etiology and Pathogenesis. Metabolic, emotional, nervous, and central nervous diseases, trophoneuroses and angioneuroses, have been considered the causes of the malady. We do not yet, as a matter of fact, understand the etiology. Its relative similarity to myxedema, the fact that thyroid medication has improved some cases, and pituitary extract others, as pointed out by *Johnston* (in the 1915 Session of the American Dermatological Association), indicates that probably, at least, some phases of the disease depend upon disturbance of glands of internal secretion. In one case of guttate morphoea I saw marked improvement with pituitary extract; in a generalized scleroderma none. The latter patient had a tremendous sugar tolerance. He was able to ingest three hundred grammes of pure sugar without getting alimentary glycosuria, although he developed a hyperglycemia of 0.2 within an hour. Three-quarters of all the cases occur in women. An effort has been made to regard syphilis as the cause of the illness, because in a certain number of cases the Wassermann reaction has been present. In the few cases in which I endeavored to confirm this I have failed, but had the Wassermann reaction actually been present, I think I should have interpreted the phenomenon as a case of scleroderma developing in a syphilitic, just as pneumonia or any other disease might have developed.

Treatment. Both the local treatment which should be symptomatic, and the general treatment with the usual alteratives employed have been unsuccessful. Until thyroid and pituitary gland extracts were used the disease appeared hopeless, and even with these newer remedies the outlook, though better, is none too good.

Prognosis. The prognosis as to cure is always bad. Often the disease does not progress and if this is the case the patient may live to a fair old age. This is even possible with the generalized forms, but in these the last years are spent in helplessness. Death is due to some intercurrent illness, or marasmus. The prospects in morphoea are invariably good so far as life is concerned.

All forms of cutaneous atrophy about to be described are un-

common, if not rare, and from the general physician's standpoint, unimportant. While the medical student should be familiar with the existence of these conditions, and with their commoner features, it is easy to exaggerate their significance for they are incurable. Failure to recognize them, although a major offence for an expert, would hardly be even a lesser crime for the general practitioner, and could in no sense be counted against the student.

Diffuse Idiopathic Atrophy or *Atrophia Cutis Diffusa Idiopathica* may begin anywhere, some say with infiltrations and others without. The original foci are small, but coalesce and advance over the entire trunk, finally leaving the skin translucent, thin, and of the consistence of cigarette paper. When pinched up the integument very gradually returns to its former place, not springing back as normal tissue would. It feels thin and as if no paniculus lay below. The vessels shine through and the lax tissue tends to be wrinkled or folded. The condition may be associated with one of the other forms of primary atrophy. There is no cure, but the patient rarely suffers gravely.

Acrodermatitis Atrophicans Chronica (Fig. 39) ends with the same type of atrophy seen in the diffuse variety. It begins with edema, infiltration and redness on the back of the hands and feet, the digits escaping. It progresses gradually, involving the extremities, and on the thigh reaches to just below Poupart's ligament, ascending laterally, however, to the level of the iliac crest, where it ceases to advance. Along the ulna and tibia inflamed, thickened bands appear resembling scleroderma. This disease is often associated with scleroderma of the legs.

These two conditions are with difficulty separated from scleroderma, since one, the second, often co-exists with scleroderma, and since both begin as inflammatory infiltrations which end as atrophies. The main difference between the two conditions is that in its final stage, the scleroderma of the skin tends to be hard, and the primarily atrophic skin soft.

Atrophia Maculosa et Striata Idiopathica. This malady may appear anywhere on the trunk or extremities. It begins as infiltrated, reddish, purplish, pale blue, hard or boggy nodules, which ultimately turn dead white, leaving atrophies which clinically resemble vaccination scars, and anatomically present the appearance of any cutaneous atrophy. At times the lesions are striated. The affected skin is wrinkled, thin and inelastic. This final state is known as anetoderma, a descriptive word applicable to the ultimate condition of all atrophies. On palpating such a lesion it feels like a depression, surrounded by a ring of normal paniculus. Thus, the examining finger detects a sensation of entering a shallow pit.

Senile Atrophy, Atrophia Senilis, or *Senile Atrophoderma* is due to elastic fibre degeneration. The skin is dull yellow, wrinkled, dry, impliable, and slightly scaling. It contains telangiectases, and is often covered with senile or seborrhoeic warts.

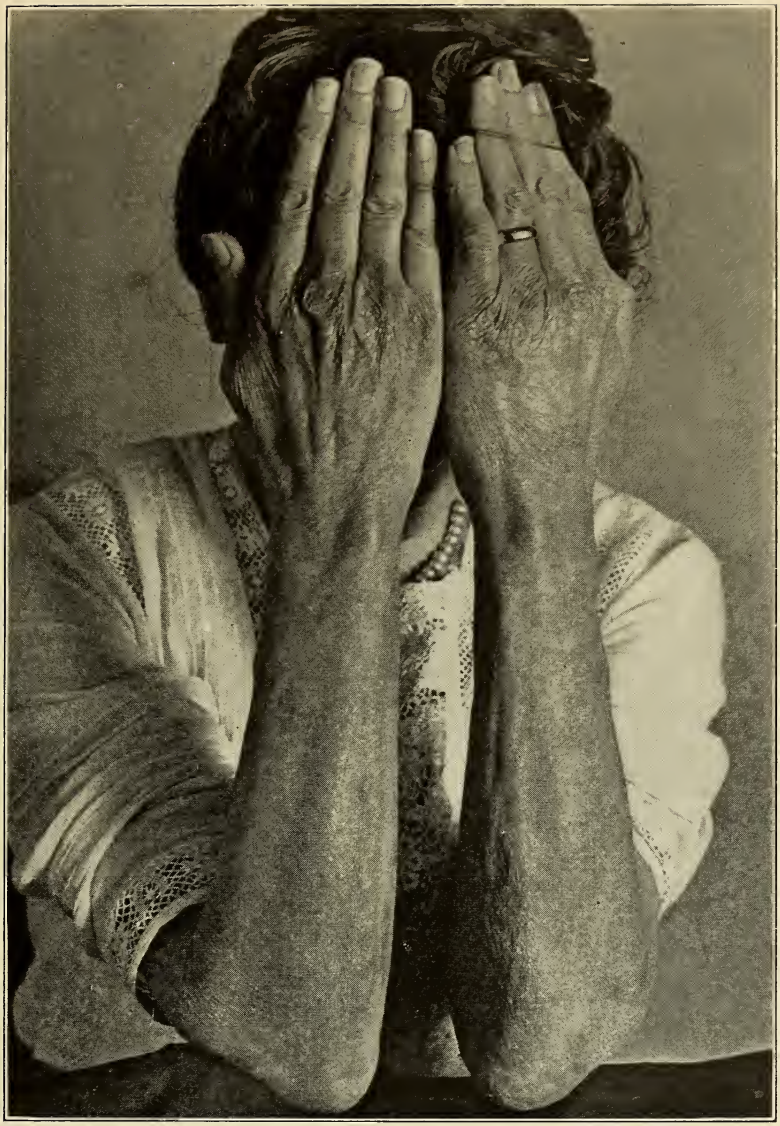

FIG. 39. SCLERODERMA WITH ACRODERMATITIS ATROPHICANS

The middle phalanges show scleroderma in the hide binding stage when the skin is tense, drawn, red or blue. The rest of the hands show the condition in the atrophic stage in which the skin is thin, puckered, lax and lustreless. This is called anetodermia and is seen in the terminal phases of the atrophies as a whole. It is also marked on the forearms.

FIG. 40. MORPHOEA

Morphoea is a localized scleroderma, and is also called guttate or macular scleroderma. It is at least closely related to white spot disease. Here the lesions are on the middle three knuckles, and are white, striated, because atrophic, and grouped to form patches. The small dark spots are freckles.

Kraurosis Vulvae is a rare atrophy appearing after the menopause, whether natural or induced. At first small, painful, red indurations appear about the introitus. Gradually, the tissue becomes thin, tense, glossy, and the process involves the neighboring regions. The labia and clitoris shrink and the normal contour of the vulva disappears. In the final stage all other atrophies are simulated.

Striae or *Maculae Distensae,* also called *Lineae Albicantes,* can best be illustrated by the common examples of the white lines developing on the abdomen and thighs during pregnancy. Similar lesions arise after any sort of abdominal distension, or in emaciation following adiposity. The condition is too well-known and of too little importance to merit more space.

Glossy Skin, or *Atrophoderma Neurotica,* first described by *Paget,* is a disease of the fingers. These become smooth, glossy, pink, or blotchy red, as if permanently frost-bitten, and the nails become convex in both diameters. At times vesicles, bullae and necroses are noted, and neuralgic pain is present in some cases upon motion. Trauma, injury, lepra, in short anything capable of causing a trophoneurosis, cause this disease, which tends to disappear spontaneously, if the underlying cause is curable.

Multiple Tumor-Like Growths of the Skin are small, bluish-white or slate colored lesions, the size of a dime. They puff out and are covered with fine, dilated vessels. On palpation, they feel like boggy masses with a soft base, surrounded by a firm ring of normal tissue. They end in atrophy of the skin and there is a strong suspicion that they are identical with atrophia maculosa.

CHAPTER XVIII

PIGMENTARY DISEASES

Disturbances of pigmentation occur in two great groups, in one of which an excess of coloring matter is deposited, and in the other of which the coloring matter disappears. The former are called hyperpigmentations, the latter depigmentations or leucodermata. They may be consecutive conditions, that is, following some pre-existing disease of which the pigmentary disturbance is an incidental sequel; or, they may be primary, that is, an essential manifestation of the condition in which they arise.

Depigmentation is always due to the absence of pigment cells, also called chromatophores or melanoblasts, or of the pigment granules in these cells, or of the pigment in the basal layer of the epidermis. Hyperpigmentation is due to either of the two following conditions, or sometimes to both. The first of these is an excess of the number of chromatophores, or of pigment granules in the chromatophores or basal layer, or all three. The second of these is the hyperpigmentation following hemorrhage into the skin. Such pigment is derived directly from hemoglobin. Pigment as formed in chromatophores is called melanin, and its derivation is not perfectly known. It is however under the control of the chromaffin system.

Besides color changes dependent upon disturbances of the pigment producing mechanism itself, there are some caused by the introduction from without of foreign particles, such as tattoo marks, or a deposit of gun powder in the skin. Certain animal parasites, notably pubic lice, cause bluish discolorations, the maculae ceruleae or taches bleuâtres. Absorption of metals, chiefly silver and arsenic, imparts abnormal tints to the skin, the former giving the well-known picture of argyria, the latter producing brown discolorations, while lead and bismuth are responsible for a blue line at the edge of the gums. In the manufacture of explosives, particularly melanite, according to *Rendu,* the skin on the exposed areas turns canary yellow.

Hyperpigmentation
- A. Derived from melanin
  - I. Idiopathic
    1. Chloasma
    2. Lentigo or ephelides
    3. Mongolian spots
    4. Naevi
  - II. Congenital predisposition often precipitated by external causes
    1. Lentigo or ephelides
    2. Pigmentation in xeroderma pigmentosum
    3. Naevi
  - III. Metabolic disturbances and infections
    1. Addison's disease
    2. Cachectic bronzing
    3. Chloasma (uterine and the like)
    4. Graves' disease
    5. Lepra, syphilis, tuberculosis, lichen planus
  - IV. Injuries, scars, etc. Radiodermatitis, weather beating, sunburn, pigmentation in scars
- B. Derived from Hemoglobin
  - I. After purpuras and other hemorrhagic diseases
  - II. Scars, particularly fresh scars, following injury, tuberculosis, syphilis, ulcers, etc.
  - III. After inflammations as dermatitis, body lice.
  - IV. Metabolic. Hemochromatosis, or bronzed diabetes.
- C. Derived from ingested, injected or absorbed drugs or chemicals, arsenic, silver, lead, bismuth
- D. Foreign pigments introduced from without
  - I. Tattooing or gun powder spots
  - II. Maculae ceruleae (pubic lice)
  - III. Blue pigmentation following hypodermatic injections

For the sake of brevity these relatively unimportant phenomena will not be treated in so detailed a manner as the more significant dermatoses.

Chloasma is idiopathic or symptomatic. Idiopathic chloasma is secondary pigmentation following any cause at all, and the word is synonymous with hyperpigmentation from an excess of melanin. Symptomatic chloasma is best illustrated by the uterine variety. This occurs as the result of a disturbance of the internal generative

organs,— pregnancy, ovarian disease, pelvic tumors, etc. It consists of a facial discoloration, yellow to deep brown in color. Such changes are physiological and transitory during gestation, when they are also present near the nipple and on the abdomen. (Linea nigra.)

Lentigo or *ephelides* are freckles.

Mongolian spots are possibly naevi in the widest meaning of the word. They are congenital blue, black, or purplish macules from one to ten centimeters large, and lying over the lower sacral regions. Usually they leave in the third or fourth year.

Naevi will be considered in a separate chapter.

Xeroderma pigmentosum is partly characterized by freckle-like hyperpigmentations. (Chapter XXIX.)

Addison's Disease may cause general or local pigmentation. It is seen as brown areas in the body folds, on or about the nipple, genitals, face, neck, and buccal mucosa. Any trauma causes hyperpigmentation. The general symptoms of asthenia are present, and the disease is generally due to tuberculosis of the suprarenal body. As the pigmentation progresses, the entire body becomes mahogany colored and the patient dies either of asthenia, or of general tuberculosis.

Bronzing is often present in any condition causing cachexia. This type is known as cachectic bronzing, and the underlying cause may be malignancy, or a chronic infection, or any other condition leading to marasmus.

Graves' Disease gives rise to freckles in large numbers on those areas in which such lesions would normally appear.

Lepra, syphilis and *tuberculosis* cause various types of hyperpigmentation.

The hyperpigmentation excited by *X-Rays* is determined by the extent and length of the exposure. It may take the form of freckles or general tanning, just as sunburn would.

The last condition mentioned is too well known to require detailed discussion.

Pigmentation in scars is derived either from hemoglobin, or is melanotic. The former is more frequently present in young, the latter in old scars. It is either in or about the cicatrix.

Purpuras and other hemorrhages in fading leave blood pigmentation for a time.

Lichen planus after fading leaves melanotic macules at the site of the vanished papules.

Some forms of dermatitis, dermatitis herpetiformis, prurigo,

herpes zoster, and body lice leave pigmented spots, frequently derived from hemoglobin, often, however, of melanotic origin.

Hemachromatosis is seen in cirrhosis of the liver and pancreas, and when associated with diabetes, the latter disease is characterized as diabète bronzé.

Arsenic causes diffuse or freckle-like pigmentation; silver, a diffuse ivory blue discoloration (argyria); lead and bismuth dark lines on the gums near the teeth.

Tattooing is well known by all.

Gunpowder, when explosions occur near the skin, is driven into the latter causing deposits which look like blue-black ink spots, just under the cuticle.

In pediculosis pubis bluish macules (maculae ceruleae) are seen chiefly on the abdomen.

After injections of morphine, particularly in adults, a rare condition of bluish discoloration of the skin is at times encountered.

The treatment of these stains is not satisfactory. In the types due to metabolic diseases, drugs or chemicals, amelioration of the underlying cause, or cessation of the drug, may suffice to cure the skin disturbance. All attempts to remove the spots are vain, as they recur. Therapy depends upon causing exfoliation of the cuticle. Thus, one to three percent. solutions of bichloride, or twenty-five percent. lotions of ammoniated mercury, or a solution such as Van Harlingen's may be employed.

℞ Hydrarg. Chlorid corrosiv. 4.0
Zinci Sulph.
Plumb Subacetat. .....aa 2.0
Aq. dest. qs. ad. 120.0
Sig. Apply twice daily.

Tattooing may be partially removed by causing inflammation with thirty parts of zinc chloride in forty of water. This causes an eschar to form, and when the latter falls off the decoration is gone. There is danger, however, of excessive destruction of tissue. Perhaps the safest way of producing good cosmetic results is by conservatism and concealment. Liquid powders to be applied as needed are the best, as follows:

℞ Zinc oxide
Mag. ustae .....aa 15.0
Glycerin 10.0
Extr. violet fluid 0.6
Alcohol qs. ad. 150.0
Sig. Shake well and apply as needed.

This fluid is to be thoroughly shaken and after it has dried upon the skin the excess may be removed with a soft, linen cloth. If a pink rather than white surface is desired, five parts of powdered calamine may be incorporated in the lotion.

DEPIGMENTATION, OR PIGMENT ATROPHY

Disappearance of pigment is due to a variety of conditions, but the ultimate cause has not yet been explained. The varieties may be classified as follows:

I. Idiopathic
  1. Congenital
    a. Albinism
    b. Naevus anemicus
  2. Acquired vitiligo
II. Metabolic and Infectious in Origin
  1. Leucoderma syphiliticum
  2. Leucoderma psoriaticum
  3. White Spot Disease
    a. Morphoea
    b. Lichen albus, sclerosus and morphoeicus
  4. Skin atrophies (other than those included in 3 a–b)
III. Injuries; Depigmented scars, lineae distensae, leucoderma from chrysarobin
IV. Degenerations; colloid milium and degeneration

Vitiligo, called achromia, piebald skin, and also having other less important synonyms, is a white macular eruption with borders of hyperpigmentation about the colorless islands. These islands are circular, oval, irregular in outline, striated or mixed in type. They are clear white, numerous or sparse, flush with the skin and in no wise changed from the norm, grossly or microscopically, except that they lack pigment. Hairs upon such patches may or may not be white. The lesions may coalesce and simulate albinism. They are found anywhere on the body, but chiefly on the face, neck, extremities, genitals and trunk. In summer, the spots become pink because they lack the protection that pigment furnishes against the sun's rays.

The appearance of a patient with vitiligo is characteristic. Snow white patches of various size and shape, as described above, fleck the skin, and they are surrounded by a zone of apparently deeper pigmentation. At first a few spots appear and gradually more develop.

In summer the tanning of the normal skin emphasizes the contrasts. No subjective symptoms are present, but often the patient becoming self-conscious grows depressed, or even melancholy. Nothing is known of the etiology of the disease and all treatment is in vain.

Albinism, which may be partial or complete, is due to a congenital absence of pigment, often including the choroid. The skin is milky or pinkish-white, the hair is almost totally white, the iris is transparent and may look pink, and recovery is impossible. Because of the ocular condition mild photophobia and pupillary instability are present. In some instances heredity plays a causative rôle.

Naevus anemicus will be discussed among the naevi. (Chapter XXXII.) *Leucoderma syphiliticum* will be described in connection with syphilis. (Chapter XLII.) *Leucoderma psoriaticum* is rare. At times after the disappearance of psoriasis there is a permanent absence of pigment. *Morphoea* (Chapter XVII) and *Lichen albus, sclerosus* or *morphoeicus* (Chapter XIII) have been described. Many of the skin atrophies (Chapter XVII), including lupus erythematosus (Chapter XXV), are pure white in the final stages.

Scars may be depigmented as well as over pigmented. Lineae distensae or albicantes, observed on the abdomen and thighs in pregnancy, or in connection with abdominal tumors, ascites, *et cetera,* are white lines. These are atrophies secondary to elastic tissue rupture, and rarification of the collagen, incidental to the unusual strain on the skin in abdominal distension. *Leucoderma* at times follows the use of chrysarobin. Colloid degeneration (Chapter XIX) is not a true depigmentation. The conditions enumerated in this paragraph are not serious, but are intractable.

CHAPTER XIX

DEGENERATION OF AND IN THE CONNECTIVE TISSUE

Of this group of diseases xanthoma, xanthelasma and xanthoma diabeticorum are important, pseudoxanthoma elasticum is of interest, while colloid degeneration, calcification and dermatolysis are of minor significance. All represent some alteration either in the collagen or elastic tissue. Xanthelasma, the old xanthoma planum, is a muscular degeneration, and dermatolysis is either an elastic tissue disturbance, or a myxomatous degeneration. The latter is closely related to neurofibroma, if not identical therewith and it is a question whether it is a new growth, or whether neurofibroma, though apparently a neoplasm, is not actually also a degeneration. Arguments favoring either view might be advanced, but in this work neurofibroma will be included among the benign connective tissue neoplasms, according to classical usage.

A great deal of confusion has existed in the past regarding the relation of xanthelasma, or xanthoma planum to xanthoma tuberosum. They are, however, both clinically and anatomically separate conditions, as will appear below, and it is mainly due to *Pollitzer's* convincing studies that they have been separated. To-day there is no justification for any confusion, and the fact that their separation has not yet been universally accepted indicates only inertia on the part of students and inability or indisposition to admit the obvious.

XANTHOMA TUBEROSUM (FIG. 41)

Synonyms. Xanthoma; Xanthoma Multiplex.

Definition. Xanthoma tuberosum is a disease characterized by the formation of patches, clusters or stripes of yellow nodules, occurring on the extensor surfaces of the large joints.

Symptoms. At first minute papules with yellow or orange elements arise. These increase in size and number forming plaques (Fig. 41), striae, nodules, or small sessile or pedunculated tumors. The lesions are discrete or coalesced, and as new ones appear among the old, great variations in size, shape and color are seen. They may be barely visible, or as large as a pecan nut, pink, yellow, or

orange, according to the proportion of vascular dilatation or lipochrome present, and in consistency they are hard or soft according to the relative proportion of fibrous connective tissue, or fatty substance. At times single lesions may exist. In general the disease is not confined to the extensor areas, but spreads, although not densely, to other parts of the body, involving exceptionally the cornea, conjunctiva, respiratory passages, alimentary tract, serous lining of the body cavities, and even the solid viscera, large vessels and some tendons.

Course. The disease usually begins in early adult life, but has been found in children, and I recall one case in a child under two years of age. As a rule the condition does not shorten life, although the lesions grow progressively and steadily more numerous.

Varieties. There is but one form of the disease. Sometimes xanthelasma is seen in patients with xanthoma. The connection is purely a matter of coincidence.

Differential Diagnosis. The histology of the condition is distinctive. A fibrous connective tissue mass, including an inflammatory infiltration, xanthoma cells, lymphocytes and giant cells in which droplets of fat are found, compose the picture. A knowledge of these facts is of diagnostic importance. The disease must be differentiated from xanthoma diabeticorum and urticaria pigmentosa (Fig. 4), also called xanthelasmoidea. It can be distinguished from the former by determining the presence of diabetes. From xanthelasmoidea it is differentiated by the fact that it occurs prevailingly in adults, while urticaria pigmentosa always begins in infancy. Xanthoma lesions are tumor-like or nodular, while xanthelasmoidea is always macular and when rubbed becomes urticarial. Finally, xanthoma tuberosum has a distinctive microscopic structure, as noted above, while the anatomy of urticaria pigmentosa is equally distinctive, the lesions consisting entirely of mast cells, none of the characteristics of xanthoma being present.

Etiology and Pathogenesis. The etiology is not clear. It is a disease slightly favoring females, and a few instances of heredity have been noted. It shares with all other dermatoses occasional association with common systemic diseases. There is no reason, however, to believe that it is caused by them any more than a primary epithelioma of the lip would be due to a co-existing gout, or cirrhosis of the liver. It is beginning to be understood, though, that the malady depends upon disturbed lipoid metabolism. In effect the lesions are connective tissue hyperplasias developing about

cholesterol-fatty acid esters extravasated into the cutis, as *Pollitzer* and *Wile* pointed out. The extravasation is referable to cholesterinaemia, and experimental corroboration of this fact has been furnished by *Lebedew,* who fed cholesterin to rabbits causing an alimentary cholesterinaemia, and producing typical lesions of xanthoma tuberosum at sites injured by a seton. These observations also elucidate the fact of the greater frequency of the condition in women who commonly have cholesterinaemia during pregnancy.

Treatment. Excision offers the only hope of cure.

Prognosis. The outlook for a cure is poor, as it is nearly impossible to remove the hundreds of tumors covering the body. Aside from its annoyance to the patient, the illness is harmless.

Xanthelasma (Fig. 42) (*Pollitzer*) is a disease of the eyelids. Other similar lesions on the face and neck are probably flat forms of xanthoma tuberosum, although those of the neck may be true xanthelasma as will be pointed out below. The lesions are either tiny or as large as the lids, neither raised nor depressed to the touch, although they often look elevated. They are more frequent on the upper lid, near the inner canthus, and may be single or multiple. In color, they are chrome, or orange, or tawny. They feel like normal skin, are sharply marginated, and if one looks away while palpitating it is impossible to tell when one crosses the lesion. They appear in middle life and their cause is unknown. In no sense are they neoplasms, and as *Pollitzer* has demonstrated (*Jour. Cutan. Dis., Vol. XXVIII, P.* 633) they are due to a fatty degeneration of the fibers of the orbicularis palpebrarum. Those on the neck are probably due to a similar process in the platysma. The best therapy is electrolysis as applied for naevi, and although the lesions never disappear spontaneously they are always curable by proper treatment. There is no connection clinically, etiologically, or anatomically between xanthelasma and xanthoma tuberosum. Their only common feature is their color. If they both exist in one person, it is purely coincidence, just as a person with cancer of the stomach may have a rodent ulcer on the face.

Xanthoma diabeticorum is a rare condition associated with diabetes. The eruption appears suddenly and, when the cause is appropriately treated, vanishes promptly leaving no trace. Clinically, there is an extensive outbreak of small conical or pointed papules with red bases and yellowish apices. The entire body is usually covered, including the visible mucous surfaces.

Pseudoxanthoma elasticum, among the rarest of skin diseases, is usually

FIG. 41. XANTHOMA TUBEROSUM

This is a papulo-nodular disease. The extensor surfaces are favored but lesions may occur anywhere, and they vary in size as indicated here. There often are hundreds of lesions on the body. Over the elbows, plaques may form. The color is canary yellow, yellowish red, orange or even purplish brown, and the surface is waxy or glistening.

FIG. 42. XANTHELASMA

This condition was formerly called xanthoma planum. The eyelids are affected. In color, the lesions are yellow, orange or brown. They feel soft and are due to a fatty degeneration of the fibres of the orbicular muscles.

found upon the trunk, near the axillae, thighs or elbows. It may be seen elsewhere, and *Kingsbury* presented a case at the American Dermatological Association in 1915, in which the lesions were present on the neck. The lesions are yellow or orange and consist of flat papules, as small as pinheads, or by fusion forming a reticular patch, sometimes four or five inches in its long diameter. Its essential microscopic feature is a disturbance of the elastic fibers which are fragmented, curled, swollen, and often basophilic, the normal fibers taking only acid dyes.

Colloid degeneration, hyaloma, or *colloid millum* is rare. The lesions are tiny flat elevations, yellowish or white in color, and range from bare perceptibility to an eighth of an inch in diameter. They glisten and contain a jelly-like substance which exudes when the lesion is pricked. They are generally found on that portion of the face above the level of the mouth, chiefly on the forehead and near the eyes. The lesions may be cured by curettage or electrolysis.

Calcification is extremely rare. Calcareous deposits form in the contents of sebaceous glands which have become minute retention cysts. The lesions are dry and white. Calcification has also been known to have occurred in inflammatory areas, notably in tuberculosis.

Dermatolysis occurs in two forms — a general laxness of the skin, and a type resembling Recklinghausen's Disease. Cutis laxa resembles the skin in kittens or puppies. It is capable of inordinate stretching and promptly snaps back into place. It is due to redundancy of the elastic tissue. The type of dermatolysis resembling neurofibromatosis is a myxomatous condition characterized by pigmentation and the presence of single or grouped papules, or small tumors resembling fibromata. This type was recently described by *Wise* and I question whether it was not actually an instance of Recklinghausen's Disease.

SECTION C. PARASITIC DISEASES AND INFECTIONS

Infections of the skin are manifold. The pathogenic agents may belong to the animal or vegetable kingdoms. The animal parasites may be either protozoa, or members of the higher orders, while vegetable parasites are either bacteria or fungi. Properly speaking, since syphilis is caused by a protozoon, it belongs among the diseases to be described in this section. Because of its importance, however, it has seemed wiser to devote a separate and entire section to this malady. (Section F.)

GROUP 6. INFECTIONS

CHAPTER XX

DISEASES CAUSED BY MULTICELLULAR ANIMAL PARASITES

Animals of several phyla are pathogenic to the skin. They act in a transitory manner as jelly fish, bees, wasps, mosquitoes; or cause veritable infections as scabies, grain itch, or pediculosis. In order to present concisely the range and character of these pathogenic agents, the following somewhat pedantic table is inserted.

Phylum	Class		Example
Coelenterates	Scyphozoa		Aurelia Aurea (jelly fish)
Plathelminthes	Cestodes		Taenia Echinococcus
Nematelminthes	Nematodes		Oxyuris vermicularis Dracunculus medinensis Filaria medinensis Filaria nocturna Trichina spiralis Ankylostoma duodenalis (Uncinaria americana)
Arthropoda	Insecta	Order Aptera	Cimex lectularius
		Order Hemiptera	Pediculi
		Order Diptera	Pulex irritans Culex anopheles Gastrophilus equi Porthesia chrysorea
		Order Lepidoptera	
		Order Hymenoptera	Apis mellifica Vespa Formix
	Arachnida	Order Acarida	Pediculoides ventricosus Tyroglyphus longior Ixodes (several kinds) Acarus (sarcoptis) Scabiei Demodex follicularum Leptus autumnalis
Numulata			Hirudo Medicinalis

Common Name	Lesions
Jelly Fish	Urticarial lesions
Echinococcus	Urticaria; skin cysts
Thread Worm	Dermatitis of the perineum
Guinea Worm	Dermatitis
Filariasis	Elephantiasis of leg
Trichina	Edema; urticaria
Hookworm	Dermatitis feet and ankles
Bedbug	Petichiae, wheals
Lice	Dermatitis, papulo vesicular. Pigmentation
Fleas	Petichiae
Gnats	Wheals
Mosquitoes	Wheals; stings
Botfly	Larva Migrans. (There is doubt at present as to whether this disease is caused by the named organism or some unidentified one.)
Brown Tailed Moth	Dermatitis venenata
Bees	Wheals, edema, petichiae
Wasps	Large urticarial or edematous swellings, petichiae
Ants	Small wheals
Mites	Grain Itch
Mites	Copra Itch
Ticks	Dermatitis
Itch Mite	Dermatitis or Itch
Demodex	Causes no lesions — resides in pilosebaceous follicles
Leech	Bite with hemorrhagic centre

The common important disease in this group are scabies and pediculosis. The common unimportant conditions herein included are the lesions of stinging insects. The rarer important diseases are larva migrans, the dermatitis of the ankylostoma larva, the urticaria of taenia echinococcus, the edema of trichiniasis, and the elephantiasis caused by the nocturnal filaria. The remaining conditions are either rare or unimportant, or not encountered in the ordinary routine of American life.

The main lesions caused by animals are catarrhal, urticarial or hemorrhagic. Thus, the pictures of dermatitis, hives, or purpura are those most frequently encountered. Consecutive lesions, scratches, scales, crusts and hyperpigmentation are almost invariably present in connection with the exudative and urticarial conditions, although hyperpigmentation is confined largely to the diseases caused by lice and mites. Some of the organisms attack the skin from

without, as bees, mosquitoes, ants, jelly fish and leeches. Others burrow into and live within the epiderm, as in scabies or larva migrans. Still others live partly in the skin, by digging their heads into the papillary body, as do ticks which nourish themselves by sucking blood. Still others reside within the body, causing incidental cutaneous manifestations, as the urticara or edema in echinococcus or trichiniasis infection; while, lastly, others dwell on the surface of the skin, as pediculi.

SCABIES (FIG. 43)

Synonyms. The Itch; Seven Years' Itch. French, La Gale; German, Krätze.

Definition. Scabies is a contagious skin disease characterized by a dermatitis which itches most intensely at night, and which is caused by a member of the spider family, the acarus scabiei, the female of which burrows into the epidermis.

Symptoms. The patient presents himself complaining of an intensely itching eruption, the pruritus being worse at night, and often severe enough to disturb or even prevent sleep. An eruption is found involving chiefly the body folds, notably about the genitalia, in the axillae, umbilicus, between the nates, near the nipples, between the breasts and chest, and above all in the interdigital spaces of the hands, on the wrists, and to a lesser extent the rest of the body from the clavicles to the knees. Involvement of the face and of the legs below the knees is most exceptional save in babies, and palmar involvement is extremely rare, but found occasionally in women and babies.

The lesions themselves are numerous. In the first place, marking the site of the burrows, are small elevated lines, a millimeter or more in length, at one end of which rise tiny vesicles. In cleanly individuals these lesions are yellowish or colorless. In ill kept people, however, they are gray or black, due to the presence of dirt. Scratching causes the formation of crusts, minute hemorrhages, papules, even lichenification, and when infection takes place an impetigo develops. Rarely one sees suppurative paronychia. This occurs more frequently in young children than in adults and in the former impetigo of the face may develop, resulting from bacterial inoculation by impetiginous scabies lesions, but not from the transfer of the itch mite to the face.

At times in well cared for people the cases are atypical, for only a few lesions develop, but these itch intensely at some or all of the

usual sites. The consecutive lesions are usually lacking. On the other hand, in uncleanly people, the process may go on for so long that the combination of filth, crusting, healing up of scales, the number of scratch marks, infections, et cetera, cause an exaggeration of the common picture.

Course. Untreated, the condition never heals. Proper treatment limits the disease at once. It has been stated that an intercurrent fever, such as typhoid, cures the outbreak. It would seem much more likely that the care of the very sick patient, the bathing and the like, cure the skin disease rather than that it is cured by the influence of the fever.

Varieties. A severe form, known as Norwegian scabies, was first recognized in Scandinavian lands. It is simply an exaggerated variety of the disease in people of subnormal sensibility. Thus, in anesthetic lepers, and perhaps in patients suffering with syringomyelia, the condition may be encountered.

Differential Diagnosis. The nocturnal itching and typical localization, the demonstration of the mites secured from the burrows, are diagnostic features of the disease. Dermatitis hiemalis, prurigo (Fig. 5) and vesicular dermatitis (Fig. 11) resemble scabies. The history of seasonable eruptions in the first of these, the persistence of the second, the fact that the third itches as much by day as by night, the absence of the acari, and the circumstance that in scabies usually several members of the family are ill, constitute the essential features to be considered.

Etiology and Pathogenesis. The cause of the disease is the acarus scabiei. The female after impregnation burrows into the epiderm, depositing one or two eggs a day. In from ten to twenty days she dies, but the young are hatched out in about ten days and escape to the surface of the skin where, after a brief metamorphosis, impregnation occurs, the females burrowing, the males dying. Besides the human variety of acari, there are others infecting various wild and domestic animals, both birds and mammals. These are pathogenic to man also. The disease provokes an eosinophilia.

Treatment. There are many methods of treatment, but only one which need be remembered, and for this but three substances need be employed,— sulphur, balsam of Peru and styrax. Any one of these drugs is used in ten percent. strength in some ointment base. Cold cream is the pleasantest. An ounce of this salve is rubbed in vigorously on four successive nights over the entire body from the clavicles to the shins, with special reference to the various body folds

and interdigital spaces. A long, hot, soap bath precedes the first and fourth inunctions. Exactly ten days after commencing the first course of treatment, a second identical course is begun and carried out punctiliously, whether the patient seems well or not. The purpose of this is to forestall a recurrence which may be started by a few surviving ova in the burrows. The balsam of Peru ointment is preferable, as the sulphur often causes dermatitis. Should dermatitis appear, it must be treated as outlined in the chapter on this disease. Usually, marked relief is noted after the first inunction, and complete relief after the third. At times, more than two courses of rubbings are required. Since a few of the acari may have found their way into the patient's bed, it is wise to dust sulphur powder on the mattresses, and to use fresh sheets when the first course of treatment is finished.

Prognosis. This is always good. It is rare to encounter a case which cannot be cured with three courses of rubbings with balsam of Peru salve, and it is indeed most exceptional that more than two such courses should be needed.

PEDICULOSIS

Synonyms. Phthiriasis; Lousiness; French, Phtiriase; La Pediculosis. German, Pediculose; Läusesucht.

Definition. Pediculosis is a common disease caused by the hemipterous insects called pediculi or lice. These insects are of three well-known varieties, afflicting the scalp, pubis and body.

Symptoms. Constant itching, certain inflammatory and infectious manifestations, and the presence of lice and their ova characterize the clinical picture which varies, however, according to the type and site of the infection. These features in turn are determined by the variety of the louse.

Course. Unless properly treated, the disease would go on forever.

Varieties. These, with the detailed symptomatology, diagnosis and treatment, will be considered as independent diseases.

PEDICULOSIS CAPITIS

Synonyms. Head Lousiness.

Symptoms. The scalp and beard are affected. Aside from the presence of the lice and ova, the skin may exhibit dermatitis, impetigo or both, and scratch marks, crusts, scales, seborrhoea and a nauseating odor participate in the symptom complex. The occipital and post auricular areas are most densely involved, the lice scurrying

in and out among the hairs. To the shafts of the hair, particularly behind the ears, are attached the ova. They are white or yellowish and project from the hair, as do the tufts from the stem of the pussy willow. They can be removed only with great effort, because they are glued on with a stout cement. The post cervical glands, particularly in children and weak people, are enlarged and often suppurate causing systemic symptoms. Often a mild dermatitis of the face, neck and shoulders develops, and even conjunctivitis, although the head louse never actually affects the lashes. Itching is severe and scratching constant. Thus, impetigo (Fig. 56) of the face may arise. In extreme cases the hair becomes matted with crusts and serum forming an inextricable tangle, the so-called plica polonica or Weichselzopf of the Germans.

Diagnosis. In the cleanly this may be difficult, but a careful search for nits should be made, however unlikely it seems that a given individual may have the disease. A mild persistent dermatitis of the face, the presence of enlarged cervical or post auricular glands, are very suspicious. In children, most cases of facial impetigo are due to lice or nits. At times in adults the former may be sparse and the latter numerous.

Etiology and Pathogenesis. The immediate cause of the disease is the head louse or pediculus capitis. The mode of transmission is by contact with infected people or objects. Children in school, and children and women working in shops and factories are often infected by companions. At times nurses transmit the disease to their charges. Sleeping in public beds, as in hotels, sleeping cars and on vessels are mediate causes of the disease. A common mode of transmission is by the indiscriminate use of combs and brushes, and by the trying on of hats at milliners', even those located in ultra-fashionable shopping districts.

Treatment. It is unnecessary to use the traditional petroleum or delphineum. A ten percent. solution of balsam of Peru in alcohol will kill the lice. It should be applied thickly and after twenty-four hours a shampoo is necessary. All of the living organisms and the contents of the majority of the ova will be destroyed. Should a dermatitis or impetigo of the scalp or face be present, a five to ten percent. ammoniated mercury cream applied once daily will effect a cure. The best way to remove the nits in little boys is to cut off the hair. In girl babies this is also feasible. In older girls and adults the hair should be washed off with equal parts of vinegar and hot water, and while still wet should be fine combed. It is wise to con-

tinue this once a week for three months, as occasionally a viable ovum may be overlooked.

PEDICULOSIS CORPORIS (FIG. 44)

Synonyms. Pediculosis vestimenti; Body Lice; Phthiriasis.

Definition. Pediculosis corpis is a polymorphous irritative disease caused by lice which inhabit the seams of underclothes and travel thence, temporarily to thrive upon the host whose blood they suck.

Symptoms. The lice suck the blood from the follicles. They provoke itching and thus the host scratches, leaving characteristic excoriations, linear, parallel (Fig. 44), crusted, and usually found where the clothes press against the skin. Hence, the regions most affected are the scapular areas and the waist. Here in great number, and elsewhere in smaller number are seen the above mentioned excoriations. Papulo-vesicular dermatitis which is often crusted, pigmented and impetiginous completes the picture. The pigmentation at times is as deep as that of Addison's Disease. In neglected cases the skin becomes dark and lichenified, constituting the so-called vagabond's disease. Secondary infections and glandular enlargements are frequently seen. Obviously, the patients complain of intense itching and when undressed constantly scratch. The ova are deposited in the fibres of the clothing.

Course. The disease lasts as long as the cause remains.

Differential Diagnosis. Prurigo (Fig. 5) and chronic vesicular dermatitis are simulated, but the class of patients usually affected and the characteristic excoriations usually indicate the real nature of the disease. The lice are found in the seams of the underclothes, particularly woolen ones. The nits are attached to the filaments of the material.

Etiology and Pathogenesis. The immediate cause is the pediculus vestimenti or corporis. Filth and crowded surroundings favor infection. This organism is the one now famous in military lore as the "cootie." In the past few years its nature and significance have become better understood than formerly. This is due to the fact that it transmits the organisms of typhus and trench fever. The body louse can survive neither in very high nor in very low temperature. Thus when patients develop a febrile disease, the lice desert the host. In very cold weather their activity is diminished, particularly as to reproduction.

At times the body louse can infest the scalp, and it is supposed by

some writers that the body and head louse are identical. This is probably not true as the typical body louse is larger and darker in color than is the variety infecting the scalp.

Treatment. The underclothes should be boiled and the outer garments subjected to dry heat. A ten percent. balsam of Peru ointment should be applied to the skin for three or four nights.

The management of large numbers of infested people, a problem recently developed in our armies, has taxed the ingenuity of sanitary experts. It is simple enough to rid the the body of lice, but the task of delousing clothes is rather puzzling, for dry heat does not penetrate and moist heat shrinks the cheap fabrics with which the world was flooded during the war. Major Harry Plotz devised an elaborate delousing plant which in all respects met requirements, save that the soldiers complained that the moist heat wrinkled their uniforms.

Prognosis. This is always good.

PEDICULOSIS PUBIS

Synonyms. Crabs; French, Morpion.

Definition. This is a parasitic disease caused by the pubic louse which attacks all of the body hair and eyelashes, but which usually remains restricted to the pubic region.

Symptoms. Severe itching and the presence of the lice and ova constitute the picture of the disease. The organisms and nits are found in the pubic, axillary and other body hairs and eyelashes. The nits cling to the hairs as do those of head lice. Evidence of inflammation and pruritus are present.

Course. The disease ends only with the removal of the cause.

Varieties. Phthiriasis palpebrarum, due to the presence of the lice in the eyelashes, causes blepharitis, the pediculi burying their heads in the follicles.

Maculae ceruleae, or taches bleuâtres, are slate gray or inky macules of a diameter not exceeding one and one-half centimeters. They are found on the chest, thighs, belly and arms, chiefly in blonds, and are pathognomonic of pediculosis pubis.

Differential Diagnosis. The presence of the lice, ova and blue spots differentiate the disease from ordinary dermatitis.

Etiology and Pathogenesis. The immediate cause is the pubic louse (Fig. 45) which is usually transmitted through intimacy, although the malady can be acquired by contact with infected bedding or clothing.

Treatment. Mercurial salves and particularly gray ointment, and shaving off the affected hairs are to be urgently condemned. The former causes dermatitis, the latter more irritation than the disease. Balsam of Peru as employed in other types of pediculosis will effect a cure within four days. A one percent. yellow oxide of mercury cream is to be employed for eyelash involvement.

Prognosis. The prognosis is good.

STINGS

Flies, mosquitoes, ants, gnats, black flies, bees, wasps, bed bugs and fleas all cause well known lesions. The less severe ones need no treatment. Others, particularly those of bees and wasps, will respond to wet dressings. At times the lesions become infected whereupon the treatment must be adapted to the secondary condition. Bed bug and flea bites are characterized by central hemorrhagic points resembling petichiae. The eruptions of purpura hemorrhagica and petichial sepsis are simulated, but the perfect health of the patient excludes these conditions.

Larva Migrans. *Synonyms.* Creeping Eruption; Dermamyiasis Linearis, Migrans Aestrosa. This rare illness is characterized by the progressive extension of a linear lesion, somewhat raised and wheal-like. It advances as much as an inch in twenty-four hours. The moving point is bright red and urticarial. The older portion of the track is pink. Straight, curved and tortuous elements are present, and often the burrow doubles on itself. The organism has rarely been seen, but *Knowles* succeeded in demonstrating it in its burrow. Its nature is still undetermined. Although there is no substantiation of the fact that it is the larva of the bot fly of the genus gastrophilus, this view has not been entirely abandoned. Slight itching characterizes the lesions of which as many as thirty have been present at once. Treatment is difficult. Even excision of the entire burrow often fails. Sand papering the advancing area and rubbing in antiseptic ointments, as suggested by *Whitehouse,* appears to possess some value. Among our soldiers, after the recent Mexican campaign, several cases developed.

Ground Itch. *Synonyms.* Uncinarial Dermatitis. Uncinarial dermatitis is caused by the larva of the ankylostoma duodenale. It occurs prevailingly on the feet, and begins as an itching dermatitis with vesicles, bullae and papules which become secondarily infected. These lesions are the portals of infection of the hook-worm. The larvae travel from the affected sites to the duodenum, there to lodge and produce the symptoms of ankylostomiasis. The local condition is best treated by mild antiseptic fomentations and salves. The general condition requires the use of male fern, eucalyptol or beta naphthol internally.

Echinococcus. Echinococcus cysts rarely occur in the skin. They may suppurate or calcify and give the signs of any other cystic tumor. Rup-

tured visceral cysts produce urticaria. This condition also sometimes follows aspiration.

In this connection it may also be mentioned that cysticerci of the taenia solium may develop in the skin. They feel like any other nodular lesion and are recognizable only under the microscope by finding the scolex in the cyst contents.

Trichina causes edema of the skin, particularly near the eyes. Hyperhydrosis, tingling, pruritus and urticaria are also present. The myalgia and eosinophilia are corroborative factors in reaching a diagnosis.

Filaria sanguinis hominis is introduced by the culex fatigans. Elephantiasis develops due to a choking up of the lymphatics with enormous numbers of worms. Another variety of filaria, the filaria medinensis, dran-cunculus or Guinea or medina worm, causes papular, nodular, furuncular or ulcerative lesions. These lesions appear near the ankle or foot and more rarely on other parts of the body. They develop where the mature worm endeavors to emerge. Near the lesion, extending radially thence, may be felt the body of the worm under the skin. It feels like a soft, raised, elongated mass, three or four inches long by one-eighth inch broad. Before rupturing the lesions are tense. In endeavoring to withdraw the worm, it sometimes breaks and lymphangitis, or even sepsis develops.

Oxyuris vermicularis, or thread worm, inhabits the lower bowel. At times parasites looking like threads escape to the perineum setting up a local inflammation or dermatitis. Weak bichloride enemas, one to ten thousand, cure the internal condition and ammoniated mercury ointment the external.

The Aurelia Aurea or common jelly fish of our Atlantic shores causes urticarial lesions.

Pediculoides ventricosus causes grain itch, or acarodermatitis urticarioides. This disease was studied by *Schamberg* and *Goldberger* in an epidemic in Philadelphia. The lesions caused are urticarial, papular and vesicular and there are consecutive lesions due to scratching, for the pruritus is intense. The arachnida inhabit the straw in cheap mattresses.

Ixodes or ticks are numerous in their varieties. They insert their beaks into the skin and suck blood. They may best be removed by dropping a little turpentine or benzine on the exposed part of the parasite. This causes the head to be withdrawn.

Copra Itch produced by the tyroglyphus longior causes a dermatitis resembling grain itch. *Demodex folliculorum* is a harmless mite inhabiting the sebaceous follicles, chiefly near the nose. *Leeches* attack human beings bathing in fresh water. They are also rarely employed in medicine. The bite is purple, about the size of a dime, and has a red centre at the site of the puncture.

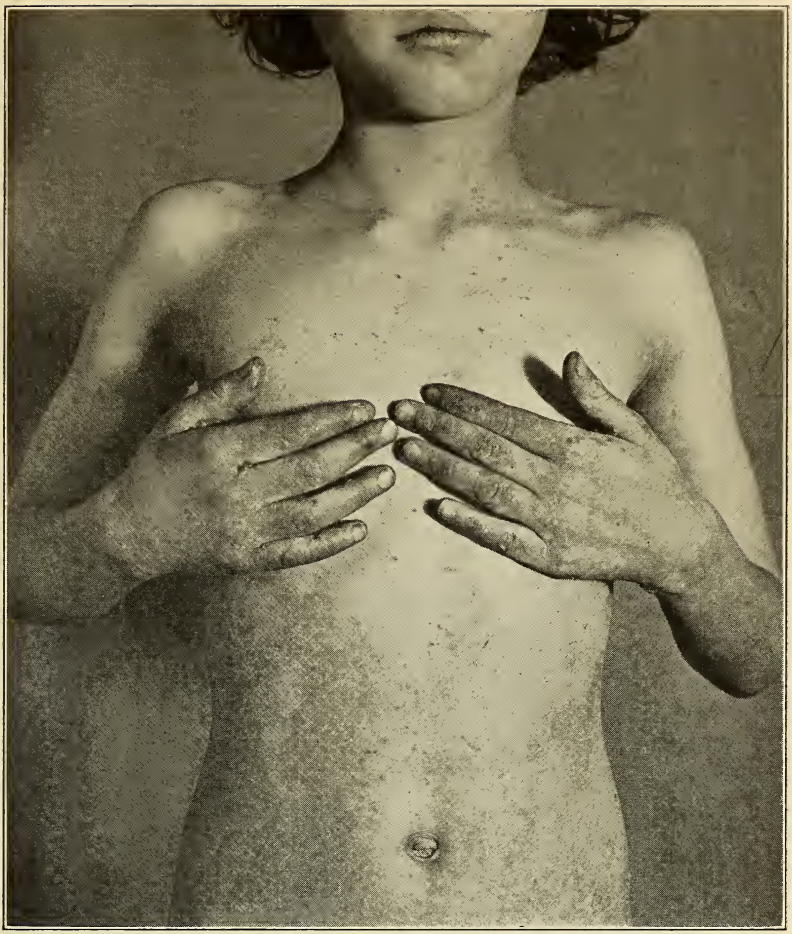

FIG. 43. SCABIES

Note the wide distribution of vesicular papules from the level of the clavicles down. Also observe the grouping near the axillae and in the interdigital spaces. This patient being a child, the relation of the lesions to the breasts is not shown.

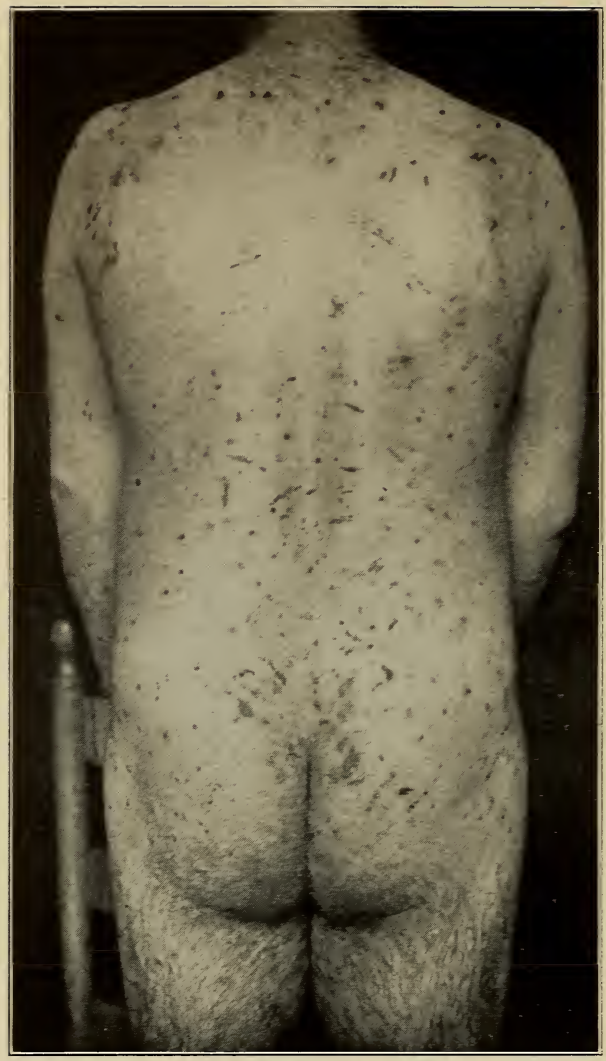

FIG. 44. PEDICULOSIS CORPORIS

In this form of pediculosis the lice inhabit the underclothes, but irritate the skin as they move about. Thus the patient scratches, and the characteristic feature of the disease is the shape and location of the scratch marks, which are most profuse where the underclothes press against the skin. Thus the shoulders and waist are more densely covered with excoriations than other sites. The scratches are linear and often parallel, as is shown near the right shoulder and left flank.

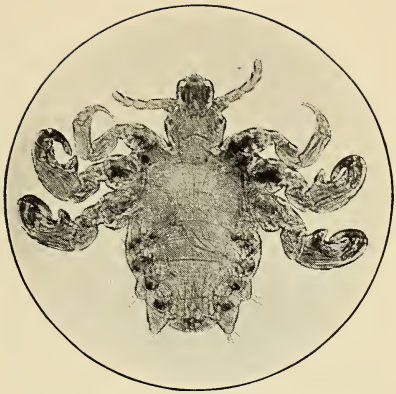

FIG. 45. PEDICULUS PUBIS

As this parasite causes no typical lesions, but merely a simple dermatitis near the affected hairs, the organism itself is shown, as the diagnosis sometimes depends upon recognition of its appearance. The pubic, axillary and body hairs in general are involved. At times the eyelashes are affected.

CHAPTER XXI

INFECTIONS CAUSED BY PROTOZOA

Two conditions are caused by the Leishmania, oriental sore and uta; three by spirochaetes, frambesia, syphilis and ulcerating granuloma of the pudenda; one by trypanosoma, trypanosomiasis; one by an unknown probably protozoön organism, verruca peruana; while rat-bite fever is possibly due to a spirochaete and possibly a streptothrix. Should the latter prove to be correct, rat-bite fever will have to be included among diseases caused by vegetable parasites. In the meantime, because the disease is cured by salvarsan, it will have to be regarded as of animal origin. Because of the extent and importance of the subject, syphilis will be discussed in separate chapters at the end of the book.

ORIENTAL BOIL

Synonyms. Oriental Sore; Aleppo, Delhi or Biskra Boil; Biskra, Oriental, Gafsa Button, etc. French, Bouton de Biskra; Bouton d'Orient; Chancre de Sahara. German, Endemische Beulenkrankheit.

Definition. Oriental boil is an infectious granuloma of the exposed areas, chiefly the face, which runs a definite course and terminates spontaneously in a scar.

Symptoms. At first a maculo papule arises, which is hard and situated on an inflamed base. This scales and secretes a serous fluid which dries into an adherent crust covering an ulcer. The ulcer grows peripherally, and new lesions running a similar course appear near by. By confluence an irregularly outlined ulceration develops, the total diameter of which may be three inches. Within any period up to a year the lesion scars, leaving a pigmented, depressed cicatrix.

Course. The disease has a developmental and involuting stage as described above. Spontaneous recovery takes place within from a few months to a trifle over a year.

Varieties. A verrucous form has been described.

Differential Diagnosis. In countries in which the disease is

endemic, the diagnosis is easy. The facts that it affects only exposed areas, that there are no other evidences of syphilis, and because the Wassermann test is negative, rule out the diagnosis of lues.

Etiology and Pathogenesis. The disease is caused by the Leishmania furuncolosa (Firth) which may be demonstrated in sections by the Nocht-Romanowsky stain.

Treatment. There is no satisfactory treatment, nor is any necessary, since the disease always heals of itself.

Prognosis. The prognosis is good.

Uta is a Peruvian disease clinically resembling lupus vulgaris, and caused by a species of Leishmania.

FRAMBESIA

Synonyms. Yaws; Pian.

Definition. Frambesia is a contagious disease seen chiefly among the natives in Northern Africa, Madagascar, Mosambique, Australia, the West Indies and Asia. It is caused by a spirochaete and produces raspberry-like granulomata.

Symptoms. There are three periods; that of incubation, a primary and an eruptive stage followed, as is believed by some, by a later stage corresponding to the tertiary epoch of syphilis. The incubation period lasts from a fortnight to a month, during which there are mild febrile symptoms, headache, general pains and malaise. Thereupon appears the primary lesion which is always extragenital. This lesion is a crusted, weeping papule usually surrounded by others of a similar character. These unite, forming an ulcer with jagged edges. The primary lesion is soft and found on the exposed parts of the body. Since native clothing is sparse the exposed surface is extensive, so that only the trunk and genitals escape. The primary sore either terminates in a scar, or assumes the character of the secondary lesions which appear after about three months and are immediately preceded by constitutional manifestations similar to those of the onset.

The secondary lesions are irregularly disseminated, red papules with crusts. After existing for several weeks they fade leaving finely scaling areas; or they develop into raspberry-like granulomata varying from one-half to three centimeters in diameter. They exude a thin fluid which dries. Such lesions favor the face and extremities, and commonly form coalescent rings, chiefly at the mouth and anus. Later these lesions grow warty. The palms and soles may be

involved by lesions in all of the stages mentioned, or hard papules form containing corneous plugs. Itching is occasionally seen and within a year the lesions involute spontaneously, pigmented patches remaining. Ulcers, too, may form during the eruptive period, and later in the disease gummata arise. The mucosa is rarely involved, and there are slight adenopathies chiefly in the neck and groin.

Differential Diagnosis. The course of the disease and distribution of the lesions vary slightly from syphilis with which it may easily be confused. The Wassermann reaction is positive, but the mucous membrane is not involved. It has been believed that yaws is a form of syphilis. Frambesia itches and the raspberry lesions are absent in syphilis, nor does frambesia immunize against subsequent attacks of syphilis, which perhaps today is not a point of great weight in so far as there are authenic cases of syphilitic re-infection.

Etiology and Pathogenesis. The disease is caused by the spirochaete pertenuis (Castellani).

Treatment. Arsphenamin affects yaws as it does syphilis. Mercury and potassium iodide are also indicated.

Prognosis. This is invariably good.

Ulcerating Granuloma of the Pudenda. This disease is rare in our climate. It is characterized by an invasion of the genitals and their vicinity with a cicatrizing ulcer. Extension is peripheral while the centre scars over. The scar is nodular and the margin tortuous, elevated, pink or red, and shiny. At times other parts of the body than those mentioned may be involved, and the lesions may be verrucous, crusted or vegetating. The cause is unknown, a bacillus, coccus and spirochaete having been found by various observers. Women are more frequently affected and the disease always comes after puberty. There are no glandular swellings, and all treatment save excision is unsatisfactory.

Trypanosomiasis is capable of causing varied cutaneous manifestations. The disease is due to infection by the trypanosoma gambiense (Dutton) transmitted by the tse-tse fly (glossina palpalis) of the Congo. Patients afflicted with the sleeping sickness have several varieties of skin lesions. The sting of the fly resembles that of a bee or wasp. It is red or blue, tender and on an exposed surface of the body. During the course of the disease itself, toxic, urticarial and vesiculo-papular eruptions appear.

VERRUGA PERUVIANA

Synonyms. Peruvian Wart; Carrion's Disease.

Definition. Verruga peruviana is a cutaneous infection, the actual cause of which has not yet been demonstrated. It is very likely to be a protozoön. This disease is commonly associated with

Oroya fever caused by an unidentified organism, the Bartonia bacilliformis, of a new genus and species. Verruga peruviana occurs on the extensor surfaces of the extremities and on the face and neck, but rarely on the mucosa. It somewhat resembles frambesia. The lesions at first are pink or red and slightly raised, rapidly becoming papular or nodular. In this stage they are conical or hemispherical and about a centimeter broad. Later they grow warty, red and have either a broad or constricted base. At times they are hemorrhagic. Subcutaneous lesions also develop which later ulcerate and crust, giving off a fetid discharge from a gray or black floor.

When associated with acute Oroya fever, a severe intermittent or remittent fever, prostration and pernicious anemia develop. This condition lasts up to four weeks or so and usually ends in death, a few cases, however, recovering. It is after the gradual abatement of Oroya fever that the verrucae appear. Chronic Oroya fever runs a protracted, mild, febrile course, usually ending in recovery and associated with verruca.

Course. Verruga peruviana appears after acute Oroya fever which is not fatal, or in the course of chronic Oroya fever. The cutaneous lesions disappear spontaneously as a rule.

Varieties. There are none.

Differential Diagnosis. Yaws and syphilis are to be excluded. The lesions of yaws contain the spirochaete pertenuis, and many of those of syphilis the spirochaete pallida. Neither is associated with Oroya fever and in both the Wassermann reaction is present, which is not the case in verruga peruviana; nor is the last disease influenced by arsphenamin.

Etiology and Pathogenesis. Verruga peruviana has been known for at least four centuries on the western slopes of the Peruvian Andes, but never below an elevation of one thousand feet. Occasionally cases, as that reported by Jadassohn and Coles, reach Europe and North America. The cause is unknown and, as Strong and his associates point out, has nothing whatever to do with that of Oroya fever. This has been experimentally proven by inoculating a sound person with skin lesions of verruga peruviana. After sixteen days typical lesions developed, but no sign of Oroya fever. The cause of the latter is a hematozoön attacking the red blood corpuscles. It is an independently motile organism occurring in rod shaped and rounded forms, and has been named after its discoverer, Barton.

Treatment. This is symptomatic.

Prognosis. From ten to forty percent. of those affected with Oroya fever die. The prognosis of verruga peruviana is good.

RAT-BITE FEVER

Definition. Rat-bite fever is a remittent or intermittent febrile disease accompanied by skin manifestations. The pathogenic agent is introduced by the bite of the black or brown rat.

Symptoms. At the point of the bite a swelling develops which is indurated and bluish or red. Lymphangitis radiates from the injury and the regional glands are swollen. The exanthem which then develops is bluish red, consists of macules which vary in size from two to ten centimeters, or even more. It is generalized and at times vesicular or pustular lesions are interspersed among the others. Rarely the rash is purpuric and at times urticarial. As the fever diminishes the exanthem fades, but the latter recurs with each recrudescence. The cutaneous manifestations thus resemble those of erythema multiforme. According to Tileston, bluish-red circular spots develop. These are one to three centimeters broad, sharp, a trifle elevated, and they occur on the cheeks, arm, neck, back, chest, belly and thighs. The centre is dark red, five millimeters in diameter, and the lesions fade and re-appear precisely at the same sites with each recrudescence. This form simulates erythema perstans.

With the rash there is fever (103°–105°), malaise, chills and a small, rapid pulse. The patient is prostrated and as the fever drops a profuse perspiration arises. The tongue is coated and white. Nausea, constipation or diarrhoea, salivation and dysphagia, suggesting rabies, are present. At times, too, there are splenic and hepatic enlargement, myocarditis, and nephritis. The tendon reflexes are increased, and occasionally neuralgia, hemiplegia, stupor and coma have been noted.

Course. The disease begins with the rodent's bite. After an incubation period averaging twelve days (the extremes being one to thirty-five days) the first paroxysm develops. This is a febrile attack as described above, accompanied by a rash. The paroxysm ends within four to five days, and a free interval of similar length ensues, followed by another paroxysm. In this manner the disease may progress with from one to twenty-six recrudescences, the average being three to ten. The entire course usually lasts two months, but may be much shorter or prolonged even for two and a half years.

Varieties. There are febrile, sustained febrile and intermittent

types, or a mixed type without any incubation period and with sloughing gangrenous wounds.[1]

Differential Diagnosis. The history of an attack by a rat, the character of the fever, and the nature of the rash are distinctive.

Etiology and Pathogenesis. According to *Tileston* an organism resembling that of relapsing fever was found in the blood. *Schottmüller* described a streptothrix. *Tileston* cured his case with arsphenamin, a fact which indicates the probable protozoön origin of the disease. This view had been previously expressed by *Crohn* from whose work the following is quoted: "In favor of a spirochaete as the causative agent is the similarity of the disease to relapsing fever and syphilis, as regards both the marked nerve symptoms and the apparent curability by salvarsan. In favor of a protozoön cause is the alternation of the paroxysms with afebrile periods, recalling the effects of the malarial plasmodia, though this occurs also in spirochaetal diseases."

Treatment. Arsphenamin should be employed, two or three injections usually sufficing.

Prognosis. Ten percent. of the cases die.

[1] These facts have been assembled from Burrill Crohn's excellent paper entitled, "Rat Bite Fever"; Arch. Int. Med., 1915, XV., P. 1014.

CHAPTER XXII

INFECTIONS CAUSED BY FUNGI

Fungi determine a wide variety of cutaneous reactions. Some live upon the surface of the skin causing almost no morbid changes, their presence alone producing manifestations. An example of this is tinea versicolor. Others reside in the supracutaneous hair, as trichomycosis. Still others flourish in the hair and follicles, as microspora and trichophytons. Of these, some produce scaling, vesiculation and pustulation on the scalp or glabrous areas. Some of them cause purulent granulomata, as do also actinomycetes, blastomycetes, and sporotricha. Some, in addition, provoke systemic infections. The majority are easily cured by suitable local therapy. Others, however, require general medication,— notably actinomycosis, sporotrichosis, and blastomycosis. Perhaps the best way to classify these diseases is parasitologically, as follows:

I. Saprophytes
    1. Cutaneous
        a. Pityriasis versicolor
        b. Erythrasma
    2. Of the Hair
        a. Trichomycosis
        b. Piedra
II. Epidermophytia (Figs. 51 and 52)
III. Dermatomycosis
    1. Microsporosis (Fig. 46)
    2. Trichophytosis (Figs. 47, 48, 49, 50, and 53)
    3. Favus (Fig. 54)
IV. Blastomycosis (Fig. 55)
    1. Saccharomycosis
    2. Gilchrist's form
V. Ray Fungi
    1. Actinomycosis
    2. Madura foot
VI. Sporotrichosis

SAPROPHYTIC DISEASES

Saprophytic skin diseases are caused by the presence upon the skin of fungi incapable of producing profound pathological changes. The micro-organisms flourish in superficial cuticular levels, or on the hairs. The diseases in question are pityriasis versicolor, erythrasma, trichomycosis palmellina, and piedra.

PITYRIASIS VERSICOLOR

Synonyms. Tinea Versicolor; Chromophytosis; German, Kleinflechte.

Definition. This is a mild, scaling, brownish eruption seen on the trunk, and rarely on the thighs and arms as well.

Symptoms. Usually the front of the chest is covered by fawn-colored spots of various sizes. On close inspection the smallest of these macules are seen to correspond to the periphery and immediately adjacent area of the follicles. The larger ones are formed by coalescence of the smaller into irregularly circular, oval, or finely jagged macules. A very fine furfuraceous scaling is present. At times, the macules themselves run together into large sheets beyond the border of which smaller lesions arise, with areas of unaffected skin intervening. The entire chest, back, and even shoulders may be covered by one diffuse patch, or an enormous area closely studded with perifollicular lesions may be seen. At times, only a few small spots are present. Occasionally, instead of the characteristic fawn color, elements of vivid rose modify the tint. Itching is rare. The face and neck are practically never involved.

Course. Untreated, the disease persists indefinitely.

Differential Diagnosis. Pityriasis versicolor usually has a characteristic appearance, and can always be definitely recognized by demonstrating the fungus. This is done by placing a scale on a slide, adding a drop of forty percent. potassium hydroxide, and heating gently until bubbles form underneath the cover slip, which has previously been put in place. The scale, when examined with a number six objective with the light cut down, is seen to be filled with mycelia and spores.

The conditions which the disease sometimes simulates are seborrhoea, freckles, vitiligo, maculo-anesthetic lepra, fading secondary syphilis and ringworm. Seborrhoea is infiltrated, the scales are coarse and greasy; itching is often present; the face and scalp are usually involved. Freckles occur only on exposed

areas where tinea versicolor is never seen. In maculo-anesthetic lepra the general symptoms of the disease are present, but there is no scaling. Nor does fading syphilis scale, and there are other evidences of this disease, such as glandular enlargements, remains of a chancre, and the Wassermann reaction. In vitiligo the patches are white and surrounded by a hyperpigmented area. The reverse is seen in tinea versicolor. No desquamation is present in vitiligo. In ringworm the scalp is usually involved. On the body the scales are coarse and there are vesicles or pustules, features which are never present in pityriasis versicolor. The characteristic fungi of the one disease or the other further distinguish the two.

Etiology and Pathogenesis. The disease is caused by a specific organism, the microsporon furfur. The fungus flourishes in a corneous layer in the shape of mycelia and numerous coarse spores. It is difficult to perfect cultures on artificial media.

Treatment. Of the many methods of treatment employed it is necessary to mention but one because this one invariably succeeds. The patient bathes on three or four successive nights, and after bathing moistens his skin with a saturated solution of sodium hyposulphite. Before using the latter, dilute acetic acid may be dabbed on and dried off.

Prognosis. The condition is always curable. Should it recur, the immediate resumption of treatment at the outset will stop the attack.

Erythrasma is a closely related condition found on the genitals and extending thence to the neighboring regions. It is commoner in men than women, and also is seen in the axillae, below the breasts, and in the body folds of the adipose. The disease is characterized by a coppery, scaling, sharply marginated plaque and itches slightly, if at all. It is caused by the microsporon minutissimum, present in the scales, and is transmitted by contact with an infected person or with infected material. The treatment and prognosis correspond to what has been outlined in pityriasis versicolor. The disease must be differentiated from scaling dermatitis which desquamates more coarsely, itches, and may weep. It must also be differentiated from epidermophytia inguinale, a point which will be discussed below.

Trichomycosis Palmellina is a condition of the axillary hairs caused by a growth on their shafts of colonies of fungi. The organisms impart a scarlet color to the hairs at their points of colonization. At times the pubic hair is involved. Fracture of the affected hairs may take place. The treatment consists in shaving off the diseased hairs and applying a mild bichloride solution to the shaved area. In certain tropical countries, yellow and black varieties of this disease are known.

Piedra is a nodulation of the hair caused by minute concretions situated from one-half to one and one-half centimeters apart along the shaft.

These concretions are colonies of fungi, the trichospora, of which four varieties are known. The concretions are hard, fusiform or spherical, and are built about the hair with the latter as an axis. The condition must be differentiated from trichorrhexis nodosa (Chapter XXXIV) and monilethrix (Chapter XXXIV). In the former there are no concretions, the nodes being due to local fractures of the hair with raveling of the cell strands. No organisms are demonstrable as in piedra. Monilethrix hairs present alternate constructions and fusiform swellings, so that the entire shaft resembles a minute rosary. The disease is a congenital and familial maldevelopment and not due to an infection. The most promptly efficacious treatment in piedra is to shave.

Dermatomycoses. Dermatomycoses are diseases of the hairy or glabrous skin caused by the activity of parasitic fungi which attack the epidermis, derma, follicles, hairs, nails, or any combination of these structures. They produce alopecia, temporary in most instances, permanent in favus. They are capable of provoking vesicular, pustular, or merely squamous eruptions, and some varieties produce follicular suppurations and granulomata. The fungi belonging to this group fall into three varieties,— microspora, trichophyta, and achoria. The microsporon causes alopecia and squamous eruptions; the trichophyton causes these, but also may provoke pustular and granulomatous dermatoses; the achorion causes favus which produces permanent alopecia with atrophy, and on the glabrous surfaces, atrophy. Some varieties of the fungi immunize against further attacks; others render the skin allergic. All of the organisms have spore forms, and thread forms or mycelia. In this book only the commoner varieties of this disease will be discussed.

MICROSPOROSIS (FIG. 46)

Definition. Microsporosis is a disease, prevailingly of the scalp, leading to temporary alopecia and caused by a fungus with definite morphological and cultural characteristics.

Symptoms. The disease is characterized by the appearance upon the scalp, chiefly in children, of bald patches (Fig. 46) up to two or three inches in diameter. These patches are covered by dirty gray scales, are circular or oval in outline, sharply circumscribed, and on closer inspection are seen to contain hairs broken off at the uniform distance of three or four millimeters above the level of the scalp. All the hairs in the involved area are affected. On attempting to extract them, they are found to come out without resistance, and when held so that they reflect the light, or against a dark background,

they are found to look a dusty gray. Near the large patches smaller ones are frequently observed, including half a dozen hairs. There may be very few or a great many affected sites. At times, on the glabrous surfaces of the body, are seen fugacious patches, yellowish and slightly scaly, resembling seborrhoea. They never scale or crust. Recently *Lewendowsky* reported cases in Hamburg in which kerion celsi, to be described below, was caused by this organism.

Course. The disease usually begins from the age of five upwards terminating, when untreated, at puberty. It causes transitory baldness.

Varieties. There are two varieties of the disease, that produced by the human microsporon, and that by microspora of animal origin. The former is the microsporon Audouini, the latter the microsporon lanosum, the source of which is domestic animals, notably dogs and cats. Rare varieties are the microsporon fulvum, equinum, and villosum.

Differential Diagnosis. The diagnosis depends upon the clinical appearance, namely, the gray scales, gray, broken off hairs, presence of the disease in children, and the microscopic demonstration of the fungi. Psoriasis (Fig. 20), seborrhoea (Fig. 25), and alopecia areata (Fig. 78) are to be differentiated. In psoriasis other and characteristic parts of the body are involved, the hairs rarely fall out, and the scales are thicker. The same holds true of seborrhoea which, on the scalp, is diffuse, itches, and in which the hair usually tends to fall. In alopecia areata there are no scales, a few hairs are present looking like exclamation points and they are not gray. In none of these three diseases are organisms found.

Etiology and Pathogenesis. The disease is caused mostly by the microsporon Audouini, and lanosum, and exceptionally by the other forms mentioned above. The first is common in England and less so in other parts of the world, while microsporon lanosum is the commonest form in general. It is chiefly derived from cats and dogs, although transmission from person to person is very frequent. The fungi invade the follicles growing downward, and encasing the hair in a sheath, imparting the peculiar gray appearance to the hair noted in the symptomatology. Under the microscope the hair is found covered with a mosaic of spores, reaching from the broken end of the shaft down to just above the root. At this paint a few mycelia are found entering the shaft and forming Adamson's fringe. This fringe does not descend far enough to interfere with hair growth. The spores are always external.

On *Sabouraud's* maltose peptone agar medium the organism grows as a white or gray disc with a central button, and four or five radial depressions. The colony is white or grayish and tends to consist of concentric rings of increased and decreased density, imparting to the culture the appearance of a target.

Treatment. In general practice the hair should be clipped, the scalp covered with a twenty-five percent. idodine in sixty percent. alcohol, the brown color thus emphasizing the lesions, and the involved patches should be epilated. Or four drams of idodine scales to an ounce of goose grease should be applied once a day until exfoliation takes place. This often cures the patient. The best therapy in expert hands is a depilatory dose five Holzknecht units) of X-Rays, gauged by the *Sabouraud Noiré* pastille, or by the *Kienboeck-Adamson* method. By means of the Coolidge tube dosage can be measured without these means. In two to three weeks all the hairs fall and with them the fungi. Within six weeks a new growth of hair appears.

Prognosis. The disease usually disappears spontaneously at puberty, and is always curable.

TRICHOPHYTOSIS (FIG. 47)

Synonyms. Ringworm; French, Teigne Tondante; German, Scherende Flechte.

Definition. Trichophytosis is a disease manifold in its symptomatology, caused by fungi called trichophyta, of which there are many sorts. These micro-organisms provoke reactions in the hair, hair follicles, nails, epidermis and in the cutis.

Symptoms. In general they are capable of causing alopecia (Fig. 47), destruction of the nails, squamous, vesicular (Fig. 48), pustular and granulomatous responses in the skin. The alopecia may be in circular (Fig. 47), oval, or irregular patches, and accompanied by scaling vesicles or pustules. The nails may be broken, or just brittle and lustreless, with or without thickened and inflamed nail beds. The squamous, vesicular and pustular lesions are usually inflamed, circular, and more or less infiltrated. The granulomata are fleshy, discoid, purulent lesions on the scalp or body. They may or may not itch.

Course. The disease may occur at any age, all forms prevailingly attacking children, except the granulomatous and pustular varieties which attack adults equally, if not actually more often.

Untreated, the disease usually persists and gets worse, although not infrequently it may disappear of itself.

Varieties. The regional varieties are trichophytosis of the scalp (Figs. 47 and 49), body (Fig. 48) and nails (Fig. 50). The clinical varieties are follicular, ungual, squamous, vesicular, pustular, and granulomatous. The varieties according to pathogenis are determined by the type of the exciting organism. In point of fact, these purely artificial distinctions are practically not tenable, as alopecia may occur with either scaling, vesicles, or pustules, and granulomata may develop on the scalp or elsewhere, in the former eventuality causing at least temporary baldness. Pustular varieties occur anywhere on the body. Vesicular or squamous dermatitis may be simulated. Thus, an enormous number of pictures is produced.

We must here digress to discuss the simpler features of the trichophyta. These may flourish within the hair, or outside of it, or in both places. Those found within the hair are called endothrix, those without ectothrix, and those found in both places neo-endothrix. The ectothrix are again subdivided into a small and a large spored variety, and the entire classification may be represented as follows:

Class	Common Forms
I. Endothrix	Trichophyton Crateriforme
(Spores and Mycelia within the hair)	Trichophyton Acuminatum
II. Neo-Endothrix	Trichophyton Violaceum
(Spores and Mycelia both within and without the hair, as a cuff)	Trichophyton Cerebriforme
III. Ectothrix	
(Spores and Mycelia within and without the hair and filling the entire follicle)	
A. Microsporon	Trichophyton Gypseum
B. Megalosporon	
1. True	Trichophyton Rosaceum
2. Favus-like	Trichophyton Faviforme Album

TINEA

Trichophytosis capitis, or ringworm of the scalp, is also called tinea, or herpes tonsurans, tinea tondens, Scherende Flechte, Teigne Tondante. It occurs mostly in childhood as circular, oval or irregular patches, often two centimeters in diameter, at times singly, but

usually disseminated. The affected surfaces may appear entirely bald, but on closer examination, either stumpy hairs are seen curled up in the scales, or long, fragile, lustreless hairs amidst vesicles and pustules may be present. The latter are arranged in simple or concentric rings. At times, the hairs are broken off flush with the skin surface and they then look like comedones. All the affected hairs are easily withdrawn from their follicles. Seborrhoea may be closely simulated. No matter what disease is mimicked, however, the diagnosis always rests upon the discovery of the organism. The procedure is similar to that described in connection with microsporosis. The spores or mycelia are found, as the case may be, within or without the hair, or both. Several hairs must be examined, since some of them within the affected areas remain untouched, in this respect differing from microsporosis, as well as in the circumstance that in microsporosis the hairs are broken off at a uniform level above the scalp. But when the connective tissue about the follicles is affected a disc-like granuloma, the kerion celsi, develops. This will be described more fully in a subsequent paragraph. It is usually caused by some variety of endothrix, either the crateriform or acuminate.

TRICHOPHYTOSIS CORPORIS (FIGS. 48 AND 51)

This designation is applied to trichophyton infections exclusive of the scalp, bearded area and nails. There are several clinical varieties determined partly by accidents of site, and partly by the strain of the organism. Except for mycologists, the second consideration is relatively unimportant. The different pictures are determined by the nature of the cutaneous reaction to the infecting agent. Thus, there are squamous, vesicular, pustular and papular lesions. Frequently, body trichophytosis is associated with scalp or nail infections.

The squamous ringworms are red, discoid, circular, or annular lesions, and when numerous or confluent become festooned. The entire surface may be scaling, or this may be the case only at the advancing margin. In other forms there is central scaling and peripheral vesiculation or pustulation. After the centre has healed and the margin has advanced, central recrudescences occasionally occur, giving the picture of concentric active vesicular or pustular rings.

Papular ringworm is rare. It takes the form of lichenous lesions closely resembling lichen scrofulosorum (Chapter XXV). This

condition was first recognized by *Jadassohn* who named it lichen trichophyticus. The lesions consist of minute follicular elevations studded over the body. They are single or grouped as in lichen scrofulosorum, and sometimes itch. Their color is a waxy ochre to brown, and their pathogenesis is not clear. Always the lesions are associated with typical ringworm elsewhere.

Unusual pustular ringworms are those resembling acne, and a peculiar granuloma, granuloma trichophyticum, first described by *Majocchi*. This disease occurs anywhere, chiefly on the back of the hands, and the starting point is an infective folliculitis from which spreads the furuncular infiltration.

Ringworm often itches, and clinically it frequently simulates seborrhoea, vesicular or pustular dermatitis. The seborrhoeal type is squamous. The vesicular variety is of extreme importance for it looks like occupational, dishydrotic (Fig. 51) and other types of circumscribed acute and chronic dermatitis. It may occur anywhere on the body, but it is commonest on the hands and feet. It itches, and forms more or less inflamed circular or irregular vesiculosquamous areas, in themselves often not in the least suggesting ringworm. The organism may be recovered in culture, or demonstrated microscopically. Many of these cases are restricted to the interdigital spaces (Fig. 51), many are found on the palms and soles, and a discoid erythemato-vesiculo-pustular variety is found on the back of the hands and forearms.

The clinical significance of this group of diseases lies in their similarity, as already stated, to vesicular dermatitis. Unless the fact is recognized and the correct diagnosis established, the condition is likely to be obstinate. Many patients are thus indefinitely treated for a so-called chronic eczema. Quantitative blood and urine determinations are made, diets are prescribed, and the patient is enrolled as a victim of abnormal metabolism, when a strong solution of potassium hydroxide, a microscope, and a few tubes of Sabouraud's medium would furnish the correct diagnosis and a clue to proper therapy, albeit we should be deprived of the joys of pseudo-scientific profundity.

TRICHOPHYTOSIS BARBAE (FIG. 53)

This form of ringworm is a suppurative folliculitis of the bearded region, and hence restricted to men. It is conceivable that hirsute women might be infected. It is also known as barber's itch and sycosis parasitaria, in contradistinction to staphylogenic folliculitis of the same area called sycosis non-parasitaria. Such distinctions

are absurd, for whether the pathogenic agent is a bacterium or fungus it is a parasite. Thus, the one form should be called folliculitis suppurativa trichophytica barbae, and the other folliculitis suppurativa staphylogenes barbae, since it is always a staphylococcus which provokes the latter form. (Chapter XXIII.)

Folliculitis suppurativa trichophytica barbae is characterized by the presence of pustules which are inflamed, hard, often markedly infiltrated, and from the summit of which a hair projects. It is in and about the follicle containing this hair that the pathological process originates. Often several of the lesions coalesce into a large, lumpy, red patch. The entire bearded area, or only a part of it, may be involved. The process is to be distinguished from its staphylogenic cousin. In the latter there is less induration and the lesions are prevailingly smaller and more superficial. The final diagnosis, however, depends upon a determination of the presence of the fungi or bacteria, as the case may be. Iodides and bromides are capable of causing a pustular dermatosis in this region. Recognition of these conditions will depend largely on the history, and the absence of fungi in the hairs. Furthermore, in iododermata and bromodermata the lesions are less likely to confine themselves to the bearded area, and will be present on other parts of the face, neck and even body.

Kerion Celsi (Fig. 49) has been alluded to twice. It is a pyogenic granuloma usually caused by trichophyta, rarely by microsporon, and it arises on the scalp or in the bearded region as the sole manifestation of disease, or accompanying other commoner forms of trichophytosis. The lesion is a red disc, the surface of which is a granulated plateau oozing a thin, seropurulent fluid. It, too, must be differentiated from the halogen dermatoses as outlined above. Sporotrichosis and blastomycosis must also be excluded, partly on clinical grounds which will be mentioned below, and partly by laboratory methods as mentioned above, designed to demonstrate the pathogenic organisms. The disease responds readily to correct treatment, but without this may persist indefinitely.

Trichophytosis of the *Nails* (Fig. 50) exists independently, or associated with any of the other types of ringworm. It affects one or more fingers the nails of which are lustreless, brittle and often eroded, so that their surface is finely corrugated by vertical ridges. Between the nails and pulp of the fingers lies a dry mass of detritus. The nail bed is inflamed. Dermatitis, psoriasis and trophoneurotic disturbances of the nails must be excluded. The diagnosis is clinched

by finding the fungi in nail scrapings. The clinical points of distinction are as follows: In dermatitis the nails are usually transversely ridged, but may be longitudinally, are less brittle, and not so lustreless, and other evidences of the disease are present. In psoriasis, punctiform depressions are seen on the nails, as well as the general features of the disease elsewhere. In trophic disturbances the nails are distorted, rarely destroyed, and evidences of central nerve disease may be present.

ETIOLOGY AND PATHOGENESIS OF RINGWORM

Ringworm is contagious and is transmitted by contact with infected people or animals, notably domestic animals, and chiefly dogs, cats and horses. The two sexes are equally vulnerable. Unquestionably children are more susceptible than adults, but no age is immune. Trichophytosis barbae is often acquired in barber shops, but more frequently is found in people whose vocations or avocations cause close association with animals. Epidemics have been known, and from the fact that among those exposed not all are infected, it is obvious that predisposition plays an important rôle. To what extent this can be determined by modern immunological methods is questionable. Certainly people cured of kerion appear partly immunized to subsequent infection. It would thus appear that deep trichophytosis is capable of producing a certain degree of immunity. This is certainly not so with superficial infections. It would appear then, that all statements with regard to the efficacy of vaccine treatment, percutaneous tests and complement fixation in superficial forms of ringworm must be regarded sceptically. *Bruno Bloch's* work on allergy seems significant, and it is not at all unlikely that the epidermis possesses some inherent quality of susceptibility as well as resistance to the fungi. In fact, this would seem obvious, but it is another matter to conclude that the entire host must react to a superficial pathogenic agent. The determining factor in all trichophytoses is the fungus. Different varieties cause different lesions, but only the common strains mentioned earlier in this chapter will be enumerated.

Trichophyton acuminatum causes alopecia. The hairs are broken flush with the skin surface, and one or more bald, discoid areas form, reaching a maximum diameter of two inches. About each hair is a minute papule. A rough resemblance to papular acne is presented. Similar patches may appear on glabrous surfaces. At times there are vesicles.

Trichophyton crateriforme causes gray, scaly, regular, round patches similar to those of microsporosis. There are long, healthy hairs among the diseased ones, however, and in the scales one finds many gnarled stumps of hairs containing the fungi which are of the pure endothrix type. This fungus is also capable of producing a vesicular eruption in circular plaques on the glabrous skin.

Trichophyton cerebriforme causes circinate, vesicular patches which may reach a diameter of two inches. The lesions are pink and impetiginous. Painful suppurative folliculitis of the beard is also produced by this organism as well as painful subcutaneous abscesses. Scalp ringworm, pink, bald, weeping and in numerous patches one to one and one-half inches in diameter, represent the clinical picture this fungus produces in children.

Trichophyton violaceum causes tinea tonsurans, circinate, vesicular patches on the glabrous area, folliculitis suppurativa barbae and onychosis.

Trichophyton gypseum presents two subvarieties, the asteroid and the radiolate. The asteroid produces pink, bald discs on the scalp, covered by small grayish scales. The hairs break off about one-quarter inch above the skin surface. Kerion is also produced, and on the glabrous portions vesiculopustular circular patches up to two inches in diameter. It also produces suppurative folliculitis of the beard. The radiolate variety produces similar lesions, as well as vesiculopustular ones with marked infiltration.

Trichophyton rosaceum forms imperfect circles, the imperfection being caused by the effacement of segments. The patches are dry and vary in size. In the bearded region plaques up to two inches in diameter form and the hairs are broken off one-eighth of an inch above the surface of the skin. A gray conical papule rises about each follicle.

Trichophyton faviforme album forms lesions resembling pityriasis rosea.

Treatment. *The Scalp.* The object of treatment is to get rid of the fungi. This may be accomplished in two ways, either by destroying them with antiseptics, or by removing them mechanically by epilation. Many methods designed to accomplish both of these ends are contained in the literature. The best antiseptic is iodine in twelve percent. strength in goose grease or lanolin. This is rubbed in twice a day over the whole scalp until a dermatitis develops. Then, soothing ointments are applied. The process of irritating and soothing is alternated until the patient is cured. This

usually requires at least several months. Chrysarobin may be substituted for iodine, and is employed in five to ten percent. strength in equal parts of glycerine and chloroform. The general management of the case is the same as with iodine. Sulphur, creosote and resorcin have also been employed. *Sabouraud* shaves the scalp and applies twenty-five percent. iodine in sixty percent. alcohol. Epilation is accomplished either by the use of forceps which takes long, or by employing the Roentgen rays, as described in microsporon infections. The hair falls in a fortnight and re-appears within six weeks. This treatment can be properly carried out only by experts.

The Body. On glabrous portions of the body a cure can usually be effected in superficial cases within forty-eight hours by painting the patches with a ten percent. emulsion of chrysarobin in chloroform. After this the surface is coated with flexible collodion. A dermatitis develops, and the affected epidermis peels with the fungi.

The Beard. The treatment is identical with that of the scalp. Suppurative trichophytosis in non-bearded areas and occasionally in bearded areas is best treated by removing the crusts and washing the skin. A daily light shave is necessary. Iodine salves are used, or the pustules are punctured with a fine cautery. Epilation is useful, but the best treatment is to employ the X-Rays as above.

The Nails. Divulsion of the nails produces the quickest cure. If this is undesirable the nails should be scraped daily, and then immersed for a few minutes in a one percent. bichloride solution. It is also necessary to apply the medicament under the nail. This can best be accomplished by forcing a little cotton saturated in the above solution, or covered with a ten percent. ammoniated mercury ointment under the nail and keeping it there constantly.

Kerion Celsi. This may be treated as the other suppurative forms are, but excellent results are produced by iodine cataphoresis. The negative electrode of a galvanic current, the sponge having been dipped in tincture of iodine, is applied to the lesions and held there for ten minutes, while a current of four milliamperes is allowed to flow. This should be done twice a week. Although the method is a trifle painful, the results are splendid, a cure not requiring more than three weeks. Similar results may be obtained with ten percent. aqueous solution of copper sulphate, the positive pole, however, being applied to the lesion.

Prognosis. This is always good.

Special Forms of Tinea

EPIDERMOPHYTIA INGUINALIS (FIG. 52)

Synonyms. Eczema Marginatum, Tinea Cruris.

Definition. Epidermophytia inguinalis is an infection chiefly of the perineum and genito-crural folds, caused by the epidermophyton inguinale.

Symptoms. The disease occurs on the perineum and inner aspects of the thighs. It is an exudative catarrh, bright red in color, with a raised, sharpened marginated, vesicular, circular border. The lesions group themselves in plaques which coalesce into broad patches over the sites mentioned, and extend often to the pubis and sacrum. At this stage the margin is convex or festooned, and the centre may be pale. Itching is often severe and this, as well as the inflamed color, are increased by sweating and friction. At times the axillae, breasts and navel are involved. Obstinacy and tendency to relapse characterize the disease. *Whitfield,* corroborated later by *Ormsby* and others, found vesiculopustular, interdigital, palmar, and plantar hyperkeratotic forms resembling dyshydrotic and acute vesicular dermatitis. Intense pruritus is a salient symptom of these special forms. The clinical aspects of this type vary in no wise from the picture of hyperhydrotic dermatitis or pompholyx. Great resistance to ill-adapted therapy is another feature, and still another is the acuity of its development, for sometimes within twenty-four or forty-eight hours on a hitherto healthy integument a full blown rash arises.

Course. The disease persists indefinitely unless properly treated and ultimately leads to lichenification.

Varieties. The varieties, as mentioned above, are the inguinal, including axillary and umbilical, and the exudative dermatitic form resembling pompholyx. This type is palmar and plantar, as well as interdigital.

Differential Diagnosis. Pompholyx, ordinary dermatitis, and erythrasma must be differentiated. The first two look and act just like epidermophytia, and only the presence of the fungi, as demonstrated by cultivation, renders possible the correct diagnosis. Erythrasma is also a perineal disease, but there are no vesicles or rings; rather, indeed, a diffuse, slightly elevated, sharply margined coppery plaque. This condition exists almost exclusively in men. The diagnosis is clinched by demonstrating the microsporon minutissimum.

Etiology and Pathogenesis. Both sexes are attacked, men, however, more than women. Exposure in common carriers and hotels is an important factor. Excessive local perspiration predisposes to the epidermophyton, and these facts, with the organism itself, comprise the important etiological moments of the malady.

Treatment. The therapy is simple. Many methods have been employed, but usually only one, as suggested by *Whitfield,* is needed, for this is generally efficacious.

Rx. Acid Salicylic 2
Acid Benzoici 4
Ung. Aq. Rosarum
qs. ad. 30
Sig.

This should be applied twice daily to the affected sites, and usually, at the most within ten days, the eruption begins to disappear. At times it causes a marked inflammatory reaction. Thus, a soothing ointment is indicated. Often, an erythema survives the original eruption for weeks, or even months.

Prognosis. A cure is always possible under proper therapy.

MISCELLANEOUS AND UNUSUAL FORMS OF TRICHOPHYTOSIS

Dhobie Itch, Tinea Imbricata and Pinta constitute this group. There are also still rarer forms which need not be mentioned. *Dhobie,* or *Washerwoman's Itch* is a tropical disease, similar to epidermophytia inguinalis, and is caused by fungi unknown in our climate. *Tinea Imbricata* is an ichthyosiform, semicircular or circular eruption. It itches severely, and the broad scales are inbricated, get horny and curl at the edges. It is caused by an endodermophyton called by *Castellani* either the concentricum of indicum. The malady is non-inflammatory. *Pinta* is due to a penicillium and causes a generalized rash, preceded at times by systemic symptoms. The lesions are polychrim, scaling, and the fading forms resemble parapsoriasis.

FAVUS (FIG. 54)

Synonyms. Tinea Favosa; French, Teigne Faveuse; German, Erbgrind.

Definition. Favus is a fungus disease of the scalp and body characterized by the presence of yellow, saucer-shaped crusts or scutula, and leading to permanent alopecia with atrophic scars.

Symptoms. Favus affects the scalp, nails, and glabrous portions of the skin.

When the scalp is affected, minute red inflammatory papules are seen which, when punctured, give forth a puriform fluid. As the

papules grow they are surmounted by sulphur-yellow or lemon-colored, saucer-shaped structures, the concavities of which are uppermost, the convexities lying in the skin below the epiderm. It is this circumstance which causes the atrophic scar, for the connective tissue with the skin organs is permanently destroyed. The hairs in the involved area fall and the follicles are disorganized. The affected scalp has a peculiar mousy odor. The saucer-shaped crust is a pure culture of the fungus and is called a scutulum. In addition to these crusts are others formed in the usual manner through irritation and scratching. As the scutula enlarge and coalesce they form yellow, honey-combed bodies, the sulphur shade of which is often modified by dust, blood, ointments and matted hairs. The original color, however, may be restored by swabbing off the affected area with alcohol. On removal of the crusts, a bleeding, depressed surface remains, its size determined by that of the crust. Such an area is entirely bald and always remains so. The aspect of a patient who has had favus is characteristic. The surviving hair remains in tufted patches of irregular outline in the midst of an otherwise denuded scalp which is adherent below, glazed, and irregularly scarred.

Favus of the nails causes changes similar to those of tinea as described above. The etiological diagnosis is possible only by microscopic or cultural demonstration of the fungus.

Favus of the body forms a fairly characteristic clinical picture. The glabrous portion of the skin is more or less studded with scutula sometimes to an extreme degree of generalization including the whole body. The scutula may coalesce, forming enormous plaques covering the thigh, or the chest, or an entire extremity with high, honey-combed, friable, sulphur-colored crusts. Circular vesiculopustular lesions recalling ringworm may be seen. Although favus of the scalp may exist alone for years, frequently scalp, body and nail involvement may co-exist.

Course. Starting gradually, the disease persists for an indefinite period, unless properly treated. It leaves the affected areas of the scalp permanently bald, and glabrous areas permanently scarred.

Varieties. The clinical varieties are the body, nail, and scalp forms. The etiological varieties are the common type caused by the Achorion of Schoenlein, and the rarer types caused by the Achorion Quinkeanum or mouse favus, and the Achoria gallinarum, gypseum, and violaceum.

Differential Diagnosis. Active favus resembles ringworm, seborrhoea of the scalp and impetigo. Healed forms resemble lupus

erythematosus and pseudopelade or folliculitis decalvans. In the first group the yellow scutula are the distinctive feature. If the sulphur color is masked by dirt or grease, the alcohol swabbing as mentioned above will reveal the characteristic hue. Removal of the scutula discovers a roseate, superficially ulcerated, often bleeding area. The scutula examined microscopically are found to be composed of spores, and the involved hairs have spores without, and spores and mycelia within. *Sabouraud* also notes that, in boiling the hair up with hydrate of potash, a few bubbles of various sizes adhere to the shaft. The spores likewise vary in size. On *Sabouraud's* maltose peptone agar the colonies reach the size of a dime, are yellow, waxen, and honey-combed. Favus rarely, if ever, forms the rings and scales, and more rarely the vesicles and pustules, of ordinary ringworm. Nail favus clinically cannot be distinguished from ringworm of the nail. Only a microscopic or cultural differentiation is possible. Seborrhoea of the scalp causes no cicatrization, nor do scutula form in this disease. The crusts are greasy, never light yellow, and contain no fungi. The golden crusts of impetigo roughly simulate scutula, but the dried serum forms shiny beads until they become discolored, and no fungi are present. The favus scar differs from that of erythematous lupus in that it is firmer, and no other evidences of lupus are seen. The scars date back to a process which existed years before, and often the skin is bound down. Pseudopelade is found in discs like those of alopecia areata, and punctiform depressed scars are seen corresponding to atrophied follicles rather than to coarse ulcerations.

Etiology and Pathogenesis. The cause is usually the Achorion of Schoenlein; rarely the other forms mentioned. The disease is highly contagious, and is most frequently seen in Russian Jews.

Treatment. This corresponds to that of ringworm.

Prognosis. A cure of the active process can always be effected, but the cicatrization and baldness it provokes are permanent. Thus, unless treated early and well, the prognosis as to cosmetic result is bad.

BLASTOMYCOSIS (FIG. 55)

Synonyms. Blastomycetic Dermatitis; Saccharomycosis; Oidiomycosis; German, Hefenmycosis.

Definition. Blastomycosis is a partly cutaneous and partly systemic and cutaneous disease, the skin symptoms of which consist of the formation of purulent granulomatous and gummatous lesions. These are produced by either of two types of yeast fungi, saccha-

romycetes or oidiomycetes, each of which determines a relatively distinct type of cutaneous reaction.

Symptoms. Blastomycosis may be purely cutaneous. If so, ulcerations with vivid red borders arise, surrounded by acniform lesions at and near the margin. Both lesions form crusts upon the removal of which the ulcerating floor is exposed. A grumous green substance is secreted containing a crumby material. The pus, which is composed of leucocytes, epithelial cells, yeast fungi and even giant cells, is auto-inoculable and produces other lesions on the patient. It is always on exposed areas that the process begins. Osteomyelitis, pulmonary, hepatic and general systemic involvement may accompany the picture.

At times the ulcers are numerous; at times sparse. The characteristic features are the vivid margins, the secretion, the outlying acniform pustules, and often the floor fungates verrucously, forming vegetating granulomata of a purplish hue. The vegetations bleed easily, glisten, and give off a secretion precisely like the one described above.

This description outlines the American variety of the disease first recognized by *Gilchrist*. *Buschke,* in 1894, described the first case of blastomycosis known to the world. The pathological work was done by *Busse*. The European variety had some of the features seen in *Gilchrist's* type, but began more as a subcutaneous gumma and abscess forming process which secondarily invaded the skin. This case was fatal. The Europeans consider their type of blastomycosis due to saccharomycetes, and the *Gilchrist* variety as due to oidiomycetes. The latter, our native form, is distinguished from the European clinically by the fact that the acniform abscesses develop on its outer margin and are very minute, and by the further fact that the surrounding skin is inflamed in a manner suggestive of dermatitis. Hence, it is called blastomycetic dermatitis.

In the systemic cases which resemble sepsis, plus the cutaneous features of blastomycosis, numerous ulcers appear all over the body. At times, both in the local and general forms, tremendous skin surfaces are involved. When healing occurs, a pliable scar remains.

Course. Only one lesion may appear, or several, or the entire body may be covered, many systemic cases giving the associated picture of acute osteomyelitis, pneumonia, or sepsis. Early death may occur, or death may follow a long chronic illness, but many cases recover.

Varieties. There are two forms of the disease, the European or

saccharomycosis, known as the *Busse-Buschke* type, and the American or oidiomycosis, known as the *Gilchrist* type. Symptomatically, there are local or systemic forms, and clinically, the ulcerating, acniform and granulomatous types, all of which have been referred to above.

Differential Diagnosis. An examination of the pus in all forms, or of sections, will reveal the nature of the disease. The fungi, double contoured, highly refractile bodies from ten to forty microns broad, are present. At times budding forms are seen. Sections from the lesions show a granuloma consisting of numerous abscesses, many of which are included in epidermal walls. Giant cells are usually numerous, but may be absent, and they contain the organisms. Clinically, the systemic forms must be differentiated from sepsis. This can be done by blood culture. The skin lesions should give the proper clue, and the necessary laboratory examinations should clinch the diagnosis.

Tuberculosis, syphilis, sporotrichosis, and bromide and iodide rashes must be excluded. In all of these diseases the differentiation depends, in a large measure, upon the examination for fungi. Tuberculosis, save tuberculosis verrucosa cutis (Chapter XXV) and syphilis never show small abscesses, the tuberculin and Wassermann reactions are positive, and in many forms tubercle bacilli are found in the tissue, while the pus never shows blastomycetes. Other evidences of both tuberculosis and syphilis are often present. Sporotrichosis closely resembles blastomycosis, as to the lesions themselves, but their arrangement is usually striking in that they are chain-like, following the lines of lymphatic vessels in the involved regions. The sporothrix is easy to cultivate, and grows in heaped up, mucoid, gray or black colonies on almost all media; while the colonies of the European blastomycosis are indefinite, light gray, yellowish, flat and sticky, and the colonies of the American varieties (of which there are several) grow in various ways, usually in discs. They are dry and covered by feathery-white hyphae. The halogen dermatoses closely resemble blastomycosis, and only the history of drug ingestion and the absence of fungi lead to a correct conclusion.

It is almost impossible to confuse lepra with blastomycosis, but the differentiation would depend upon the discovery of either of the respective organisms of the two diseases. The deep forms of trichophytosis, namely kerion celsi and pustular ringworms, closely resemble blastomycosis, and the differentiation here depends absolutely upon laboratory examinations. It is possible for breaking down

sarcomas to resemble isolated blastomycosis lesions. In these cases only histological and pathological examinations will prevent error.

Etiology and Pathogenesis. The disease is caused by a yeast fungus. The European variety is due to saccharomycis, a round, double contoured organism, whose diameter is equal to or twice that of a lympocyte. The centre is granular and basophile, the margin hyalin. It is often seen in the budding stage. The American variety is due to an oidiomycis resembling the above, but often twice the former's size, and frequently associated with mycelia in the tissue. At first the Europeans considered the organism described by *Gilchrist* in the American variety as a contamination. In recent years, however, these doubts have disappeared.

Treatment. Treatment is internal and local. The former consists of huge doses of the iodides, from one half drachm to two drachms three times a day. Local excision, cauterization, or the X-Rays should be used, if there is no prompt response to iodides. It is unnecessary to state that if no radical local measures are employed, in any case local antiseptic treatment is indicated. It is particularly in isolated or in sparse lesions that the X-Rays are of value. A Holzknecht unit, applied three times at weekly intervals should suffice. Another excellent method for local treatment is that of cataphoresis described in connection with ringworm granulomas.

Prognosis. Except in the rare systemic cases, of which only about ten percent. survive, the prognosis is good.

SPOROTRICHOSIS (SCHENCK-HECKTOËN)

Definition. Sporotrichosis is a cutaneous disease usually due to lymphatic involvement from a primary sore produced by a sporothrix. The lymphatic lesions include the skin by extension so that subcutaneous nodules, abscesses, and cutaneous ulcers and granulomata resembling tuberculosis verrucosa cutis are formed. The disease is largely ascribed to *de Beurmann* of Paris. This is entirely unjust, as there is no question that *Schenck* preceded him by a number of years, and it was only because of ignorance of the foreign literature that the Parisians assumed priority, and it was due to the same cause that Americans permitted this.

Symptoms. As a rule, a wound on the finger secreting a watery or serous pus appears. This develops into a granulomatous ulcer, not unlike a chancre. In the course of weeks or months swellings appear along the regional lymph vessels. These remain as subcutaneous nodules which are hard, somewhat tender, and vary in size up to that of a pigeon's egg. Some soften forming subcutaneous

abscesses. Both the nodules and abscesses may involve the skin, and irregular ulcers are formed. This is the form resembling tuberculosis verrucosa cutis, and frequently small lesions in the neighborhood suggest tuberculides.

The earliest lymphatic nodules feel like a cherry-stone imbedded in the skin. These grow, as mentioned above, and a fluctuating point develops, indicating abscess formation. The abscess presently ruptures externally forming a fistula, the cutaneous margin of which is red or purplish and jagged. Very little infiltration surrounds the abscess so that its walls readily collapse. *De Beurmann* calls this form "sporotrichosis in multiple disseminated subcutaneous gummata." Another variety resembles ecthyma, and still another kerion celsi. Involvement of the eye and lids has been noted in this country. At times there are periosteal abscesses.

Course. An acute form has been described. The majority of cases, however, are chronic, as mentioned above.

Varieties. Clinically, the varieties are those included in the text, namely, the subcutaneous, lymphatic, abscess forms, verrucous, gummatous, papulo-necrotic, granulomatous and ecthymatous. Etiologically, there are several forms determined by one of the eight strains of organisms, the important ones of which are those of *Schenck* and *Hectkoën*, and *Beurmann* and *Gougerot*.

Differential Diagnosis. In the last analysis the diagnosis depends upon finding the organism. This is difficult to do except culturally, or by animal inoculation. The conditions to be excluded and the methods to be followed parallel those enumerated in blastomycosis.

Etiology and Pathogenesis. The cause of the disease is a sporothrix. This organism grows in irregularly, mucoid, convoluted colonies on most media, but most luxuriantly on those containing sugar. Spores and septate granular mycelia characterize the minute appearance of the fungi. The organism is pathogenic to dogs, horses, mules and rats, and the serum of patients agglutinates the fungus, while reciprocal agglutination exists between sporotrichotic, and actinomycotic sera and the respective organisms. Likewise a positive percutaneous reaction can be produced in the sense of *Pirquet*.

Treatment. Iodides are specific, and local treatment is surgical when abscesses have formed.

Prognosis. With proper treatment the prognosis is invariably good.

Actinomycosis, or "Lumpy Jaw," is a granulomatous, ulcerous disease of the skin of the neck and over the inferior mandible. The bones are primarily involved and the cutaneous manifestations are secondary. The lesions are lumpy, hard in spots, fluctuating elsewhere, red or purple, and in places pustular and ulcerated. From the ulcers pus exudes containing yellow granules which microscopically are seen to be composed of colonies of ray fungi. At times, in connection with pulmonary actinomycosis, cutaneous lesions similar to those described above are seen on the chest. Large doses of iodides cure many cases. Untreated or recalcitrant cases are fatal, death being due to inanition or chronic sepsis.

Madura Foot, also known as mycetoma foot, morbis pedis entophyticus, endemic degeneration of the foot bones, etc., is a ray fungus disease of the foot, which is swollen, covered by abscesses, fistulae and granulomata, and the pus contains the mycetoma which Castellani has divided into yellow, black and red varieties, with fifteen sub-varieties. The disease is closely related, at least, to actinomycosis, and its treatment is identical therewith. Sometimes amputation is necessary.

Coccidoidal infections are extremely rare and resemble blastomycosis.

There remain two further forms of fungus infection to be described, which cannot readily be classified. One is *Erysipeloid* of *Rosenbach;* the other *Hairy Black Tongue.*

Erysipeloid is a mild inflammatory fungus disease occurring in people engaged in handling live animals, or their flesh. It is particularly common among fishermen. After some injury, a roseate, slightly indurated, edematous progressive lesion appears which suggests erysipelas, but which never becomes bullous. It is slightly painful, hot and tender, and its margins are not quite so abrupt as are those of erysipelas. (Chapter XXIII.) There are no chronic systemic symptoms, but occasionally the temperature rises a degree or so. The disease is probably caused by a streptothrix, one of the sub-varieties of discomycetes. It is harmless and readily cured by wet dressings.

Hairy Black Tongue, the clinical features of which will be described in another chapter (Chapter XXXIX), is probably caused by a saccharomycis.

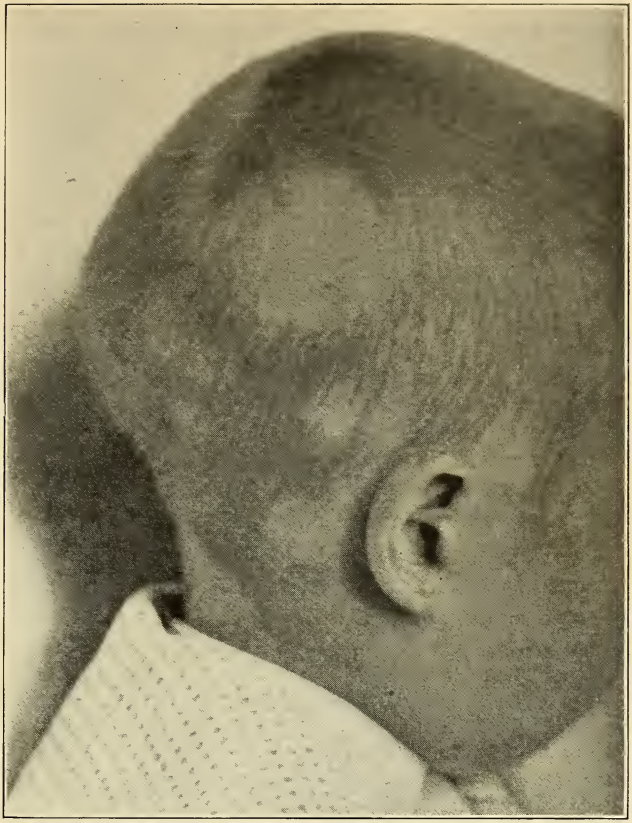

FIG. 46. MICROSPOROSIS

Note the circular, sharp areas of baldness. The scaling excludes alopecia areata; and the lack of atrophy excludes pseudo-pélade and lupus erythematosus. Stumps of broken hairs project from the involved areas.

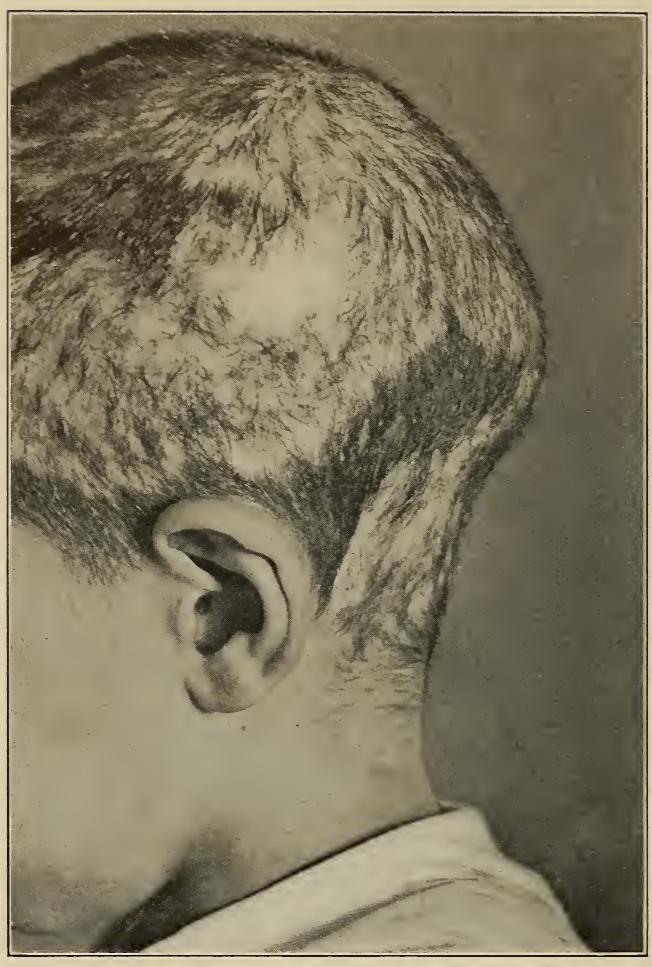

FIG. 47. TINEA CAPITIS

This is the large spore type of ring worm. The patches are irregular in size and shape; the hairs matted, irregularly twisted, and broken; and the scalp scaly or pustular.

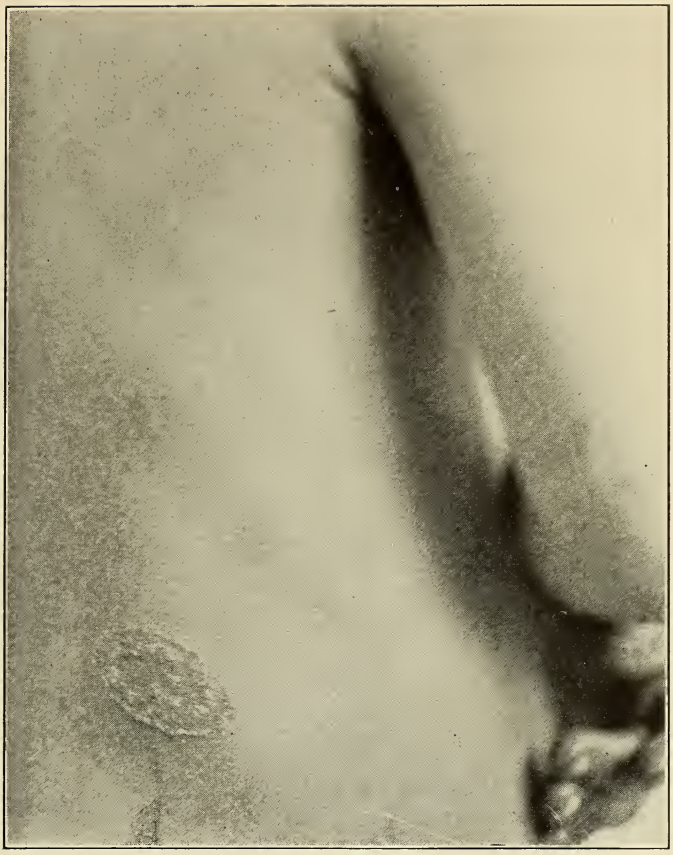

FIG. 48. TINEA CORPORIS

The ringed outline is characteristic. The ring may consist of papules, scales or vesicles, each element always being minute. The color is light red, pink or buff, and at times there are concentric rings. One or many lesions may be present and there is wide variation in size.

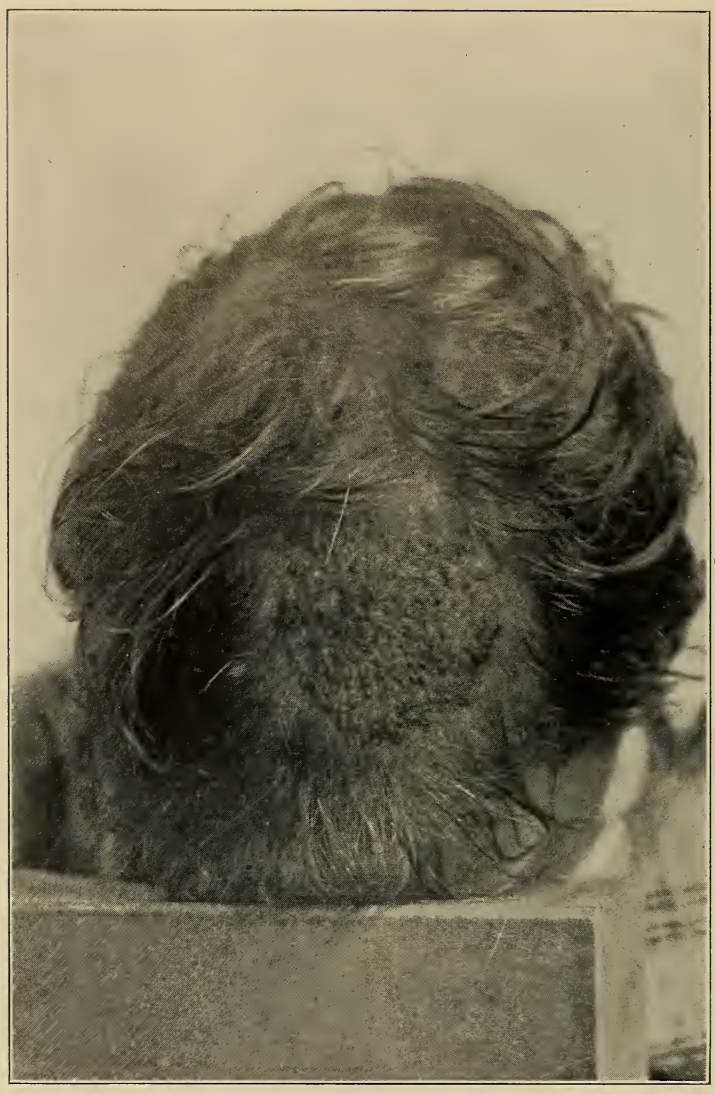

FIG. 49. KERION CELSI

This condition, otherwise called deep trichophytosis, is a granuloma provoked by pyogenic trichophyta such as infest live stock. The lesions are elevated red discs resembling plateaus of granulation tissue. The hairs are gone, or loose, and droplets of pus may be expressed from the follicles. Tinea of the beard belongs in this group.

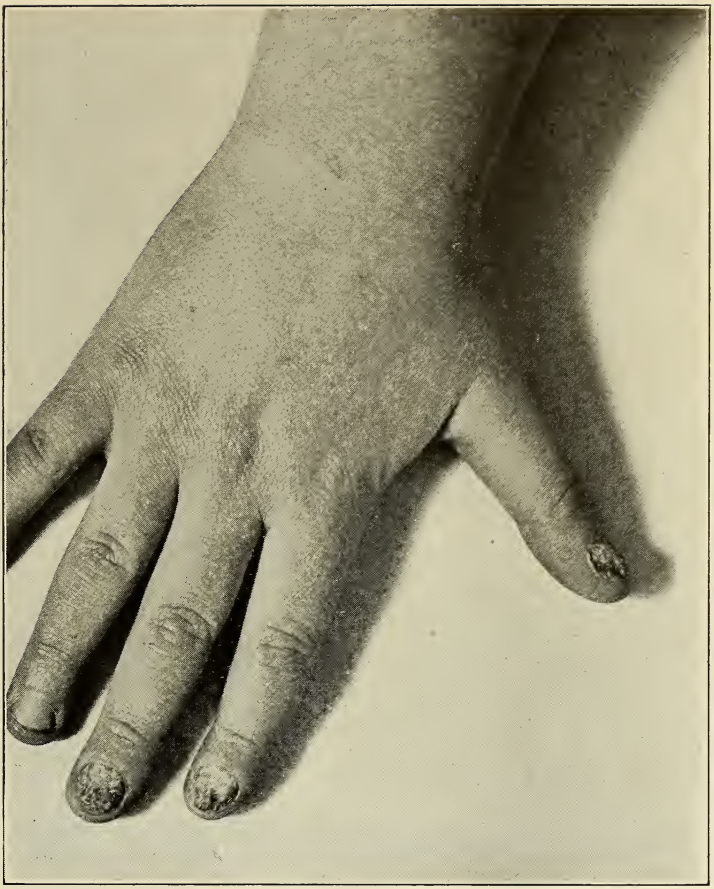

FIG. 50. TINEA OF THE NAIL

The nails are rough, pitted, lustreless and deformed. In psoriasis the nails show pin-point depressions, and in chronic dermatitis other evidence, elsewhere, of the condition exists.

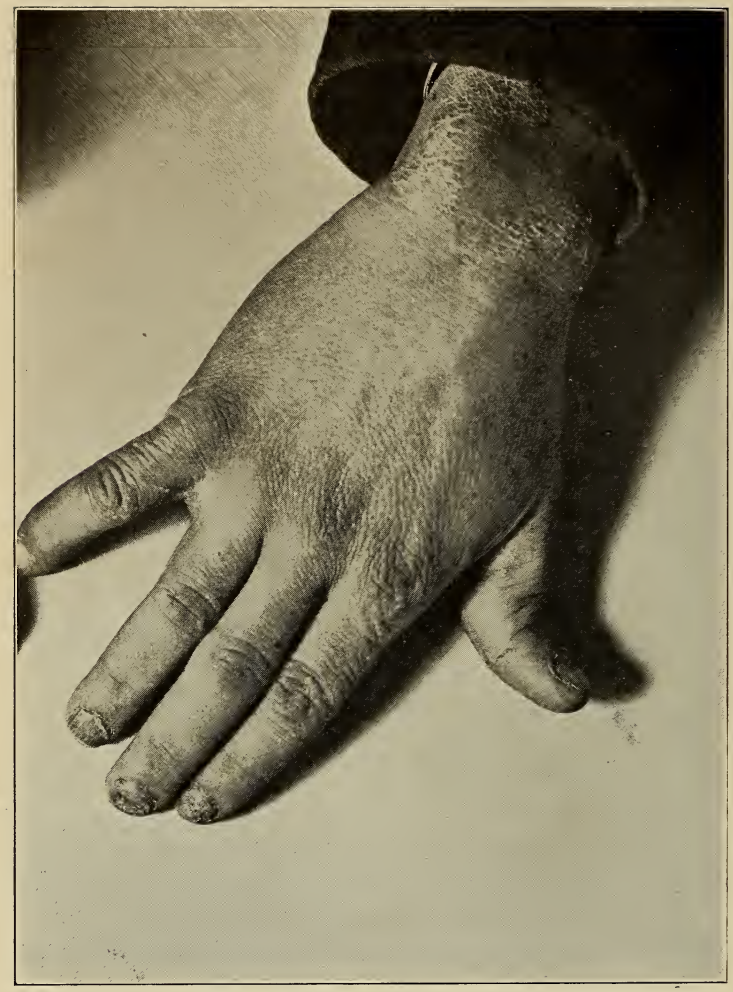

FIG. 51. DERMATITIS DUE TO FUNGUS INFECTION

The lesions often, as is here shown, do not look like ringworm, but are like those of any other vesicular dermatitis. A great many cases of what is called pompholyx are caused by fungi.

FIG. 52. EPIDERMOPHYTIA INGUINALIS

The cause of this disease is a special fungus favoring the groin, perineum, scrotum, and at times the hands, feet, and even other areas. In the groin it produces the picture of tinea circinata, but by rapid extension patches form, as here shown.

FIG. 53. TINEA OF THE BEARD

Hard, raised, purple, red or brown granulomatous masses, admixed with pustules, as shown on the chin, characterize this condition.

FIG. 54. FAVUS

The scalp shows cup-shaped saffron lesions; pure cultures, in fact, of the fungi. Elsewhere there are areas of atrophy. In this extensive case, there is complete baldness.

FIG. 55. BLASTOMYCETIC DERMATITIS

This disease varies in appearance. In America, the commonest form is here illustrated. It consists of numerous small abscesses, composing a vegetating mass as seen near the base of the thumb. The European type more closely resembles kerion.

CHAPTER XXIII

STREPTOCOCCUS AND STAPHYLOCOCCUS INFECTIONS OF THE SKIN

Streptococci cause impetigo contagiosa, ecthyma, erysipelas, solid edema of the face, and perlèche. Staphylococci cause sycosis nonparasitaria, or simple sycosis, impetigo of Bockhart, ecthyma, furunculosis, suppurative hydrosadenitis axillarum, carbuncle, infectious eczematoid dermatitis, dermatitis papillaris capillitii and the pustular phase of acne. Both organisms cause phlegmon, suppurative paronychia and pyodermites.

IMPETIGO CONTAGIOSA (FIG. 56)

Synonyms. Impetigo. German, Eiterflechte.

Definition. Impetigo contagiosa is a bullous dermatosis caused by a streptococcus (Fehleisen) which gains access to the epidermis through a solution in the continuity thereof.

Symptoms. The disease may arise spontaneously but is usually secondary, depending on inoculation through a minute epidermal injury. At first a pink macule appears which changes with increased exudation to a bleb, the roof of which is so flaccid that it soon collapses. Thus the bullous stage of the disease is rarely actually seen. The contents of the ephemeral bulla escape and dry into an irregular crust formed of hardened drops of honey colored serum. The crusts may be brown from a rich admixture of blood, or dingy from dust, scales or grease. After a few days the crusts fall leaving a pink epithelialized area corresponding in size to that of the original lesion. Should the crust be forced off too early a pink superficially ulcerated surface is seen which soon becomes crusted again and heals without scarring. Often impetiginous surfaces weep freely. The patches may be small or as large as a dollar, discoid, gyrate or bullous. The disease is commoner in children than in adults. It occurs anywhere on the body and may be localized or general. Occasionally there is slight itching or burning.

Course. The life of the individual lesion from onset to end is about ten days. Untreated, the disease may persist indefinitely. With proper care it is easy to curtail it.

Varieties. Some of the varieties of the malady depend on purely artificial linguistic distinctions. Thus impetigo gyrata is a form in which there is peripheral extension and central healing. Impetigo bullosa is determined by a longer survival than usual of the bullous roof; otherwise it is precisely like the ordinary form. Impetigo bullosa may also gyrate if the margin advances and the center heals.

An actual variety of the disease meriting special mention is the so-called pemphigus neonatorum, a poor name indeed since the disease is no pemphigus but a bullous form of impetigo affecting the new born.

Differential Diagnosis. Ordinary impetigo resembles no other skin disease. The bullous form simulates pemphigus (Fig. 17) from which it is to be differentiated by the nature of the crusts, by the presence of an underlying cause and by the fact that impetigo lesions are seated upon an inflammatory base and are flaccid, while pemphigus lesions have a non-inflammatory base and the bullae remain tense for a longer time. Varicella is to be excluded by the presence of crops of lesions, many of which are papular, and by the umbilication of the pustules. Impetigo of the scalp sometimes resembles ringworm and a differential diagnosis can be reached only when trichophyta are demonstrated microscopically.

Etiology. The one and only cause of impetigo contagiosa is a streptococcus. Everything written on the subject before, endeavoring to establish the pathogenic rôle of other organisms, is wrong. Staphylococci, ubiquitous on the skin surface, secondarily invade the pustules or crusts, but the exciting agent is always a special streptococcus. Predisposing causes are any itching dermatosis or any vesicle which may become inoculated. Thus, herpes dermatitis, prurigo, urticaria, scabies, pediculosis, etc., may be the underlying factor. A most common cause is pediculosis capitis, particularly in children. Next to this comes scabies and prurigo. The disease may be transmitted from an infected individual to a sound one and is likewise autoinoculable. Thus nursing mothers acquire it from their infected babies. Nasal discharges and purulent otitis media often furnish the organisms. Lesions similar to impetigo contagiosa are produced by the Klebs-Loeffler and pyocyaneus bacilli.

Treatment. Volumes have been inspired by this topic and therefore little need be said here. Above all, whatever the cause, repeated recourse to soap and water in itself frequently determines the cure. A two to ten percent. ammoniated mercury salve applied to

the lesions twice daily will cure the average case within a week. The crusts should previously have been removed with soap and water or olive oil. More persistent lesions should be painted daily with a twenty percent. solution of silver nitrate. Predisposing causes, notably lice and scabies, should be removed. In impetigo contagiosa of the bearded regions in men, frequent application during the day of a two percent. solution of salicylic acid in dilute alcohol, and nightly applications of two percent. ammoniated mercury cream produce a prompt cure.

Prognosis. This is always good.

ECTHYMA (FIG. 57)

Definition. Ecthyma is a pustular disease characterized by the formation of blebs which subsequently resolve into crusted ulcers.

Symptoms. Either in conjunction with impetigo contagiosa, independently thereof, or secondary to an itching dermatosis, blebs arise which soon exclude a brownish or yellow secretion. This rapidly dries into firmly adherent crusts which upon removal uncover a superficial ulcer, but deeper than in impetigo. The floor is bright pink, the margins less than one millimeter deep and the entire lesion is surrounded by a red zone which gradually fades into the normal skin. The ulcers are rarely larger than a dime, the areola is rarely broader than half an inch, making the entire maximum diameter about one and a half inches. About four weeks are required for the entire evolution and involution of the lesion which heals under the crust leaving a slightly depressed scar, at first pink and later white. At times the ulcers are gangrenous, in which event the resultant scars are large and more depressed. Many lesions may be seen, but as a rule there are only a few, and the disease tends to be restricted to the lower extremities and buttocks in children. Tension, burning and pain are present but these symptoms are not marked.

Course. The course of the disease coincides with the persistence of the predisposing factor. That of any lesion is about four weeks.

Varieties. There are none.

Differential Diagnosis. Ulcers with adherent crusts on the buttocks and lower extremities of children suffering with impetigo or some itching dermatosis are usually ecthymatous. Only crusted syphilides are simulated, the latter being infiltrated and the Wassermann reaction being present. Impetigo itself differs from ecthyma

in its superficiality, this difference in degree constituting the only distinguishing feature between two diseases otherwise identical both in significance and causation.

Etiology. The predisposing cause of ecthyma may be impetigo, pediculosis, scabies, prurigo, urticaria or any other itching dermatosis. The exciting cause is the streptococcus.

Treatment. The treatment is precisely like that of impetigo contagiosa.

Prognosis. No difficulty is found in curing the malady if the underlying cause has been controlled.

ERYSIPELAS

Synonyms. St. Anthony's fire. German, Erysipel, Rothlauf. French, La Rose, Erysipèle.

Definition. Erysipelas is an infectious disease of the skin characterized locally by swelling, redness and heat of the affected parts, and by systemic reaction.

Symptoms. After a prodromal period of two or three days during which malaise, loss of appetite, chilliness, chills and rigors may be present, a red spot appears on the skin near a previous injury such as a scratch, cut, surgical wound or upon the umbilicus in the newborn. The red spot increases in size, is swollen, glazed, hard and slightly tender, and sharply outlined by a steep margin terminating abruptly against the healthy skin. No outlying spots are seen. The original lesion progresses peripherally, maintaining the character already depicted and usually stopping at the hair lines. At times, bullae, vesicles or gangrene arise. At the end of a few days to a week the spot fades and the fever disappears fairly abruptly or lytically. The skin peels and finally becomes normal.

Course. The course has been partly outlined above. At times rheumatic symptoms appear; rarely, sepsis, nephritis, purulent otitis media, meningitis or local abscesses arise. A large proportion of the complicated cases end in death.

Varieties. Acute, chronic, recurrent and ambulatory erysipelas, as well as solid edema of the face, constitute the varieties. Acute erysipelas has been described. Chronic erysipelas is a low grade, afebrile infection lasting weeks, months or years. Recurrent erysipelas is rather a series of diseases than one disease just as recurrent coryza is not one disease. It represents a peculiar susceptibility. Ambulatory erysipelas differs from the common type in that the lesion spreads unboundedly while its older portions heal, thus trans-

gressing the ordinary limitations characteristic of the malady. Solid edema of the face is a condition created by recurrent attacks of mild erysipelas maintained by a low grade of infection from a persistent source. Nasal fissures serve as an example. Ultimately a productive inflammation develops causing permanent dilatation of the lymphatics.

Further forms of the disease are the nonsurgical and the surgical. Save for the latter, practically all varieties of erysipelas are facial, some nasal injury or disease, or a scratch, being the portal of entry. Surgical erysipelas develops near infected wounds anywhere on the body.

Differential Diagnosis. The constitutional symptoms, the hard, glazed surface of the lesion, the characteristic margin and absence of outlying islands of infection are the diagnostic points. Acute dermatitis and facial zoster may resemble vesicular or bullous erysipelas. When the salient features of the disease are considered, however, confusion is unlikely. Chronic erysipelas resembles rosacea (Chapter XXXV) and a special form of lupus erythematosus (Chapter XXV), but other evidences of the latter rule out erysipelas. Rosacea is characterized by persistently dilated veins and less infiltration than erysipelas.

Etiology. The cause of erysipelas is the streptococcus of Fehleisen which gains access to the body through an abrasion of the skin.

Treatment. When high fever is present the patient should be put upon salicylates with suitable stimulants in cases of cardiac depression. The local treatment consists of the use of wet dressings. For these may be employed Burrow's solution in ten percent. dilution applied as in the treatment of acute dermatitis (Chapter IX), or a hot saturated magnesium sulphate solution. If the former type of dressing is employed the fomentation should be continued without interruption day and night except when the procedure interferes with sleep. If hot wet dressings are employed, they should be applied for fifteen minutes every hour, followed by a lotion of the calamine and zinc type. Such a lotion may also be used in conjunction with cold wet dressings if the latter are interrupted while the patient rests. The purpose of this treatment is simply to reduce the congestion. In general the cold fomentations afford more relief and work more rapidly than the hot, although isolated instances are encountered in which the latter seem better. After the inflammation has subsided desquamation begins and any grease or oil overcomes the dryness of the skin. Icthyol ointment, iodine and similar

remedies have been employed, but they do little good, make the patient uncomfortable and have no rational basis. Anti-streptococcus serum has been used, but it possesses little that is commendable. Isolation of the erysipelatous is as senseless, although it is still practiced, as the isolation of a patient with a streptococcous empyema, appendicitis or middle ear infection. Such cases are not segregated although the pus from these conditions is full of streptococci. Ordinary asepsis will render erysipelas a safe disease from the standpoint of transmission.

Complications of the disease have their special therapeutic indications, a discussion of which has no place in a work on dermatology.

Prognosis. Save in the aged, or in cachetic adults and babies, the prognosis is good. Complicated or hyperpyretic cases often end in death.

PERLECHE. Perlèche is a disease of the labial canthi in children, chiefly infants. It is a rhagadiform eruption of both commissures. A whitish film covers them and the process often spreads toward the mesial line of the lips and often extends to the skin. It is probably caused by the streptococcus plicatilis and can readily be healed by the use of silver solution and ammoniated mercury ointment.

FOLLICULITIS SUPPURATIVA BARBAE STAPHYLOGENES (FIG. 58)

Synonyms. Sycosis; Non-Parasitic Sycosis; Sycosis Vulgaris; Sycosis Staphylogenes. German, Bartfinne, Bartflechte. French, Sycose.

Definition. Folliculitis suppurativa barbae staphylogenes is well described by its name. Its most popular title, non-parasytic sycosis, is absurd for the staphylococcus is certainly a parasite.

Symptoms. The disease usually begins acutely with an eruption of papules and pustules on the bearded region. It is thus commoner in men than in women or children. At times the pubis and axillae, and rarely the eyebrows and scalp, are involved. The lesions are follicular and perifollicular, a hair usually piercing the pustule. Upon withdrawing the hair a drop of pus is found adherent and a crateriform opening oozing pus remains behind. The lesions are often clustered and covered by crusts and the skin is thickened, red, uneven, itchy or painful. Thus the picture of pustular dermatitis is closely simulated. Successive crops occur.

In long standing cases an atrophy develops, the skin becoming stretched over the malars, the cheeks being sunken below. This

imparts a skull like appearance to the face. At times there is permanent alopecia but this is unusual since, on the whole, the hair papillae withstand the process.

When the scalp is affected, the process closely resembles pustular dermatitis. The disease is often associated with furunculosis. Coryza, both acute and chronic, not infrequently inaugurates the picture, or an injury, or otitis media or ordinary dermatitis, determines the onset.

Course. In general the disease is acute and is rapidly terminated by proper treatment. It may, however, become chronic, lasting months or years. The chronic form may be pustulo-papular or pseudo-atrophic as already described.

Varieties. Some of the clinical varieties were mentioned in the previous paragraph. A form exists in which the pustules form a ring enclosing an atrophic center. This is called lupoid sycosis and by Unna was renamed ulerythema sycosiforme.

Differential Diagnosis. The acute form resembles folliculitis barbae suppurativa trichophytica (Fig. 53), acne vulgaris and halogen acne. The fungus form is more indurated, often associated with other forms of ringworm and the fungi are demonstrable. Acne is not confined to the bearded areas and comedones are present. The iodide and bromide acne is more varicelliform and a history of drug ingestion may be elicited.

Chronic forms resemble lupus vulgaris and lupus erythematosis. Lupus vulgaris, however, is less pustular, is cicatricial and not confined to the hairy areas. Lupus erythematosus is never pustular but scales, and in color is purplish. In fact, it is only in the atrophic stages that the two processes are similar. If no other evidence of either disease is present a correct estimation of the cause of the atrophy is almost impossible.

Etiology. The immediate cause of the disease is the staphylococcus pyogenes aureus or albus. Contributing causes are coryza, injuries, direct infection through combs, brushes and razors at barber shops. In this, as in all pilosebaceous diseases, disturbed sugar metabolism is often present. It is obvious, since people once infected are readily reinfected, that there must be a predisposition.

Treatment. The treatment of staphylogenic sycosis must be directed toward the predisposing and exciting cause. Thus, in cases following coryza, treatment of the nose is indicated. Patients who shave too closely must be cautioned, and if there is evidence of

disturbance of carbohydate metabolism the starch and sugar intake must be limited and a suitable diastase administered.

The local treatment consists of the removal of the pustules and infected hairs, the use of wet dressings, lotions and salves. The pustule may be opened with a comedo extractor, the hairs removed with depilation forceps, or if the involved area is extensive five Holzknecht units of Roentgen rays should be given to cause depilation. The patient should also be directed to shave lightly once a day. When indicated, the employment of Roentgen therapy and iodine cataphoresis is valuable. The medicaments employed are either a two percent. salicylic acid and resorcin lotion in alcohol, sponged on several times a day and followed by talcum, or the use of very weak bichloride solution either sponged on or used in the form of fomentations. At night a three percent. white precipitate or one percent. yellow oxide of mercury ointment may be employed. The patient must be warned that the lotions suggested may hurt for a few moments after their application. In the atrophic stage nothing can be accomplished by therapy.

Prognosis. The average case can easily be cured. Now and then, however, great obstinacy is encountered, particularly when the disease depends upon a local predisposing cause such as coryza.

IMPETIGO OF BOCKHART

For the sake of simplicity this condition may well be considered as a sub-variety of the preceding. The disease is a superficial follicular infection caused by staphylococci. It occurs anywhere on the body, at any age and in both sexes. It may be primary, but is often secondary to a preexisting and usually itching dermatosis. The affected areas are studded with minute pustules, many of which are pierced by lanugo or other hairs. The treatment need never be drastic. Usually the lotions and salves already mentioned in the previously described disease will promptly cure an outbreak.

FURUNCULOSIS

Synonyms. Furunculus, Boil; French, Clou; German, Eiterbeule, Eitergeschwür, Blutbeule.

Definition. A furuncle is a staphylogenic infection starting in a hair follicle, about which a local cellulites develops which terminates in necrosis. A succession of such lesions constitutes furunculosis.

Symptoms. At first a painful induration develops. This is red, feels hot, throbs, and is tender. It gradually increases in size, each of the above features augmenting, and often the neighboring tissue though not inflamed is edematous. The center softens as pus forms, and a yellow, ever increasing point develops. Frequently a hair pierces the center. Finally the abscess ruptures, pus which is creamy and yellow or blood tinged escapes, and after a day or two a necrotic plug follows. Thereupon the lesion heals, leaving a purple hyperemic patch which ultimately fades, the skin again becoming normal in tint. A small scar remains if the slough was small, a larger scar if healing took place by granulation. The face, arms, body folds and neck are most frequently affected, but any area may be. Not uncommonly mild febrile symptoms accompany a developing boil, which vanish after its evacuation.

Course. There may be a single boil, its life being no longer than ten days, or a series of boils giving the picture of furunculosis. Or there may be recurrent attacks. As a rule the cases are uncomplicated, but occasionally sepsis with its usual features develops.

Varieties. A variety of furunculosis is a disease of the axillae which does not originate in the hair follicles but in all other respects resembles the chronic form. This is hydrosadenitis suppurativa axillarum, or abscesses of the axillary coil glands. This is a staphylogenic infection of the deep sweat organs and is extremely painful and often accompanied by profound systemic symptoms. There may be an associated axillary furunculosis of the hair follicle type. Etiologically, prognostically and clinically the two conditions are identical.

Differential Diagnosis. A boil resembles nothing else save an infected cyst and the difference between the two is merely academic. Perineal furuncles simulate rectal abscesses from which they must be differentiated. It is possible for a boil to suggest the appearance of a chancre but the demonstration of spirochetes should prevent error.

Etiology. The cause of furunculosis is the staphylococcus. The hair follicle and adjacent sebaceous glands are infected, the inflammation involving the immediate connective tissue. Predisposing causes are diabetes, starch indigestion, hyperglycemia, inanition, infantile marasmus and weakness following serious illness. Local predisposing causes are itching dermatoses and excessive sweating. The latter is particularly the cause in the axillary form.

Treatment. The treatment of furunculosis may be divided into

three objectives, the abortion of a developing furuncle, the cure of a developed furuncle and the control of the predisposing factors, both local and general.

When a furuncle is developing abortion is possible in just one way, by the use of cold wet dressings. Any mild antiseptic may be employed, such as boric acid, aluminum acetate and very weak bichloride solutions, or the ice bag may be used. Wet dressings should be applied constantly, changed at short intervals and never be allowed to reach body temperature. They should be bulky and not covered by oiled silk or rubber tissue which prevent evaporation. The object of wet dressings in reducing acute inflammations of the skin is not widely enough understood. The coolness of the fomentations and the evaporation constitute their effectiveness, an aim that would be defeated by using impervious substances to cover them. Warm moisture favors the flourishing of the bacteria.

When a furuncle has passed the abortable stage the treatment again has various objects, the first being to hasten the softening down, the second to favor evacuation of the abscess contents. To hasten softening, hot wet dressings are useful. They should be as hot as tolerable, without risking burns or scalds, and should be applied constantly until definite pointing has taken place. Hot flaxseed poultices are as good as wet dressings, and hot water bags applied over moist flannel are also excellent and simpler and cleaner than the hot poultices. Small furnucles may early be brought to complete softening by the use of twenty percent. salicylic acid, or soap and salicylic acid plaster.

After the furuncle is soft it should be punctured with a sharp sterile toothpick, and such of its contents as flow out easily should be expressed with the gentlest pressure. Cold wet dressings should then be applied. Usually by the day following the puncture, but rarely later than two days after, even large furuncles will empty themselves and the necrotic plug or "core" will escape. Thereupon prompt healing takes place.

A furuncle should never be incised unless there is an associated lymphangitis with regional adenopathies, or unless there are marked systemic symptoms. Furuncles of the upper lip, however, should be incised early and freely as they are often dangerous. The time honored crucial incision for all furuncles is an anachronism, as the healing is slow, the scar unsightly and the method unwarrantedly drastic. In ninety-nine cases out of a hundred, simple puncture will suffice.

The general treatment of furunculosis consists first of removing the predisposing cause. Thus scabies, pediculosis, dermatitis and urticaria must be cured. Should filth be the cause, bathing must be urged.

Removal of the general predisposing cause must be attempted after its nature has been determined. Thus gastric, fecal, blood and urine analyses should be made. In diabetes, gastric hyperacidity, starch indigestion and hyperglycemia, the indications are obvious. In marasmus, debility, post-febrile weakness, etc., general dietetic and tonic treatment will suggest themselves.

The best general therapy for furunculosis lies in the use of staphylococcus vaccines. My own experience leads me to prefer polyvalent stock vaccines of which an injection should be given every five to seven days. The initial dose is four hundred million and it should be gradually increased to a billion. These injections are continued until a fortnight has elapsed without the appearance of new lesions. It is ideal to follow this by six fortnightly and six monthly injections. I have seen only one case fail to respond to this line of therapy within three months, or recur if the after treatment was sustained. Incidentally, a massive vaccine injection, 500 to 700 million to a billion will often abort a developing lesion, or hasten its softening.

Radiotherapy has its followers. The application of four Holzknecht units of Roentgen rays will often cure the axillary form and diminish the sweat secretion. The Kromayer light is advocated by some.

Prognosis. The prognosis of simple furunculosis is good. Furunculosis based upon diabetes is obstinate. When the upper lip is involved, or when there is systemic involvement, the cases not infrequently terminate in death.

CARBUNCLE

Synonyms. Carbunculus, Anthrax; French, Anthrax; German, Brandschwur.

Definition. A carbuncle is an acute infection of a restricted area of the skin and subcutaneous tissue, larger, however, than the area occupied by a furuncle which it otherwise resembles. The name anthrax is misleading as it is never caused by the bacillus anthracis.

Symptoms. Often the disease is preceded by marked systemic symptoms, malaise, chilliness, chills, rigors, fever or even hyper-

pyrexia. Thereupon the lesion develops. It is first a hard, red, tender indurated mass, the size of a walnut or egg. Gradually softening takes place and pus exudes from one or more apertures. The involved area is undermined and the entire mass is cribriform, inflamed and fluctuating. A slough forms which escapes in toto. Healing is gradual and a large scar remains. As the local process wanes the systemic symptoms, often very severe, abate. Any part of the body may be involved but the disease is commonly manifested at the nape of the neck.

Course. This may last from two to five weeks.

Differential Diagnosis. Nothing else simulates carbuncle.

Etiology. This parallels that of furuncle. Diabetes is a very common predisposing cause.

Treatment. The treatment consists of wet dressings as in furunculosis, and wide incision and drainage. Ormsby favors the use of pure phenol injected into the mass. This causes a softening of the entire lesion. Vaccines are useless.

Prognosis. This is good save in diabetes and in severe systemic complications.

ACNE VULGARIS AND DERMATITIS PAPILLARIS CAPILLITII OR ACNE KELOID

These, in their pustular phases, are analogous in their pathogenic significance to staphylogenic sycosis. Certain particular elements, however, come into consideration in these conditions which render it important to group them with seborrhea (Chapter XXXV). Thus their description will be deferred.

Gayle. A rare special form of staphylogenic inflammation is caused by the staphylococcus hemorrhagicus. This disease is acquired by those engaged in flaying ewes which died of a peculiar puerperal infection. The human lesion is a slate colored bleb containing serum.

Infectious Eczematoid Dermatitis is a disease characterized by many features of weeping dermatitis and exuding a seropurulent discharge. Ordinarily the illness arises in skin that has been bathed for a prolonged period in pus. Thus in patients with discharging otitis media or chronic purulent coryza the resistance diminishes in the skin constantly wet with discharge. A dermatitis (Chapter IX) is precipitated, the vesicles of which become infected, probably with an organism derived from the exciting purulent discharge. The skin is edematous, scaly, pruritic and often difficult to heal. Its normal sites are the ears, scalp, lips, nose and cheeks. The treatment is a combination of that of acute dermatitis and impetigo contagiosa.

Phlegmon and Pyodermas are caused either by the streptococcus or

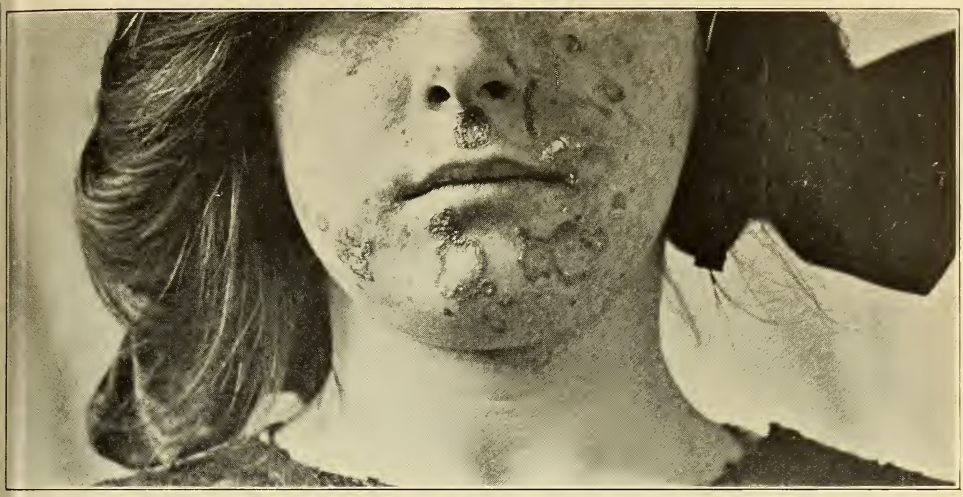

FIG. 56. IMPETIGO CONTAGIOSA

The lesions of impetigo contagiosa are fragile bullae which rapidly rupture and crust, as on the upper lip. At times circinate progress of the margins occurs, as on the chin. On the left cheek is seen a recently ruptured bulla.

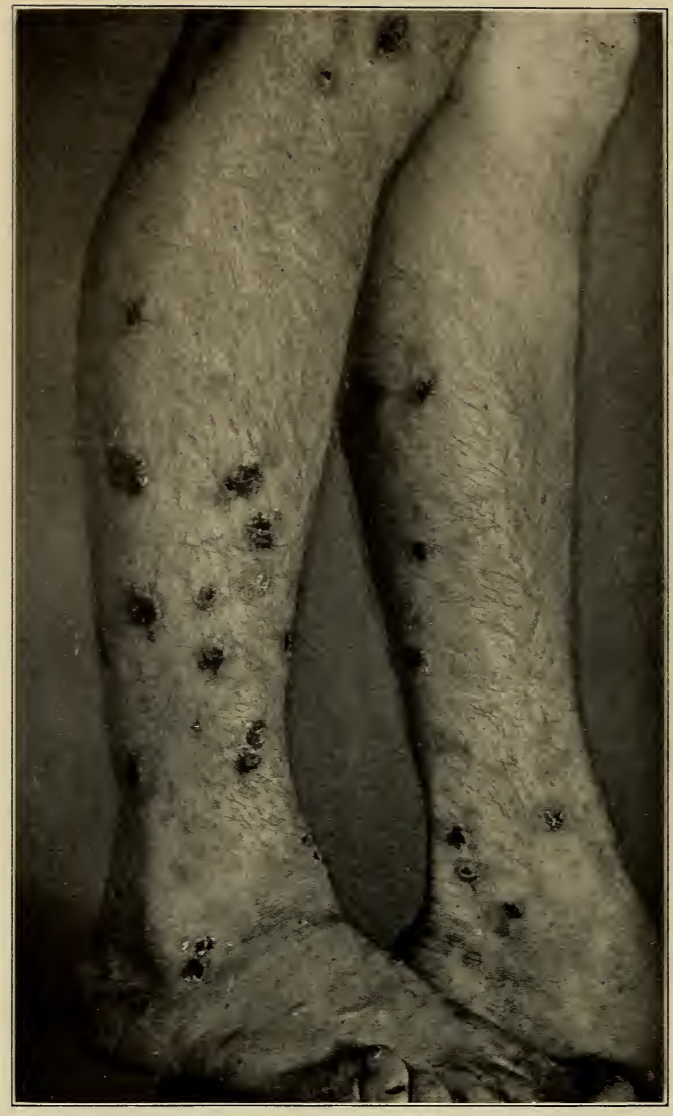

FIG. 57. ECTHYMA

Ecthyma is impetigo contagiosa, but the lesions are deeper. They are caused by streptococci, and look like shallow ulcers which are crusted with dried blood or pus. The floor of an ecthymatous lesion is vivid red and moist. Ecthyma is due to inoculation by scratching, regardless of the cause of the itching.

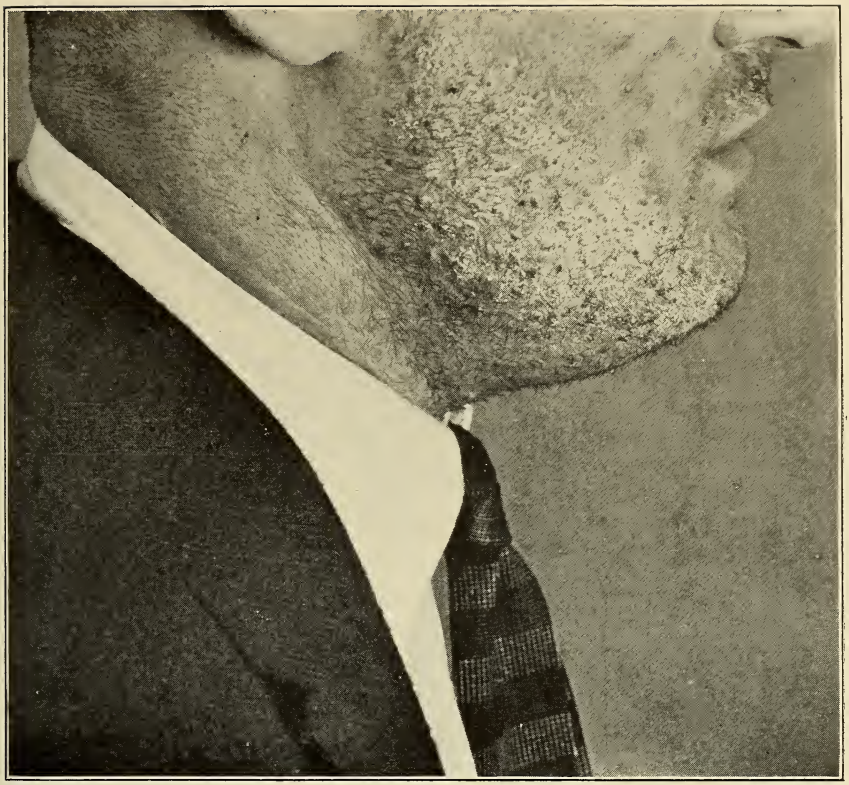

FIG. 58. FOLLICULITIS BARBAE STAPHYLOGENES

This condition, otherwise known as simple sycosis, occurs as pustules about the beard follicles. When these rupture, crusts form. In trichophytic folliculitis, more induration is present.

staphylococcus. The former is an infection of the subcutaneous tissue secondary to boils, carbuncles, erysipelas or any other infectious skin disease. Cellulitis develops, and therapeutically the condition is surgical.

Pyodermas, or pyodermites, as the French term them, constitute a loose concept implying the aggregate of all pustular skin infections due to the two common pyogenic cocci. Thus everything in this chapter is included. Often enough combined pictures are formed, such as axillary hydrocystadenitis and furunculosis, impetigo and ecthyma, etc.

Another variety of pyoderma not included in the text books is a rare form seen only in sepsis. Infarcts lodge in and below the skin. They appear suddenly, are painful, break down and suppurate, forming pustules, or furuncular lesions, or localized phlegmons. Their treatment is local and medical or surgical, as the case may be. The prognosis is one with that of sepsis.

In this connection it may be added that anatomists, butchers, surgeons and those engaged in the care of the sick, or in trades involving the handling of animal carcasses or their products, acquire infections known as dissection wounds, or post-mortem pustules. The lesions are not unlike those of gayle but are often serious, leading to cellulitis or sepsis. As a rule the staphylococcus or streptococcus causes the infection, but tetanus, anthrax or equinia may be similarly acquired (Chapter XXIV).

CHAPTER XXIV

MISCELLANEOUS BACTERIAL INFECTIONS

The miscellaneous bacterial infections of the skin are bacillary or coccygenic in origin, or of variable or presumptive bacterial causation. Those caused by bacilli are anthrax, malleus, diphtheria and rhinoscleroma; that caused by a coccus is blenorrhagic keratoderma; those presumably bacterial are granuloma pyogenicum and gangosa; and those due to various bacteria are balanitis errosiva et gangrenosa and dissection wounds. Although all of these diseases are rare their importance is sufficient to warrant brief reference.

ANTHRAX

Synonyms. Malignant Pustule; French, Charbon; German, Milzbrand.

Definition. Anthrax is an acute infectious disease, caused by the anthrax bacillus, which gives rise to a furuncle. The latter is a primary lesion and the starting point of a general sepsis.

Symptoms. In from twelve to seventy-two hours after infection a macule develops. This rapidly becomes an itchy blister or bleb containing bloody serum. It breaks, crusts and is surrounded by a red halo near which satellite pustules arise. The lesion is stony hard and involves the deep tissues while an inordinate edema develops even as far as a foot away from the malignant base. Local adenopathies appear. At first there is low fever; later hyperpyrexia. Often there are chills and the patient presents a picture resembling typhoid, sepsis or meningitis. Pus from the lesion, and often the circulating blood, contains anthrax bacilli.

Course. The disease starts acutely. It ends in recovery if the process becomes localized. In this event the lesion behaves as would any furuncle. All cases in which sepsis occurs die within five to ten days.

Varieties. There is but one form.

Differential Diagnosis. The character of the lesion, the extensive edema and the presence of the bacilli are the diagnostic points Ordinary furuncles may be simulated for at times they too cause a marked edema, and on the upper lip may prove to

be as serious as anthrax. Bacterial study will differentiate the two.

Etiology. The cause is the anthrax bacillus. The mode of transmission is vocational; hence herders, ranchmen, tanners and butchers are oftenest infected. Lack of asepsis in caring for the affected, or carelessness at operations or autopsies favor transmission. At times, unwitting contact with infected material or people suffices to convey the malady.

Treatment. Early recognition of the disease is essential to successful therapy, if indeed the patient can be saved. Cauterization, or wide excision of the pustule are indicated and a specific serum exists which is efficacious.

Prognosis. This is always serious.

Equinia. Malleus, Farcy, Glanders; French, Farcin or Morve; German, Rotz.

This is a highly infectious and usually fatal systemic disease with cutaneous manifestations. It is caused by the bacillus mallei. There are acute and chronic forms. The acute variety starts with some systemic manifestations, rigors, chills, diarrhoea, hyperpyrexia and, at the site of infection, an erysipelas-like surface which rapidly becomes bullous, pustular, or ulcerous. The discharge from these lesions, as well as from the tear ducts and nasopharynx, is full of bacilli. They are also present in the blood. Local glandular enlargement and lymphangitis are marked. The chronic variety starts acutely, or is indolent from the first. It resembles variola or pemphigus, the blebs often ulcerating and their discharge being highly infectious. The glands may remain unaffected. The cases are usually fatal, the acute ones promptly so, while the chronic ones may drag on for months or even years. Therapy is of little avail and should be systemic.

Diphtheria of the Skin. Cutaneous diphtheria occurs in two forms, either as an infection of wounds, or as a spontaneous condition. These forms are primary. Or, it may occur as a mild superficial infection by extension of nasopharyngeal or middle ear diphtheria. The primary form is characterized by an ulcer or gangrene coated with a diphtheritic membrane, and its clinical and prognostic significance parallel those of ordinary diphtheria. The secondar form resembles impetigo or ecthyma near the nose, mouth or external auditory canal and is seen chiefly in children who harbor attenuated Klebs-Loeffler bacilli in the respective secretions. Both the primary and secondary forms may give rise to eruptions resembling dermatitis herpetiformis or pemphigus. The primary variety is serious; the secondary type is not, though the bullous type, whether accompanying the primary or secondary form, may be serious. The diagnosis depends upon isolating the bacteria as one does in suspected pharyngeal diphtheria. The treatment is that of ordinary diphtheria, namely antitoxin injections. The best local agent is ammoniated mercury salve. A practical point to be emphasized is that in children with impetigo due to chronic coryza or otitis media a search should invariably be made for the diphtheria bacillus.

Gangosa. This is a rare ulcerative granuloma of the nasopharynx. It is a tropical disease and presumably caused by a bacillus resembling that of diphtheria.

RHINOSCLEROMA (FIG. 59)

Synonyms. The term is universal.

Definition. Rhinoscleroma is a chronic infectious granuloma of the nasopharynx, nose and lip, caused by the Frisch bacillus.

Symptoms. At first small nodules appear on the septum of the nose. These increase in number and size until a diffuse, elevated, hard mass develops involving the entire nose, adjacent portions of the lips, often all of the structures of the mouth and the pharynx. A stony hardness arises and the mass, though painless, is tender. Its color is ivory white, red or purple and its surface is glazed and often vascular.

Course. This is slow and chronic, extending over years.

Differential Diagnosis. The lesions remotely simulate tuberculosis, gumma and sarcoma. Tuberculosis is excluded clinically by the absence of ulceration, by the hardness of the lesion, its extent, absence of tubercles and absence of the tuberculin reactions. Syphilis is excluded by the first two features and the absence of the Wassermann reaction. These two diseases with sarcoma, which clinically may closely resemble rhinoscleroma, are further excluded by histological examination. A section of rhinoscleroma presents the microscopic features of an ordinary granuloma plus three pathognomonic traits, the presence of the bacilli, Mikulicz's cells and Russell's bodies. The bacilli are elongated, encapsulated organisms clustered either in the collagen fibers or in structures known as Mukulicz's cells. The latter are frothy areas taking a light stain, and they are two or three times the diameter of a lymphocyte. They have no nuclei and are probably small lymphocytes filled with bacilli. Russell's bodies are circular hyalin end products of collagenous degeneration, and although found in many other skin lesions, they are most numerous in rhinoscleroma.

Treatment. X-rays, excision and plastic surgery constitute the modes of attack.

Prognosis. The disease, though slow and benign, is usually incurable and if temporarily controlled is likely to recur. Death, in extreme instances, is not due to the disease itself but to stenosis caused by the mass in the upper respiratory passages.

Etiology. The Frisch bacillus causes the disease, most cases of which have arisen in south-western Russia.

Balanitis Errosiva et Gangrenosa is an unusual inflammation of the prepuce and glans which travels thence phagedenically. The ulcer is undermined, has a granulated surface and shows no tendency to heal. It is possibly caused by a Vibrio, but possibly by a spirochete of which the Vibrio is a stage or phase. It may be obstinate but usually is cured by hydrogen peroxide, since the exciting agent is an anaerobe. Although the disease is in the main venereal it may develop upon surgical wounds.

Nosocomial Gangrene or Noma is a buccal gangrene which usually follows measles. (Chapter VII.)

keratoma blenorrhagicum

Definition. This disease is characterized by the formation of hyperkeratotic lesions in gonorrheal subjects.

Symptoms. There are local and generalized forms, the latter being the rarer. The lesions are minute, isolated or confluent, yellow or yellowish waxy pustules mainly found upon the leg and foot. A dense scale surmounts them and their arrangement suggests a relief map. Usually they accompany arthritic types of gonorrhea and often develop under immobilizing dressings over the joints.

Course. The disease is an intercurrent phenomenon of gonorrhea and disappears with the latter.

Varieties. The local type affects the hands, feet and nails, sparing the palms and soles. The general type appears anywhere except on the head and neck, but favors the legs and forearms.

Differential Diagnosis. A papular, horny dermatosis in a patient with gonorrhea is likely to be blenorrhagic keratoma. Syphilis and psoriasis may be simulated, but the absence of the Wassermann reaction and adenopathies and the history of the case exclude these two diseases.

Etiology. Gonorrhea is a causative element although the gonococci have not yet been found in the lesions. It is held by many that the casts employed for gonorrheal arthritis have much to do with the development of the lesions.

Treatment. The treatment is that of gonorrhea and the local use of resorcin or salicylic acid creams or lotions.

Prognosis. A cure is always to be expected.

granuloma pyogenicum (fig. 60)

Synonyms. Bothryomycosis, Pseudo-Bothryomycosis, Granuloma Telangiectaticum.

Definition. This is a disease characterized by pedunculated

tumors from one-fourth to one-half an inch in diameter which arise upon a previous injury.

Symptoms. In the early stages the tumors are red, moist and painless. They look like granulation tissue, are crimson, bluish or brown and though usually lentil shaped or roughly spherical, they may be flattened down like mushrooms. As a rule there is but one tumor. It secretes a foul, seropurulent matter and the surface may be smooth or contain pus filled crypts. At times the lesion resembles a raspberry. The fingers, toes, hands, feet, lips, chin, cheek, umbilicus, back and shoulders are the favorite sites.

Differential Diagnosis. Papillomata when inflamed or ulcerated resemble granuloma pyogenicum. A microscopic examination would clear this up as the lesion has the structure of granulation tissue.

Etiology. An injury precedes the lesion. Presumably it is an infectious granuloma caused by an unidentified organism. At first is was regarded as a human form of bothryomycosis, but this view has been abandoned and the majority of writers agree, although without final proof, that the staphylococcus aureus is at fault.

Treatment. Excision and cauterization of the base, or the Roentgen rays will cure the disease.

Prognosis. The prognosis is good and although the lesions may recur they can always be ultimately cured.

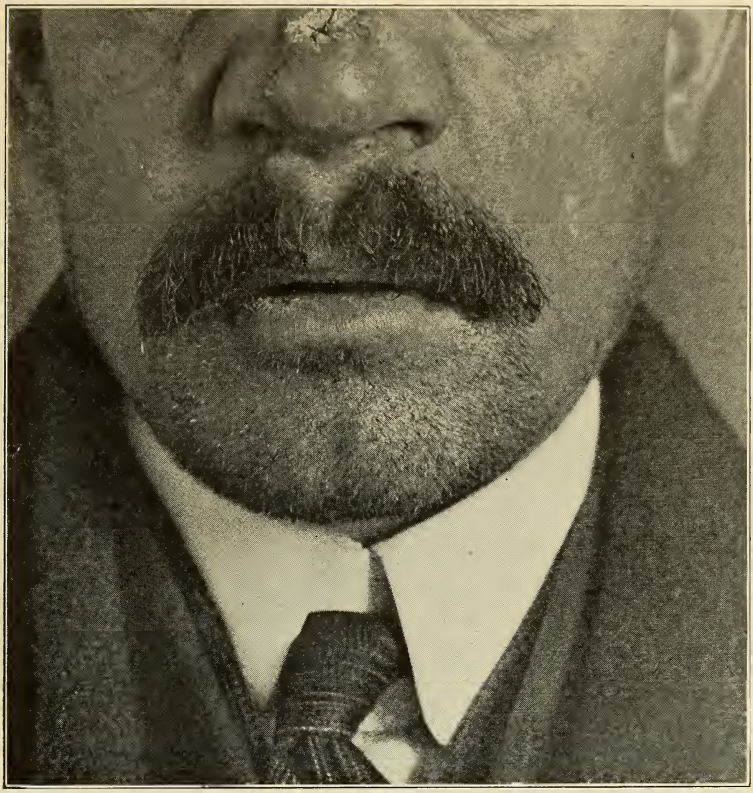

FIG. 59. RHINOSCLEROMA

As a rule the nares, tip of the nose, and areas adjacent to the nose show a stony hard, white or reddish tumor, with the type of nasal distortion here shown. Sarcoma is often suggested, and a histological examination may be required in order to make the correct diagnosis. Syphilis and tuberculosis must also be ruled out.

FIG. 60. GRANULOMA PYOGENICUM

The lesion is a bright red, raspberry-like, glistening tumor, either pedunculated or sessile. It is actually a small mass of granulation tissue, and is more frequently encountered on the fingers than anywhere else.

CHAPTER XXV

TUBERCULOSIS AND LEPRA

Tuberculosis and lepra are bacteriologically closely related diseases, in that the respective causes, the Koch and Hansen bacilli, have many features in common. Morphologically and in their standing affinities they are similar. Culturally they differ. The type of cutaneous reactions they evoke are not unlike. On the other hand, lepra has two clinical bonds with syphilis, the appearance of many of the lesions and the fact that in tubero-nodular lepra the Wassermann reaction is often present. It is manifest from the foregoing that the two diseases in question and syphilis can cause the utmost confusion, both as to their gross objective characteristics and as to some of their subtler ones.

Skin tuberculosis is in many respects a most puzzling malady. Its range of cutaneous reactions is wide, and the mechanism in their production is most varied. It is a disease almost as protean in its manifestations as syphilis and it is far less understood although perhaps nearly as important. The lesions are caused either by virulent tubercle bacilli, attenuated tubercle bacilli, or by bacillary toxins. Thus there seem to be two great groups of lesions: those which are actually infective and those which in their response to toxins are in effect allergic reactions. The latter are thus natural phenomena of which the von Pirquet reaction is the artificial replica. As our knowledge of the subject increases an ever greater number of pictures is being removed from the group of toxic tuberculosis into that caused by the bacteria. There is a further class of conditions not definitely toxic in origin but which, because of certain circumstantial points, are included with tuberculosis.

The details of these matters will be discussed in later passages. It is necessary, however, to bring out one more general point here. Those forms of cutaneous tuberculosis not considered as immediately bacterial are called tuberculides. The term is unfortunate for its significance is inconsistent with that of the word syphilide as applied to syphilodermata. A syphilide is a cutaneous manifestation immediately due to the causative organism of syphilis. A tuberculide, however, is precisely not a lesion caused by the tubercle

bacillus, but provoked by the latter's toxins, or perhaps only remotely connected with tuberculosis. This contradiction in usage has become customary and the terms are so securely intrenched that we must make the best of it and accept them. Thus we divide cutaneous tuberculosis into three groups: first, true or bacillary tuberlosis; second, toxic or presumably toxic tuberculosis, namely the tuberculides; and third, tuberculides of presumptive tuberculous nature. A fourth group might be added, namely, diseases which have not been suspected of having a tubercular etiology, but the minute anatomy of which suggests tuberculosis. The diseases themselves may be classified as follows, according to the foregoing sketch.

A. Infective Tuberculosis.
   I. Massive Granulomata.
      1. Lupus Vulgaris.
      2. Tuberculosis Verrucosa Cutis.
      3. Scrofuloderma.
   II. Tuberculosis Milaria et Ulcerosa.
      1. Tuberculosis Ulserosa Miliaris.
      2. Tuberculosis Ulcerosa (not miliary).
      3. Tuberculosis Miliaris Propria Disseminata.
   III. Lingual Tuberculosis.

B. Non-Infective Tuberculosis. Tuberculosis of toxic origin, and dermatoses associated with tuberculosis.
   I. Tuberculides.
      1. Lichenoid Types. (Lichen scrofulosorum.)
      2. Papulonecrotic Types. (Acnitis, folliclis, acne necrotica, acne cachecticorum.)
      3. Lupus Erythematosus. (Discoid, disseminated or exanthematous.)
      4. Sarcoids. (Boeck, Darier-Roussy, etc.)
      5. Erythema Induratum, Bazin.
   II. Presumptive Tuberculides.
      1. Pernios.
      2. Pityriasis rubra (Hebra).
      3. Angio keratoma (Mibelli).
      4. A vague group resembling Malcolm Morris' follicular "eczema."

C. Diseases sometimes histologically resembling tuberculosis.
      1. Lichen nitidus.
      2. Granuloma annulare.

INFECTIVE TUBERCULOSIS

In dealing with this subject, the order hitherto followed in the individual diseases will be slightly modified. Therapy will be considered separately in connection with the two subdivisions, massive and miliary tuberculosis.

The massive granulomata are voluminous, destructive and ulcerative. The miliary granulomata are small, more superficially destructive and likewise ulcerative. The latter group tends, on the whole, to be more serious from the standpoint of life and health. It is necessary, however, to grasp the fact that the distinctions enumerated are in no sense fixed and that they are artificial to the extent that they conform with fact only in a majority of instances, while the minority form a borderline which is not distinct.

This overlapping is due to the fundamental truth that the basic feature in all tuberculosis is the tubercle, running just as true to type in the skin as in the lungs. If the skin and lungs were interchanged, miliary cutaneous tuberculosis would be miliary pulmonic tuberculosis; lupus vulgaris, tuberculosis verrucosa cutis, and tuberculosis fungosa would be tubercular pneumonia; ulcerative forms would produce cavities or abscesses. The anology need be pursued no further.

Certain broad principles apply in diagnosing skin tuberculosis. In the first place, evidences of the disease elsewhere should be sought. Thus a careful, general, physical examination should be made, especially of the lungs and lymph glands. The tuberculin test should be made, particularly the hypodermatic injections, as the von Pirquet and Moro procedures are of little value. A negative von Pirquet test has some significance but the Moro test has none, whether positive or not. Often, by the first method, a local reaction appears in the lesion which becomes redder and more swollen. Syphilis is excluded by the Wassermann test.

The methods by which the lesions themselves are differentiated from those which simulate them are by inspection, microscopic examination and animal inoculation. Inspection will be dealt with further on, for clinical dermatology is indeed but the science of refined and astute inspection. Microscopic study is directed to the anatomy and bacteriology of the lesion. The anatomy is that of tuberculosis elsewhere. Frequently, however, tuberculosis looks like syphilis in its minute as well as gross anatomy. In all forms of tuberculosis the bacilli are present except in lupus vulgaris where

they are hard to find. The biological test is of great value as it is frequently positive. If some of the suspected tissue is introduced into the peritoneal cavity of a guinea pig, the latter nearly always develops tuberculosis.

LUPUS VULGARIS (FIG. 61)

Synonyms. Lupus tuberculosis. Lupus of Willan.

Definition. Lupus vulgaris is a form of chronic cutaneous tuberculosis originating in small foci. These foci, or lupus nodules, tend to invade large areas, forming flattened patches in which there is slight if any softening.

Symptoms. The characteristic feature is the above mentioned nodule which is a translucent speck in the skin, and about the size of a pinhead. The color is yellow, the shade being that of honey, apricot or apple jelly and the lesion resembles a small particle of one of these substances imbedded under the epidermis. The speck is clean cut, rounded and the skin near it is normal, or a trifle shiny. On palpation nothing is to be felt save a subtle density of the skin when gently pinched up. When such a lesion is punctured by a small blunt sound, the affected point is felt to be boggy and soft and the sound encounters no resistance. A drop of blood quickly gathers and when removed it leaves a small hole behind it where the nodule was. If the skin is rendered bloodless by pressure with glass, the nodules stand out clearly against a white background, their jelly-like appearance becoming more pronounced. This procedure overcomes the blurring of the picture by hyperemia about the nodules which, though usually slight, suffices to conceal their characteristics.

As time goes on more and more nodules appear forming plaques. Now more inflammatory elements enter the picture and the entire affected area becomes red, humid and inelastic. Such a plaque varies in shape and size and even elevation. Scaling occurs and often slight softening. Regressive changes set in and a scar forms, for the process causes a loss of substance unimaginably profound considering how superficial the elemental lesions look. There may be an annular or serpiginous arrangement of the nodules, the center healing. At times ulcers or crusts develop forming rupial or ostraceous lesions. Irregular scars remain within which new nodules often develop, reinaugurating the process.

The scars are white, livid, reddish or red, and beyond them, as beyond all types of tuberculous lesions, fresh nodules are often found.

More voluminous forms than those mentioned, are known, giving the picture of lupus tuberosus, prominens, nodosus or tumidus, or, if they ulcerate, the picture of lupus exedeus. The ulcers may vegetate, giving the picture of lupus vegetans or verrucosus. From the scars skin horns may arise and not infrequently great mutilation of tissue takes place, creating lupus mutilans.

It is necessary to mention these descriptive varieties since the affixed adjectives eliminate lengthy disquisitions, albeit it would be regrettable for the reader to assume that these varieties are fundamental.

Fully four-fifths of all lupus attacks the face and in one-half of the cases the lesions are found on the nose or near it. Three-fourths of the patients present involvement of the nasopharynx. The lips, eyelids, ears, even the drums, are often involved. Next to the face, the upper extremities are the most frequent site affected, while the lower extremities and other areas of the body are seldom involved. The portion of the nose favored by the process is that proximal to the cartilaginous area. The softer parts often entirely disappear leaving a small beak. Fingers or toes, and rarely the hand or foot, may be mutilated to the point of spontaneous amputation.

Lupus of the mucosa appears in all the forms described on the skin, but prevailingly in nodules within the nares, less often anywhere else in the buccal orifice. Destruction of the lips from within or without converts the mouth into a rigid slit, and a process on the eyelids produces a like effect upon the palpebral orifice. Involvement of the conjunctiva causes pannus and even destroys the sclera.

There are very few subjective symptoms, except such as might be ascribed to rhagades or deformities. At times there are fever, bronchitis, amyloid degeneration, pulmonary, laryngeal, bone and gland tuberculosis. As a rule the disease starts in youth, is more frequent in women than in men, and is found in the slums, particularly in Southern Europe where it is a common disease. There is, however, a great deal of lupus in the Scandinavian countries and in India.

Course. The disease tends to begin in early youth or childhood, but may arise at any age, and usually lasts for years. It cicatrizes, and more rarely ulcerates before doing so. Isolated patches may heal of themselves. Very frequently lupus is the starting point of visceral tuberculosis. In this event death occurs earlier than it would from lupus alone. Indeed, in uncomplicated lupus death is rarely due to tuberculosis.

As to the lupus lesions themselves, their spread is most gradual. It may occur by local extension, or extension via the lymphatics. In the former case great plaques are formed which spread peripherally and heal or break down centrally. In the variety which spreads through the lymphatics, a mother lesion is noted, and nearby smaller daughter lesions. In widely disseminated lupus the distribution occurs through the blood current's agency, and far flung lesions are scattered over the skin. This is the style that most frequently involves the viscera. The lesions may persist unhealed forever, or scar over, or ulcerate, or become the site of repeated attacks of erysipelas. Recurrences may take place in the scars, and the latter may be the site of epithelioma. The disease may persist from youth to old age, and in mild cases the patient remains in comparatively good health.

Varieties. In addition to the descriptive varieties enumerated above, there are lupus miliaris, lupus pernio, lupus vulgaris erythematodes, lupus tumidus, and many other even rarer varieties.

Lupus miliaris is a mildly disseminated form, one of the well known synonyms of which is acne telangiectatica. It occurs chiefly on the face and consists of disseminated nodules. It arises suddenly and progresses in successive crops. It may involve the body extensively and often accompanies tuberculides.

Lupus pernio clinically resembles pernio and anatomically tuberculosis. Formerly it was considered a variety of lupus erythematosus. It comes on the nose, cheeks, chin, fingers, toes, and ears. Its cause is obscure.

Lupus vulgaris erythematodes clinically simulates lupus erythematosus and histologically tuberculosis. Lupus nodules are present. Lupus tumidus is a voluminous involvement of the ear lobe or lobes and causes a swelling studded with lupus nodules.

Differential Diagnosis. Lupus vulgaris must be differentiated from lepra, syphilis, blastomycosis, sporotrichosis, lupus erythematosus, sarcoid, erythema induratum of Bazin, and papulo-necrotic tuberculides. The first three are diseases of a different etiology from lupus. The remainder constitute a group, the differentiation of which is largely academic, since they, as lupus, are tubercular, or probably so, in nature. The general principles of diagnosis already outlined must be applied. Lepra will be dealt with below.

Lupus resembles late syphilis. In the latter there are no lupus nodules. There is more destruction, and when ulcers form their margins are punched out, and the grouping of the lesions is reniform

or fanlike. Lupus begins early in life, and the Wassermann reaction is absent. When syphilis attacks the nose it involves the bony architecture, which causes sinking of the bridge, the tip remaining. Thus the well known picture of the saddle nose is created.

Blastomycosis and sporotrichosis are more purulent or ulcerative. The lesions are shorter lived and the respective organisms are present.

Lupus erythematosus is scalier, atrophic, and can be confused only with the rarer lupus vulgaris erythematodes which has the histological structure of tuberculosis.

Sarcoid is more superficial, or deeper (see below), lacks the nodules, but often has tuberculous structure, and in that variety known as miliary lupoid of Boeck, Boeck has found tubercle bacilli. Erythema induratum, which closely resembles sarcoid, restricts itself almost entirely to the calf in young women. Papulo-necrotic tuberculides resemble either lupus disseminatus or miliaris, but no lupus nodules are present.

Etiology. Lupus vulgaris is caused by the bacillus of human tuberculosis. It may be the starting point of, or a complication of general tuberculosis, and its dissemination is either lymphatic or hematic. Measles appears to predispose to it. The disease is largely continental. In recent years, however, it is becoming common in the United States, particularly along the Eastern seaboard, at the great landing points of immigrants.

Prognosis. Uncomplicated the prognosis of lupus vulgaris is good as far as life and health are concerned. Cosmetically the outlook never is encouraging, even though with suitable therapy the lesions may be caused to disappear, for unsightly scars remain. In necrotic forms, such as those destroying the nose, the outlines of the mouth, eyelids, etc., the disfigurement is horrible. In complicated forms the prognosis becomes that of the complicating disease.

Treatment. The general treatment corresponds with that of any other forms of tuberculosis. The best local treatment is with the Finsen light, but massive doses of X-rays and radium are also useful. Carbon dioxide snow is good only in small patches. But in these, wide excision is better. Curettage is dangerous, as it opens the lymphatics and promotes dissemination. Destruction with the actual cautery is excellent. The use of pastes such as pyrogallic acid, resorcin, etc., is fairly successful at times. Tuberculin injections are useless. The best cosmetic results by far are those obtained

by Finsen therapy. Lupus of the mucosa is best treated by the actual cautery or radium. Tuberculin is utterly useless.

TUBERCULOSIS VERRUCOSA CUTIS

Synonyms. Lupus sclerosus, Lupus verrucosus, Verruca necrogenica, Tuberculum Anatomicum, Dessection tubercle; German, Leichentuberkel.

Definition. Tuberculosis verucosa cutis is a verrucous tubercular lesion of the skin due to local inoculation with the tubercle bacillus.

Symptoms. The disease begins upon a previous abrasion as a papule, pustule or vesicle with an infiltrated reddish areola. The lesion spreads, forming indurated, horny excrescences which crust and become pigmented. It may remain small for months or years, or gradually enlarge, suppurate, or become secondarily infected.

At times the disease becomes relatively extensive, always retaining the same features as those already described. The advancing margin of the lesion is steep, the surface warty, and the color varied. It may be yellow, purple, brown or black, and present horny plugs as described. Small abscesses are often beyond the advancing erythematous zone, but never in it, as is the case in blastomycosis. Superficial erosions or ulcers may arise. The hands are chiefly involved, but other parts of the body may be. The disease may last for months or years, and although complications are rare, occasionally systemic involvement occurs.

Varieties. There are two rare varieties. One is fibromatosis tuberculosa cutis in which a sclerotic process develops amongst the features described. This is found on the lips, nose, cheeks, anus and vulva. The other is elephantiasis tuberculosis cutis, and represents tuberculosis verrucosa cutis plus elephantiasis due to blocking of the lymphatics.

Differential Diagnosis. Blastomycosis closely resembles tuberculosis verrucosa cutis as to site and appearance. The discovery of the blastomycetes in the one, and tubercle bacilli, which are easy to find, in the other, completes the differential diagnosis. The peripheral abscesses are present in the red margin of blastomycosis and beyond it in tuberculosis. Other conditions resembling tuberculosis verrucosa are lupus vulgaris, syphilis, sporotrichosis, etc. The differentiation follows along the lines indicated in lupus.

Etiology. The cause is the tubercle bacillus, locally inoculated.

Vocation has a distinct bearing on the matter, as physicians, anatomists, butchers, and undertakers are most frequently affected. On the other hand, miners and people going unshod are often infected, probably through the accidental introduction of tubercle bacilli into a preexisting skin injury.

Treatment. The therapy is identical with that of lupus. Small lesions, however, may be successfully and easily excised.

Prognosis. Save for the rare instances in which systemic tuberculosis develops, the prognosis is good. The cosmetic defects resulting are unimportant, as the disease involves the hands and feet chiefly. In the sclerotic type affecting the vicinity of the ends of the alimentary tract, and in the elephantiastic type the prognosis is poor, particularly from the cosmetic standpoint.

SCROFULODERMA

Synonyms. Tubercular tumors, Tuberculosis Fungosa, Tuberculosis Colliquativa (Gummosa) Cutanea et Subcutanea; Scrofula, King's Evil, Cold Abscess of the Skin; French, Gomme Scrofulo-Tuberculeuse; German, Kalte Haut und Unterhaut Abzesse.

Definition. Scrofuloderma is a colliquative tuberculosis of the skin and subcutaneous tissue usually causing necrotic, perforating, circumscribed nodules which arise by a direct extension from tuberculosis of the deeper structures, but occasionally apparently within the skin itself.

Symptoms. The skin becomes swollen, red or purple, and intumescent. After a prolonged period of development, an abscess forms which slowly, en masse, points and perforates. An ulcer is formed which lies deep in the infiltration from which it develops. The margins are inverted, undermined, and collapsed, the floor covered by unwholesome granulations secreting a thin watery substance. Such lesions may be sparse or numerous. They are usually found in connection with an underlying tuberculous process, as tubercular adenitis of the neck, tubercular osteomyelitis of the jaw, ear, tear duct, long bones, or tubercular fistulae of the rectum, tubercular orchitis or vulvitis. In healing they leave either depressed pigmented or irregularly linear or more rarely oval or circular scars.

At times they seem to arise independently as irregularly distributed lesions varying greatly in size. This form, rare as it is, is commonest in children, but may be present at any age, and the lesions are often mixed with papulonecrotic tuberculides. At times

scrofuloderma may arise from true lupus. Other forms arise, as nodular lesions along an infected lymphatic (tubercular lymphangitis), and involve the skin subsequently. Affected patients usually have the symptoms of general tuberculosis; sweats, fevers, asthenia, amyloid disease, etc.

Course. The cutaneous lesions are highly indolent, persisting with the underlying cause.

Varieties. The only notable variants from the common picture are the disseminated tubercular tumors and localized tubercular tumors of which tuberculosis fungosa cutis, lupus tumidus, and frombesiformis, are rare and unimportant examples.

Differential Diagnosis. Syphilis and other conditions must be excluded as in lupus. Lupus itself is excluded by the absence of nodules. The distinction is academic. The diagnosis is generally easy because of the underlying cause. The lymphangitic forms closely resemble sporotrichosis and blastomycosis, the differentiation depending upon a determination of the organisms. Scrofulous tissue is usually rich in tubercle bacilli which can be determined microscopically or by animal inoculation.

Etiology. The disease is due to direct involvement of the skin from deeper organic tuberculosis, chiefly of the glands and bones, but at times from tubercular lymph vessels.

Treatment. Surgical removal of the underlying cause cures some cases. General medical treatment common to all forms of tuberculosis is indicated; and local therapy corresponds to that of lupus.

Prognosis. If the underlying cause is traceable the prognosis is good, otherwise the prognosis becomes that of the general basic condition. In lymphangitic forms the outlook is poor.

Tuberculosis ulcerosa miliaria is rare. Small jagged ulcers follow tiny brownish scaling papules. They rarely exceed a diameter of one inch and the ulcers are shallow. The usual site is near the body orifices, hence the term tuberculosis orificialis. It is usually secondary to intestinal or pulmonary tuberculosis.

Nonmiliary Tubercular Ulcers. This is a vague clinical condition. Isolated tuberculosis ulcers, now resembling lupus, now scrofula, now tuberculosis verrucosa cutis arise. Any site may be involved. The ulcers are large, and somewhat deep, arise spontaneously, or after injury, and have an inconsistent clinical aspect. This class need be mentioned only to be dismissed with a word.

Tuberculosis miliaris propria, or disseminated miliary tuberculosis. The

disease represents the cutaneous phase of acute general miliary tuberculosis, or a hematogenously disseminated tuberculosis of the skin from a visceral focus.

LINGUAL TUBERCULOSIS

The forms of lingual tuberculosis are numerous and deserve special mention. Thus this somewhat artificial separation is justified by convenience.

Lupus vulgaris attacks the tongue as a series of nodules. The ulcerative forms may be miliary, the tongue being covered with lesions suggesting tuberculosis orificialis, or there are numerous larger ulcers. These are relatively shallow, have finely jagged edges and cover the tip and dorsum. They are painful and when deeply infiltrated must be distinguished from gummata and epitheliomata, a matter often requiring bacteriological and histological study.

Clinically, syphilis may be excluded by the fact that leucoplacia is usually present in the latter, or interstitial glossitis, and because gummata of the tongue ulcerate less easily and are more numerous. Epithelioma is usually single, more painful, and prone to arise at or near the side of the tongue at a site opposite the molars. Epithelioma, however, often springs from a leucoplacia.

TUBERCULIDES

Earlier passages in this chapter dealt with the limitations of the term tuberculides. This matter deserves elaboration. The number of clinically dissimilar conditions now included in this group is large, and it is often difficult to conceive of any inter-relationship among them. A relationship with tuberculosis is implied. Thus, tuberculides have been considered as due either to attenuated or dead bacilli (minute granulomas), bacillary toxins (allergic; erythemas or minute granulomas). Evidence favoring the assumption that these lesions are tubercular may be summed up as follows. They are often associated with some other form of tuberculosis, including tuberculosis of the skin. Bacilli have been demonstrated in the lesions microscopically and by animal inoculations. The lesions are often tubercular in minute structure. In a large number of instances these facts are undemonstrable in lesions clinically tuberculides. These are the bare outlines of the problem. The conclusion is inevitable that a group of skin lesions exists, the relationship of which to tuberculosis is often apparent, often presumptive, often impossible to prove. Because of their clinical similarity it

is at present still justifiable to group them together, with the mental reservation that when their bacterial origin is established they cease to be tuberculides and become skin tuberculosis, and when they are shown to be alien to tuberculosis they must obviously no longer be considered tuberculides. To one acquainted with the doctrine of skin reactions this is neither new nor astonishing.

LICHEN SCROFULOSORUM

Synonym. Lichen scrofulosus.

Definition. Lichen scrofulosorum is a benign disseminated form of tuberculosis characterized by the presence of grouped minute lichenoid papules, chiefly on the trunk of children and young adults.

Symptoms. Minute yellow, flesh-colored, or dirty gray, papules appear, and are isolated, grouped in discoid patches or in circular or crescentric figures. Each papule is covered by a tiny scale. There are no subjective symptoms, and often general evidences of tuberculosis are present. The sites of election are the sides of the torso from the axillae to the iliac crests, but often the buttocks, back, belly and chest are involved, and rarely the extremities.

Course. The disease may last for weeks or months. It vanishes spontaneously but sometimes recurs.

Diagnosis. Lichen planus, lichen acuminatus, papular dermatitis and lichen syphiliticus are to be differentiated from lichen scrofulosorum. All of the above conditions usually occur at a later age than lichen scrofulosorum, and the first three itch. In lichen planus there is involvement of the mucosa and in lichen syphiliticus there are other classical evidences of secondary syphilis.

Etiology. Jacobi, Haushalter, and Pellizari demonstrated the tubercle bacillus in lichen scrofulosorum. This transferred the condition to the group of true tuberculosis wherein it would have to be considered a benign miliary type. Its structure is tubercular. Infancy, childhood, and adolescence are the periods favoring the disease. It is commonest in the first of these periods. In some forms no evidence of tubercular origin can be found.

Treatment. The general tuberculosis underlying the disease furnishes the therapeutic indications.

Prognosis. In so far as the rash is concerned the prognosis is good.

PAPULONECROTIC TUBERCULIDES (FIG. 63)

Synonyms. Acnitis, Folliclis (Barthelémy), Lupus Erythemato-

sus Disseminatus (Boeck), Acne Telangiectatica (Kaposi), Paratuberculosis (Johnston), Toxituberculides (Hallopeau), etc.

Definition. Papulonecrotic tuberculides are papules on the face and extremities. They are hard, infiltrated, come in crops and disappear spontaneously leaving pitted scars. The condition is often associated with general or skin tuberculosis of another type.

Symptoms. At first brown, red or purplish papules appear at the sites affected. These are indolent, and gradually a crust develops centrally, the removal of which discovers a necrosis or ulcer. Healing is gradual and a central pitted scar remains. Slight itching or burning may be noted but usually the patient complains of nothing at all. Other evidences of systemic tuberculosis may be present, or the lesions may exist coincidentally with other forms of skin tuberculosis, notably lupus vulgaris or scrofuloderma, or with other tuberculides, chiefly erythema induratum or lupus erythematosus.

The lesions tend to come in crops over periods of years and may be numerous. They may be sparse indeed, particularly in babies, in whom they often represent the first evidence of a systemic infection terminating with the picture of pulmonary, meningeal, or some other form of visceral bone or gland tuberculosis.

Course. The course may be inferred from the foregoing.

Varieties. Acnitis is that form which involves the face, especially the cheeks, brow, lips and chin. The lesions may reach the size of a pea and are hard and shotty. They are associated commonly with the next form to be described.

Folliclis. The appearance of folliclis is identical with that of acnitis, and except for localization no difference exists between the two. The hands, forearms, feet and legs are affected, the body being rarely involved. It is in acnitis and folliclis that crops are commonest, the course of each crop approximating four to six weeks. At times the necrotic centers are purulent and this is especially the case in a rare subvariety seen chiefly in London and called acrodermatitis pustulosa hiemalis, the name describing the salient attribute of the condition. This form favors the knuckles. Acne scrofulosorum is also a rare subvariety involving the outer aspects of the limbs, the lower extremities being more frequently involved. A shallow pitted scar remains. The sacral region may be involved. This form is closely related to acne cachecticorum, the lesions of which favor the trunk. In general it may be stated

that the differences among these types are artificial rather than actual.

Acne Necrotica is a condition hitherto not classed among the tuberculides. Its best known synonyms are acne varioliformis and granuloma necroticum. The lesions resemble those of acnitis and are similarly situated. They differ, however, in being follicular. Their association with tuberculides has been mentioned by Schamberg.

Differential Diagnosis. Save for syphilis nothing can be confused with tuberculides. The differentiation can be made by clinical and serological study.

Etiology. Often some form of systemic or cutaneous tuberculosis is ascertainable. This, together with the facts that histologically tuberculosis is often suggested, that tuberculin reactions both general and local may frequently be elicited and that at times the tubercle bacillus has been demonstrated microscopically or by animal inoculation, indicate clearly that at least a substantial proportion of the cases are tubercular. On the other hand, in many cases these criteria fail, and in these the causation is unknown.

Treatment. The general management of tuberculosis is to be pursued where indicated. The best local treatment is the use of weak ammoniated mercury salve. Tuberculin is useless. As a matter of fact the vagaries of the disease are its most striking feature, and measures that appear efficacious at one time may be fruitless at another in the same individual.

Prognosis. The prognosis as to the lesions is good, as they tend to disappear spontaneously. If they be associated with tuberculosis the prognosis becomes one with that of the major condition.

lupus erythematosus (figs. 64 and 65)

Synonyms. Lupus Erythematodes (Cazenave); Erythema Centrifugum (Biett), Ulerythema Centrifugum (Unna), Seborrhoea Congestiva (Hebra).

Definition. Lupus erythematosus, in its usual form, is a common inflammatory disease, the lesions of which develop gradually as symmetrical red or purple scaling patches, and involute slowly leaving a delicate atrophic scar. Although the disease is chronic as to its entire course, the lesions may sometimes develop and resolve quickly so that the individual exacerbations and remissions may in time be chronic or acute. In addition there are acute

varieties which terminate quickly and fatally, or which, developing suddenly, become chronic.

Symptoms. The condition starts at adolescence or in early adult life with patches on the head, hands, or rarely elsewhere on the body. The portions of the head most involved are the nose, cheeks, forehead, ears, scalp, vermilion border of the lips, and the mucous covering of the tongue and cheeks.

At first the lesions are pale pink, small, slightly elevated macules which may grow gradually over a period of weeks, months or years, reaching great proportions at times, but usually ranging between one-eighth of an inch and two inches in diameter. The lesions (Fig. 64) are circular or oval and sharply circumscribed, with a slightly elevated border, or one more markedly raised and infiltrated. The entire patch is sometimes as high as the margin, or sunken; and often there is central involution surrounded by a more or less broad active ring. Such a lesion may remain as described for varying periods even up to years, or gradually grow, or remain stationary, grow suddenly, and again become stationary. The advancing or stationary lesions are a vivid red, but often a distinct brown or purple hue is noted which is accentuated in low temperature.

The surface of the lesions is covered with adherent greasy scales, which are white, yellow or gray, from the lower surfaces of which, when they are lifted off, project minute horny plugs. These plugs are scaly casts of patulous pilosebaceous follicles. Thus, when denuded of its scales, the lesion is pitted because of the dilated follicular openings.

The lesions may be single but it is more characteristic for them to be multiple and symmetrical. Their circular or oval outline, due to coalescence, becomes festooned or irregular. At times nearly the entire face is masked by merging of lesions (Fig. 65). A figure frequently formed is that of an open butterfly when lesions covering the bridge of the nose and adjacent cheeks, unite into one plaque.

After involution an atrophic pliable, delicate, white and superficial scar remains. Upon the ears such scars destroy the soft parts of the helix down to the cartilage, giving a picture identical with that of chilblain scars. The active lesions themselves, particularly on the nose, ears and fingers also suggest the active stage of pernio. Recurrence in the scars is possible.

The process is destructive. Thus under the microscope the skin

organs are absent, and when atrophy has occurred the entire follicular structure and elastic fibers have vanished. Hence the scar is pearly white or pink, pliable except in the scalp where the flesh cannot be pinched up, and it puckers easily and is moveable. When scars have formed on the scalp there are local areas of baldness, in contour not unlike those seen in alopecia areata.

Lupus erythematosus of the lips causes edematous, red patches with pearl gray slightly raised margins. Healing produces atrophy with a peculiar glassy surface. Similar lesions are noted when the tongue is involved. Active lupus erythematosus of the scalp often is elevated and the patch scales markedly but is even more markedly the site of hyperkeratotic plugs. Thus the surface suggests that of a strawberry to the touch and eye, save that it is never so vividly red.

Course. The course of the disease is long, that of the lesions or crops of lesions varies as indicated in the definition.

Varieties. Lupus erythematosus discoides (Fig. 64) may be localized or disseminated (Fig. 65), but the true disseminated type consisting of typical lesions, is not lupus erythematosus discoides, but is better known as lupus erythematosus exanthematicus and will be described below. At times, however, the true exanthematous form rises after or leads to the discoid type.

Differential Diagnosis. To make this clear it will be necessary to subdivide the manifestations of the disease arbitrarily and to deal with the disease in its individual various manifestations. The active lesions resemble seborrhoea, psoriasis, rosacea, lupus vulgaris, sarcoid, lepra and chilblains. The scars on the glabrous areas resemble those of superficial macular atrophies and morphoea. The scars on hairy areas suggest alopecia areata, pseudopelade and favus. The mucous lesions suggest lichen planus, syphilitic leucoplacia and superficial basal celled epithelioma prior to ulceration.

Seborrhoea itches, is yellowish, less infiltrated, more disseminated, more localized to the nasolabial and post-auricular folds. The scales are less adherent and have no inferior plugs. The disease responds more readily to therapy than lupus erythematosus, and there is no atrophy.

Psoriasis is covered with nacreous white scales, is more typically localized, and is distinguished by these differences along the lines indicated in seborrhoea. Bleeding upon excoriation is another important diagnostic feature of psoriasis.

Rosacea resembles the butterfly wing type of lupus erythematosus,

but it is not elevated, scales more lightly and is greasier. The flush increases in warmth rather than in lower temperatures, and it is associated with pustular folliculitis.

Lupus vulgaris differs from lupus erythematosus in the greater irregularity and tumefaction of the lesions and in the presence of the yellow lupus nodules revealed by glass pressure. Sometimes the diseases coexist. Similar yellow nodules are seen in lupus erythematosus. They are clumps of degenerated elastic tissue, but the nodules of lupus vulgaris yield easily to a blunt probe, while those of lupus erythematosus do not. Microscopically lupus vulgaris has the structure of tuberculosis.

At this point it may be mentioned that the microscopic study of lesions helps in differentiation from sarcoid and lepra also. In sarcoid there is a granulomatous structure suggesting tuberculosis. In lepra the lepra cells and Hansen bacilli are found except in the maculo anesthetic forms. Lupus erythematosus is a chronic inflammation leading to destruction of elastic and connective tissue and shows nothing suggestive of a tubercle or leproma. Maculo anesthetic leprides are insensible. Chilblains are clinically like lupus erythematosus both when active and in their destructive propensities, differing only in that the latter are less marked and more infrequent, and that the condition is confined to cold seasons.

The lupus erythematosus scar is like that of macular atrophy, but the early stages of the two diseases differ. A puffy white, red, or faintly violet lesion precedes the scar of macular atrophy. Without a knowledge of the early forms the two could hardly be distinguished. The same applies to morphoea save that the scars are firmer.

Alopecia areata resembles the scar of lupus erythematosus only in outline. The alopecia patch is not a scar. The follicles are present, the hairs rapidly grow again, the skin is pliable.

Pseudopalade cannot be differentiated from the cicatrix of lupus erythematosus except in so far as other evidence of either disease exists.

Favus is practically restricted to Russian Jews, the scar is irregular and it binds down.

Lichen planus of the mucosa consists of minute polygonal papules and, as a rule, typical skin lesions indicative of either disease are present.

Leucoplacia is dead white, irregular, and serological and other clinical evidences of syphilis are found.

Superficial epithelioma can usually be ruled out easily as the lupus margin is less rolled. A microscopic examination would clear up the puzzling cases.

Etiology. The etiology of lupus erythematosus is not definitely known. Darier considers the disease an atrophying erythematous tuberculosis. Microscopically and bacteriologically there is nothing whatever to justify this point of view. The disease, however, is frequently associated with systemic and other forms of skin tuberculosis, as are other tuberculides. Thus the non-tubercular types may be assumed to be due to some focal infection or metabolic disturbance.

Therapy. Treatment is general and local. By virtue of our ignorance of the cause of the disease there is no rational general treatment other than those vague measures employed in managing any serious disease. Local treatment consists of the use of acids or certain actinic rays. Of the former, trichloracetic acid is the best. It is applied once a week, each time until the skin is blanched, which occurs in a fraction of a minute. The best ray therapy is furnished by the use of the Kromayer light, applied unfiltered for three or four minutes to each patch, weekly. X-rays are contraindicated, but radium has been found of value. Some cases respond well to one form of treatment, others to another, and even different lesions, in the same individual, vary. The internal administration of quinine coupled with tincture of iodine locally has had wide vogue, particularly in Germany. In the very acute forms soothing lotions of the calamine and zinc type are to be employed until the skin is ready for the revulsives mentioned. Destructive local treatment is the only logical form to employ. A small proportion of cases in intractable.

Prognosis. This is usually bad as to prevention of recurrences and good as to curing the attack. Sometimes the attacks, however, will not disappear either spontaneously or with all our arts. As a rule discoid lupus erythematosus does not menace general health.

LUPUS ERYTHEMATOSUS DISSEMINATUS

This rare form of disease, better called lupus erythematosus exanthematicus, arises spontaneously and acutely and may run an acute, subacute, or chronic course. At times its starting point is in lupus erythematosus discoides, more rarely it terminates in this picture. Usually it looks nothing at all like lupus erythematosus but closely resembles erythema multiforme. Its systemic features, however, are

severer, it often terminates fatally, there are fever, abdominal cramps, pains in the bones and joints, and even delirium and coma. Subacute forms are characterized by a milder course and frequent occurrences.

A very serious form of the condition is Kaposi's erysipelas perstans faciei, which starts with facial lesions resembling the discoid or disseminated sort. These coalesce, the face becoming swollen and dusky. Severe systemic symptoms develop, and lesions on the body suggesting either lupus erythematosus discoides or disseminatus are found, or other evidences of systemic or skin tuberculosis. The patients usually die after a few weeks or months. Albuminuria is common and nephritis frequent. The therapy is symptomatic, the etiology obscure and the prognosis bad.

Lupus pernio, also known as chilblain lupus, has been alluded to in connection with lupus vulgaris. Many authors associate it with the latter, others with lupus erythematosus. It is a papular, cyanotic disease of the fingers, toes, nose, ears, etc., and it is scaly and leads to atrophics and mutilating scars. Thus it closely resembles lupus erythematosus, but microscopically it often is tubercular in structure.

Lupus erythematosus telangiectaticus is rare and resembles the ordinary forms of the disease — but is more vascular and less scaling. It causes atrophy and its vascularity takes the form of telangiectatic vessels lying in the erythematous lesion.

SARCOID

Synonym. Multiple Benign Sarcoid (Boeck), Benign Miliary Iupoid (Boeck), Nodular Hypodermic Tuberculide (Darier).

Definition. A generation ago this group of cutaneous lesions was expansive. It included mycosis fungoides, hemorrhagic sarcoma of Kaposi, xanthoma tuberosum and leucemia of the skin. The reason for this unwarranted association of entirely unrelated entities lay in the clinical fact that although they resembled sarcoma they were not sarcomas. Thus the term sarcoid had been coined. When Boeck first described the condition named for him he included it among the sarcoids because of its clinical resemblance to the group mentioned. One by one each of the diseases in question has been otherwise classified, and the list has shortened till one survivor remains, the multiple benign sarcoid, for which Boeck himself has suggested as a better designation miliary lupoid. In spite of this the term sarcoid persists. Sarcoids are chronic

granulomatous lesions remotely suggesting sarcoma and somewhat suggesting atypical lupus erythematosus in their clinical appearance. They closely simulate cutaneous tuberculosis histologically.

Symptoms. Sarcoids may be superficial or deep, in other words dermic or hypodermic. The first in turn forms small nodules, large nodules or plaques. The dermic variety bears Boeck's name, the hypodermic Darier's.

Boeck's sarcoid favors the face and shoulders and upper extremities but may be diffusely disseminated. The lesions, whether small, large, or in plaques, start as red, edematous or infiltrated spots which turn purplish or brownish, are elevated as much as a quarter of an inch, and burn or itch. They grow purple and telangiectatic centrally. The margin turns brown or yellow and scales, and as involution sets in they fade leaving a brownish discoloration and a slight atrophy. They never break down.

The small lesions are as large as a pinhead or hemp seed. The large ones may be an inch or more in diameter and higher than the smaller lesions. The diffusely infiltrated lesions may reach the size of a hand. Of course all start as small nodules. Diascopic examination shows that the elemental feature is a grayish yellow minute point suggesting a remote similarity to the lupus nodule. Hence the term miliary lupoid. The gross appearances of the nodules, then, is the sum of these numerous miliary structures set within an engorged or telangiectatic background. The elevation is due to infiltration and edema. The scarring is the result of connective tissue destruction. Patients presenting the picture here described usually seem in good health. At times the glands are enlarged.

The hypodermic form, or Darier's type, also known as Darier-Roussy's form, is a granuloma of the subcutaneous tissue. This forms a nodular lesion about the size of a hazel nut or a walnut. The tumors are globular or oval, the skin over them reacting as in Boeck's form, and they are freely moveable below. Coalescence causes them to form irregular deep plaques, some even eight to ten inches in diameter. The overlying skin, adherent to the infiltration, resembles orange peel in character, when pinched. The color is purple, slate or red. This form favors the trunk.

Course. The course of the disease is slow, the lesions requiring months or years to unfold and involute. Therapy curtails the disease.

Varieties. These have been mentioned in the symptomatology.

Differential Diagnosis. Sarcoma, mycosis, lupus erythematosus,

lupus vulgaris, erythema induratum, erythema nodosum, and periosteo-cutaneous gumma must be excluded. Sarcoma is excluded by histological study. Mycotic and leucemic infiltrations usually itch intensely, and here again microscopic study is often necessary. Lupus erythematosus when edematous is not very scaly, and lupus erythematosus telangiectaticus so closely resembles sarcoid that no clinical differentiation is possible. Only the histological structure, which is that of tuberculosis, will clear up the diagnosis. Lupus vulgaris contains yellow nodules, as opposed to the miliary spots of sarcoid. It is a process destructive through necrosis rather than through atrophy. Erythema induratum is so like Darier's form as to be considered by many identical with it. This will be discussed below. Erythema nodosum is acute and attended by mild or severe systemic symptoms chiefly rheumatoid in type. The lesions are both painful and tender and have the appearance of contusions. All of these features are absent in sarcoid. Syphilitic periostitis of the tibia secondarily involving the skin is painful and tender. The lesions resemble those of the deep sarcoids. Other manifestations of lues, however, are present and the Wassermann reaction is elicited.

Etiology. Boeck found tubercle bacilli in one of his cases and Darier and Roussy in one of theirs. The structure of the lesions is like that of tuberculosis. Women are afflicted more than men.

Treatment. Arsenic is almost a specific.

Prognosis. This is generally good.

ERYTHEMA INDURATUM (FIG. 66)

Synonyms. Erythema Indurativum; French Erythème Indurè des Scrofuleux (Bazin).

Definition. Erythema induratum is a granuloma attacking by preference the calves (Fig. 66) of young individuals, chiefly females. It is prone to break down and is often associated with papulo-necrotic tuberculides.

Symptoms. The disease begins with pain at the sites mentioned and at the age reported, but the lesions may occur elsewhere and in later years. The nodules are hard and resemble the hypodermic sarcoids, but often break down into punched out or irregular ulcers. The lesions resolve spontaneously and tend to recur.

Course. The course of the lesions themselves requires months to be completed and they leave pigmented scars suggesting those of syphilis. The entire condition is characterized by recurrences, the general health not suffering materially.

Differential Diagnosis. The disease is identical with Darier's sarcoids save that it tends to limit itself to the calf and is necrotic. To enumerate the conditions from which it is to be differentiated and the method of accomplishing the differentiation would be to repeat the corresponding passage in connection with Darier's sarcoids.

Etiology. Successful animal inoculations indicate that the tubercle bacillus is probably the cause of this disease. Women are afflicted more than men.

Treatment. Antiseptics locally, calomel injections as in syphilis, and tuberculin injections, are useful. The attacks tend to be self limited.

Prognosis. This is good.

Presumptive tuberculides, pernios, chilblains, frost bites, are in some of their forms possibly tuberculides. They are related to lupus vulgaris or lupus erythematosus.

Pityariasis Rubra (Hebra-Jadassohn) has been described (Chapter XII). It is a generalized exfoliative erythroderma, and because in so many instances systemic tuberculosis has been discovered at autopsy it is believed that at least a considerable proportion of the cases are due to tuberculosis. In one instance Brusgaard found tubercle bacilli in the skin.

Angiokeratoma of Mibelli is a rare disease usually found in adolescent girls. The toes and fingers are covered with pink, minute macules, gradually becoming cyanotic, which are hyperkeratotic, smooth or rough. Under the diascope a persistent central angioma is seen. The lesions have been found accompanying other tuberculides, but the relation to tuberculosis is not definitely established. The condition, or a similar one, is often seen on the scrotum in men obviously not tubercular.

A follicular eczema resembling Morris's has been considered by some to be related to tuberculosis.

DISEASES SOMETIMES HISTOLOGICALLY RESEMBLING TUBERCULOSIS

Lichen nitidus has been already referred to (Chapter XIII).

Granuloma annulare (Chapter VIII) or ringed eruption, is extremely rare. It is found upon the back of the hands, fingers, wrists, neck, elbows, ankles and buttocks. The lesions consist of rings formed of an elevated margin one eighth of an inch broad and about one sixteenth of an inch high. The rings may be complete,

or only in segments, and the outer aspect is deep and sharp, the inner gradual and merging into an enclosure of normal or slightly atrophic skin. Within the enclosure are papules strongly suggesting those of lichen planus. The color of the rings and papules is flesh toned, pink or vivid red. Single lesions or numerous rings are found. The etiology is unknown but sometimes the patients give a tuberculin reaction. At times the minute anatomy is suggestive of tuberculosis. The prognosis is good and the best treatment a single Roentgen exposure to three or four Holzknecht units.

LEPRA

Synonyms. Leprosy, Morbus Biblicus, Elephantiasis Greacorum, Lepra Arabium, Leontiansis. Norwegian, Spedlasked; German, Aussatz; French, Lepre, Ladrerie.

Definition. Lepra is a chronic infectious disease caused by the bacillus leprosus, or Hansen bacillus. It runs a course of years, in which there are eruptive and general phases.

Symptoms. During the incubation period, the length of which is unknown, there may be no symptoms or there may be weakness, anemia, desire to sleep, arthralgia, neuralgia, diarrhoea, headache and vertigo. As a rule the fever is slight but there may be hyperpyrexia with morning remissions suggesting sepsis. An obstinate coryza with epistaxis, and local asphyxias of the extremities are not unusual. Thereupon the macular stage arises. A roseola appears upon the face, extensor surfaces of the extremities, and particularly upon the thighs and back. The macular leprides are erythematous resembling toxic eruptions that are red, bronzed, or yellow, or even rosaceous. In addition there are depigmented macules surrounded by hyperpigmentation, and often these are anesthetic, but sometimes hyperalgesic. These eruptions are composed of lesions irregular in size and shape, and the different types may be intermingled.

At times bullae develop after the macular stage but more often it evolves directly into the nodular phase, giving the picture of tuberous, nodular or tubercular lepra. These lesions are distributed as the macules are, and may be small or large, dermic or hypodermic (as sarcoids), and impart to the face a characteristic appearance known as the leonine facies or leprous leontiasis. Nodules involve the ears, forehead, eyebrows, sides of the nose and chin in symmetrical, irregularly disposed rows that cause the brows to overhang, and the features to change in a manner suggesting a lion's face. The nose grows large and flat, the eyebrows and nails fall or at least

get lustreless, the hair grows sparse and the ear lobes are studded with nodules.

The extremities are covered with the nodules which have a coppery or red color and are somewhat waxy. Often they are scaling and erythematous, and elephantiasis of the legs is frequently seen. The glands are enlarged. Rarer participants in the process are the larynx, conjunctiva, sclera, iris, and thoracic and abdominal viscera. Orchitis is not rare. Often the nasopharynx is the site of lepra nodules.

The above description is that of nodular lepra. Another equally important picture is that of anesthetic, maculo-anesthetic, nervous or trophoneurotic lepra. It too may have a prodromal and macular stage. The peripheral nerves, however, become the site of lepromas. Thus, on palpation, they are found to be nodular and enlarged. Neuritis and trophic disturbances, neuralgia, anesthesia, analgesia, and a picture suggesting syringomyelia or Morvan's diseases is evolved. There are perforating ulcers and the like.

Mixed forms are known and both types may lead to the picture of mutiliating lepra, with amputation of fingers, toes, or even larger portions of the members. Decubiti and bony rarefaction are not uncommon and the patient ends his existence emaciated, deformed, blind, paralyzed, dying of leprous cachexia, or an intercurrent infection.

Course. This covers a period of years and is divided, as stated above, into a period of incubation, eruption, nerve involvement, or both, and finally death.

Varieties. There are two varieties, as already stated — nodular and maculo-anesthetic or nervous.

Diagnosis. When lepra is suspected the similarity to tuberculosis and syphilis is so great that diagnosis often depends upon the microscope. The bacilli are easy to find both in smears from punctured skin lesions and from the nasal secretion. The anatomy of the lesions is fairly characteristic, what with the lepra cells and Hansen bacilli. Tubercle bacilli are harder to find in sections and when present are less numerous than those of lepra, and morphologically a trifle different. The Wassermann reaction is present in nodular lepra so that this means of differentiating it from syphilis is vitiated. The tuberculin reaction is absent in lepra, but a leper may have had or may have tuberculosis, so that its absence is significant but its presence does not exclude lepra.

Neither in tuberculosis nor syphilis are there sensory disturbances,

FIG. 61. LUPUS VULGARIS

At the tip of the nose and over the bridge are seen "lupus nodules." Elsewhere the lesions have run together into confluent elevated patches. There is scaling, but no pustulation or scarring, in this patient. The color is usually red or purple. Often the distribution of lupus vulgaris and lupus erythematosus is the same, but by regarding Fig. 57 the atrophy and lack of nodules in the latter will be noted.

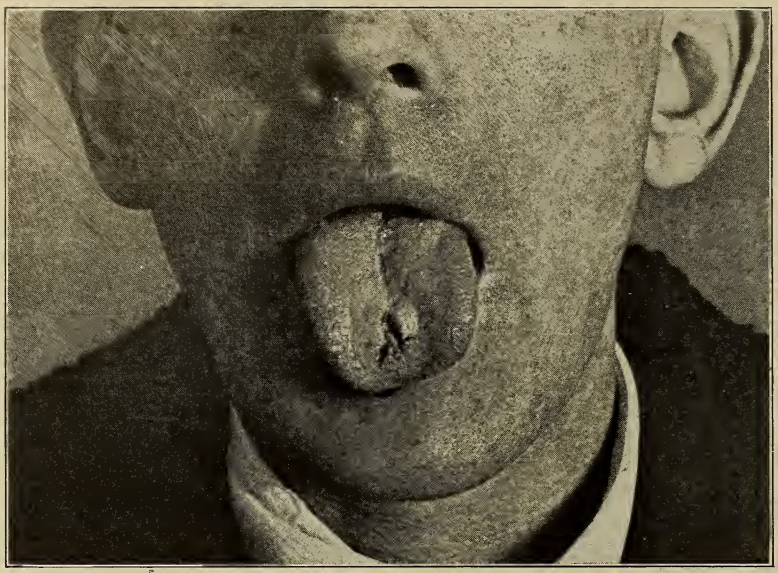

FIG. 62. LINGUAL TUBERCULOSIS

The ulcers in lingual tuberculosis are irregular, have undermined edges, a red floor, and usually the bacilli are demonstrable in smears.

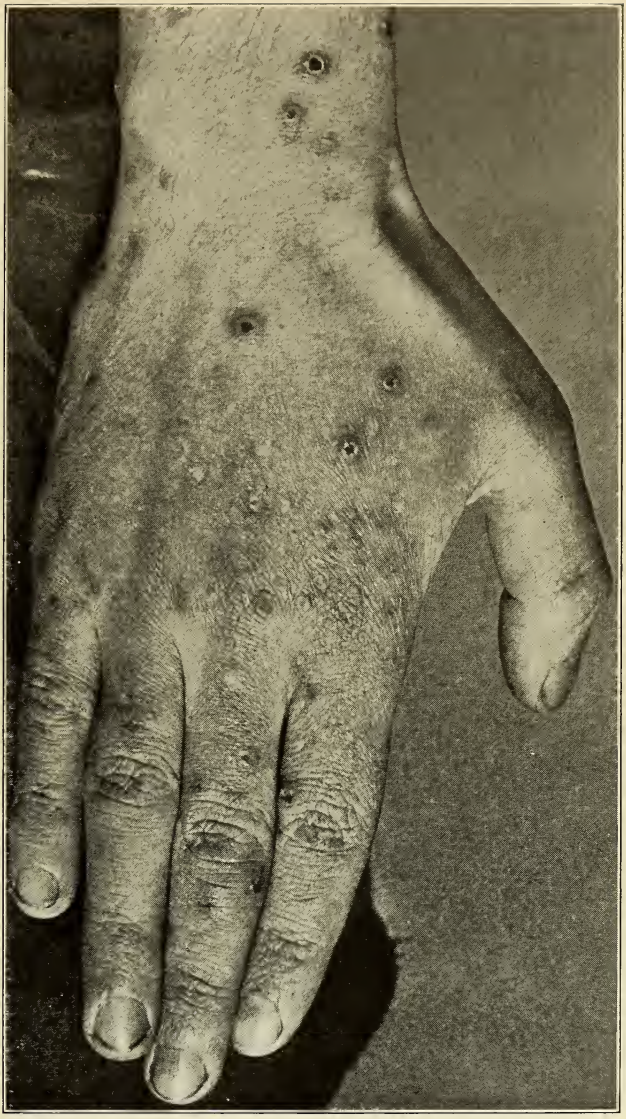

FIG. 63. PAPULO-NECROTIC TUBERCULIDES

Small papules with necrotic centres and red zones, and residual scars characterize the disease. On the face the condition is called acnitis; elsewhere, folliclis. The distinction is artificial. Often lesions are associated with those of lupus, lupus erythematosus, sarcoids, or erythema induratum.

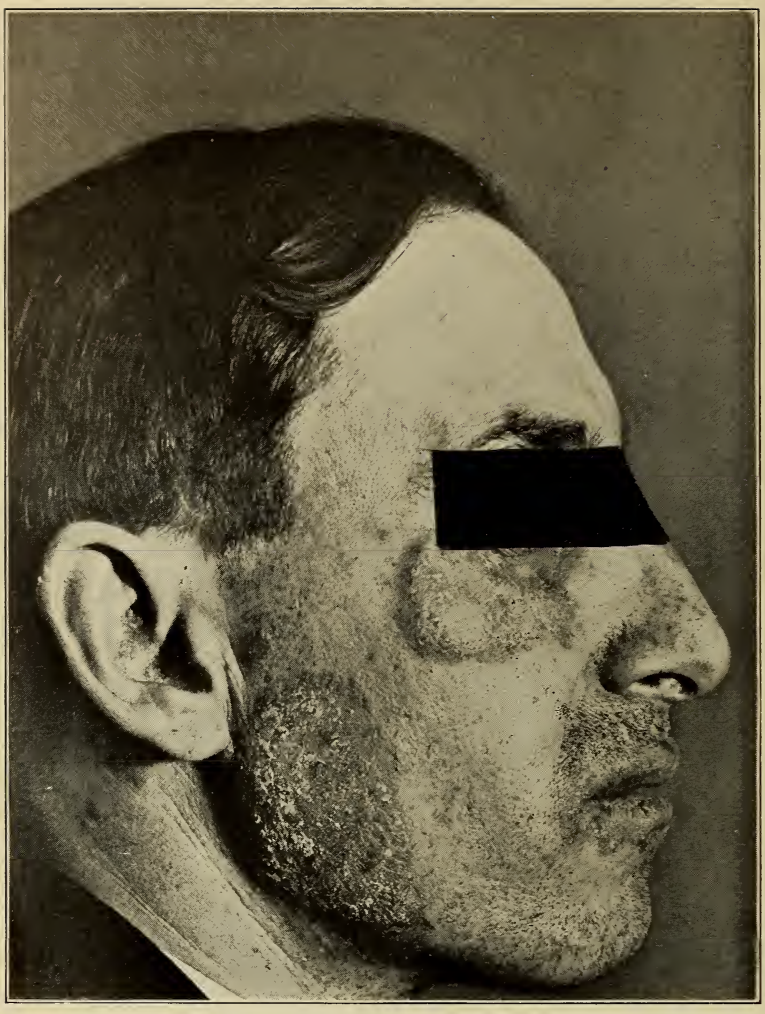

FIG. 64. LUPUS ERYTHEMATOSUS DISCOIDES

Symmetrical red patches form, usually on the face. They are slightly raised, infiltrated, scaly, and lead to atrophy, as seen over the eyebrow and margin of the ears. At the latter site the atrophy has deformed the ear.

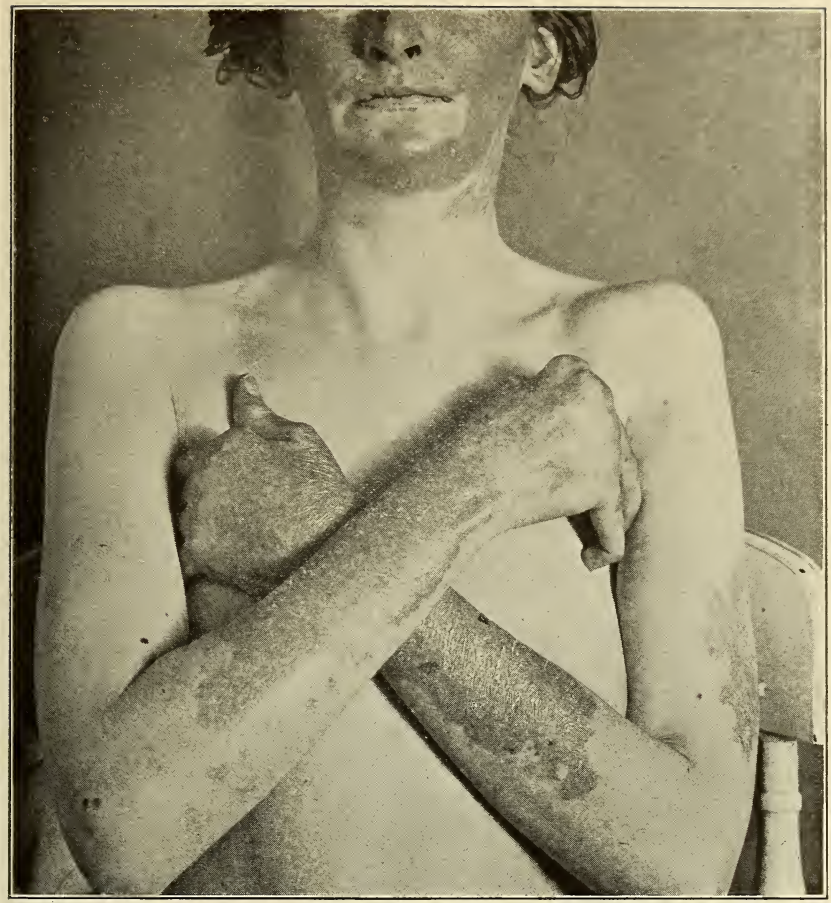

FIG. 65. LUPUS ERYTHEMATOSUS DISCOIDES DISSEMINATUS

In this disease, lesions occur over widely disseminated areas but are otherwise of the same nature as the simple discoid variety. In the acute disseminated form, erythema multiforma is more closely simulated, and the disease is at times fatal. In acute erythematous facial lupus erysipelas may be mimicked.

FIG. 66. ERYTHEMA INDURATUM OF BAZIN

The site is typical. The lesions break down, as here shown, and closely resemble gummata or deep sarcoids. Women are rather more commonly affected than men. Sarcoids rarely break down, and in gummata the Wassermann reaction is likely to be present.

but in syphilis of the central nervous system there may be trophic disturbances. Analysis of the cerebrospinal fluid would prevent confusion.

The clinical differences between the lesions of lepra on one hand and tuberculosis and syphilis on the other would have to be worked out by inspection. This has been dealt with adequately in the passages on tuberculosis. Lepra may simulate mycosis fungoides. The latter itches and is histologically different.

Nerve lepra simulates syringomyelia as to atrophy of muscle, particularly the interossei, and as to the presence of perforating ulcers and other trophic disturbances. The lepra claw is quite distinctive, the proximal phalanges being flexed, the distal extended. The ulnar, great auricular, peroneal, tibial, and the branches of the brachial plexus are palpable and irregular. In syringomyelia the skin lesions are present only within the area of nerve involvement. In lepra they may be elsewhere. The tendon reflexes are absent in the former and increased in the latter.

Etiology. The cause is the Hansen bacillus. Contributing causes are prolonged exposure, crowding and filth. In warm climates the bulk of the cases are of the nerve type. In cooler districts the reverse obtains. The exact mode of transmission is not yet known, but it is probable that an intermediate epizoön is the carrier.

Treatment. Segregation for prophylaxis is the most important measure. Tonics, arsphenamin, nastin and many other drugs have been employed with discouraging results. Of all methods, to-day, as of old, the best is the use of chaulmoogra oil. This substance is given three times daily, after meals, in capsules containing fifty to eighty drops, the average being sixty. At the same time a fiftieth of a grain of strychnin is given. I was told that at the Louisiana State Home for lepers this therapy was considered the most satisfactory.

Prognosis. Although, on the whole, the prospects of a cure are never brilliant, with the treatment outlined above, a certain number of cases are arrested, and others ameliorated.

GROUP VII. GRANULOMATA

CHAPTER XXVI

NON-INFECTIOUS GRANULOMATA

In designating the conditions to be described in this chapter as non-infectious granulomata something perhaps unjustifiable is assumed. The view has not entirely been abandoned that these conditions are infectious. There is no evidence supporting this attitude, and indeed, it must be confessed that granuloma fungoides in many respects conveys an impression of possible infectious origin. On the other hand nothing is known that substantiates this feeling. The granulomata perhaps resemble those of syphilis, tuberculosis and lepra. Dermatoses in the prefungoid stage of mycosis constitute a rough analogy to the macular syphilides and leprides. Yet reasoning by analogy is inconclusive. Certainly no real evidence of infection exists. No proof of transmission is recorded. No infectious agent is known. Mycosis and leucemia resemble sarcoma even more than they do the infectious granulomata. The iodides and bromides (Chapter VII) also cause polymorphous manifestations not unlike some of those of preleucemia and premycosis, while the halogen granulomata in turn closely resemble some of the infectious granulomata. Thus we are forced to an admission of ignorance of the nature of these diseases, and there is more circumstantial evidence that they are metabolic in source than that they are infectious. In the last analysis we must confess that, since all disease is either infectious or not in origin, a discussion of this side of the matter to-day is as fruitless as the theme of a school boys' debating society.

Granuloma fungoides and leucemia cutis have many points in common. Both possess early stages characterized by itching dermatoses; both exhibit granulomatous phases; mycosis sometimes is accompanied by blood changes, leucemia is sometimes without any. Thus the two conditions not only are clinically similar but actually are often clinically indistinguishable. Again, they clinically often resemble sarcoma. In older works mycosis and leucemic granulomata were called sarcoids, so close to sarcomata are they in appearance. Our present lack of information thus justifies the belief that they are closely related and represent a process sui generis.

The granulomas caused by iodides and bromides are obviously non-infectious, and are placed in this chapter because they constitute a picture which the practical physician must have clearly in mind. The other diseases, mentioned above, are rare, but must be recognized because of their important bearing on internal medicine.

GRANULOMA FUNGOIDES (FIGS. 67 AND 68)

Synonyms. The commoner synonyms are mycosis fungoides, lymphadenie cutanée (indicating its unknown origin); Entzünaliche fungöse Geschwulstform, and inflammatory fungoid neoplasm, indicating its clinical similarity to inflammations; sarcoma lymphadenoides myxoides, sarcomatosis cutis and sarcoma cutis multiplex idiopathicum, indicating its clinical likeness to sarcoma.

There are still other names. The only designations in common use are mycosis or granuloma fungoides. The only sensible one is the last.

Definition. Granuloma fungoides is a chronic disease of unknown origin characterized by a long course divided into various stages. The first stage is one in which there are dermatoses of various generic types, followed by an erythrodermatous period, which in turn gives way to a tumor stage. The first two phases are unhappily called premycosis or premycotic, the final stage fungoid or mycotic. It would be better to use the terms prefungoid and fungoid.

Symptoms. Partly for the sake of convenience, and partly because it is usually a fact, granuloma fungoides may be divided into three stages, an exanthematous, erythrodermal (Fig. 67) and fungoid stage (Fig. 68). At first there may be only generalized, more or less intense, pruritus. Or there may be transitory but repeated attacks of urticarial, vesiculosquamous or erythematous eruptions, the last often resembling erythema multiforme. Another early form of the disease is parapsoriasis (Chapter XI), or closely related conditions, or psoriasiform, "eczematous," bullous, or lichenous eruptions. It is common to see such eruptions limited to that part of the body below the navel, but they may be anywhere. The commonest prefungoid eruptions are those suggesting squamous dermatitis, psoriasis and parapsoriasis. The lesions are festooned, corymboform, scaly and itching; or there are gyrate plaques. The lesions tend to be bluish red and slightly infiltrated.

Now the erythrodermatous or infiltrated stage arises, a condi-

tion clinically often indistinguishable from pityriasis rubra of Hebra (Chapter XII) or dermatitis exfoliativa of Wilson-Brocq. But there is more itching and infiltration. In either of these two stages fugacious nodules, varying in size up to that of a dime or quarter, appear. They last a few days or weeks and gradually this tendency dominates the picture. The nodules develop into tumors, some of which reach the size of a mandarin or tangerine. They develop rapidly, reach a given size and involute more or less rapidly by absorption or ulcerative necrosis. Their shape is often like that of a tomato. They are slightly lobular and as often have a constricted as sessile base. Their color is dusky red, purple or brown.

While this goes on, the skin grows pigmented, atrophic, or may show evidences of the early stages. The tumors arise anywhere, but the face, groins, armpits and chest are the favorite sites. The tumors may be single or multiple and the patient gradually wastes away, although comparatively good health may endure for years. General glandular enlargements occur. There is, as a rule, only slight if any secondary anemia, often a leucocytosis with diminished polymorphonuclear count, and often a marked eosinophilia. The range of leucocytes is from normal to 112,000, that of the eosinophiles up to 54 percent., while the lymphocytes remain quantitatively normal or are diminished.

Course. The prefungoid stage may last weeks, months or years, as may also the fungoid. Thus the entire period of the malady is varied and long drawn out. Although the skin, glands and blood usually show the greatest changes, tumors may be present on the mucous surfaces in the mouth, on the vulva or conjunctiva. The termination is invariably fatal. Occasionally the sufferer dies of marasmus, more frequently, however, of some intercurrent infection. The entire progress of the disease requires, on an average, from two to four years, but it may last for ten or fifteen.

Varieties. At times the disease starts with the tumor stage, without the prefungoid period. This form is called mycosis fungoides d'emblée. It usually runs a short course. The lesions are the full blown tumors closely simulating sarcomata, and they arise in restricted areas on previously healthy skin. They often involute without ulceration or necrosis. The ultimate destiny of the patient is that seen in the commoner form.

Differential Diagnosis. In the stage in which the prefungoid lesions resemble diverse dermatoses the diagnosis is impossible. An

alert observer will always be on his guard, however, in cases of inveterate pruritus without obvious cause, recurrent urticaria, prurigo, and the like.

The parapsoriatic, squamous (eczema) dermatitis and erythrodermatous group of lesions always should arouse suspicion of mycosis, particularly the first and third in all cases, and the second when inexplicably obstinate. As a rule it is impossible to know whether these diseases are mycosis until the advent of the fungoid period, and then, of course, the diagnosis is made in retrospect only. It is often possible, though, to reach a definite conclusion in the early stages by histological studies. In early mycosis the same anatomical features are often, though not always, present as in the granulomatous period. A detailed description of the microscopic picture is not possible in this book, but a few essential points may be given. The epidermis resembles that in chronic dermatitis, psoriasis or parapsoriasis. An infiltration is present including the papillae and subpapillary area. It consists of different types of the round cell group, mitotic cells, a peculiar multinucleated cell (the chorioplaque) and a variable proportion of eosinophiles. In lepra, which may be simulated, there are bacilli; in syphilis, vascular changes and plasma cells; in psoriasis, parapsoriasis and squamous dermatitis, the infiltration is purely inflammatory. It must be remembered, however, that a prefungoid dermatosis must lack characteristic anatomical features for a long time, and that an inconclusive microscopic picture is only temporary and most unreliable evidence against mycosis.

In the erythrodermatous eruption, Wilson-Brocq's dermatitis exfoliativa, and Hebra's pityriasis rubra pilaris must be excluded. This follows the lines indicated above. In the last analysis the diagnosis of prefungoid dermatoses depends upon the course of the disease.

In the fungoid stage lepra, tuberculosis, syphilis, leucemia and sarcoma must be excluded. The histological evidence here too is of some value. A fungating lesion roughly may be divided into periods of growth, maturity and involution. In the first stage it is anatomically as described above. In the second it is largely as described, but an even amount of evidence of destruction is seen in the presence of cell detritus. In the last period there is a predominance of cell detritus, or other evidence of necrosis, and no evidence of growth. Absence of lepra bacilli, the type of vascular changes and

absence of the typical structure of tuberculosis rule out lepra, syphilis, and tuberculosis. Nor does the picture resemble that of sarcoma. In leucemia the infiltration is lower down and perivascular. All of the infectious granulomata may be excluded by the absence of prefungoid dermatoses. Lepra may further be excluded by the absence of nerve involvement, syphilis by the absence of the Wassermann reaction and tuberculosis by the occasional presence of bacilli and that of the tuberculin reaction.

Fungus diseases, such as sporotrichosis, blastomycosis and deep ringworm are readily differentiated by demonstrating the specific organism.

Sarcoma has no prefungoid stage and rarely are the lesions so numerous as in mycosis, except in Kaposi's multiple form which, however, is a disease largely restricted to Russian Jews, and different in course, localization, and so on (Chapter XXX).

Leucemia cannot always be differentiated. It is close to mycosis in course, appearance, and other attributes. This will be further discussed below.

Etiology and Pathogenesis. Nothing is known of the cause of granuloma fungoides. There are those who believe it to be infectious, those who consider it a sarcoma and those who regard it as a disease of metabolism. Much may be said in support of each view, and this very fact denotes the futility of further discussion of the topic.

Treatment. The X-Rays furnish the best treatment. Divided doses are preferred. Almost invariably the lesions can be removed by this means and kept under control for years. Arsenic either by mouth or hypodermic injection is of great value. Symptomatic treatment including the dressing of ulcerous lesions, the use of antipruritics, tonics, and in late stages anodynes to relieve suffering, are obviously indicated as need arises.

Prognosis. The disease is always fatal, but by wise measures the end may be deferred in many cases.

LEUCEMIA CUTIS INCLUDING PSEUDOLEUCEMIA

Synonyms. Lymphadenie, Lymphoderma, Perniciosa, Leucemides.

Definition. Leucemia and pseudoleucemia cutis constitute rare conditions in which as a symptom of, or in the course of leucemia or pseudoleucemia, dermatoses arise some of which are lymphatic skin tumors, true leucemia and pseudoleucemia cutis; and others of which

are vaguely associated dermatoses known as the leucemides. The leucemides bear the same relation to leucemia cutis that tuberculides do to skin tuberculosis.

Symptoms. Preleucemic dermatoses, not unlike those seen in early mycosis, are known. They are as follows: pruritus, prurigo, urticaria, simple dermatitis and dermatitis herpetiformis and erythroderma. Pruritus is particularly common in pseudoleucemia (Hodgkin's disease) and may persist for years without evidence of skin involvement. As the glands enlarge the true nature of the disease becomes manifest. The urticarial attacks are fugacious but repeated. Prurigo, unlike the true form, arises late in life, but is otherwise indistinguishable. The erythroderma resembles pityriasis rubra (Hebra). Simple dermatitis is present but extremely pertinacious. In pseudoleucemia the blood picture is not altered, but the glands are enlarged. In acute leucemia there are the usual leucocytosis and lymphocytosis; in chronic leucemia there is a relative lymphocytosis. In pseudoleucemia, as stated, there are glandular enlargements. In leucemia this is also the case, and the spleen is enlarged too. According to Arndt leucemia should be divided into aleucemic, subleucemic and leucemic varieties according to whether the blood picture is altered or whether there is merely a lymphocytosis or that with a leucocytosis. So far as the aleucemic stage is concerned, in its relation to the skin this is nonsense, for we cannot call a disease leucemia unless we know that it is.

The leucemic tumors themselves develop on the face, often giving the picture of leontiasis, and on the scalp, about the shoulder girdle, or extremities, but rarely on the body. They are purplish, reddish or brown, more or less glistening tumors varying in size from that of a bean to a hen's egg, and they are usually numerous. Although they arise after preleucemic dermatoses such as those mentioned, or pigmentations, urticaria or purpuric lesions, they do not arise on these as mycosis develops on prefungoid lesions. They do not break down, and may or may not disappear.

Often in acute leucemia, leucemic infiltrations are found in the mouth, particularly on the tongue and tonsils.

Course. The disease is acute or chronic. The acute form either ends in death in a few weeks or goes into the chronic form. The latter may require years to run its course.

Varieties. The varieties are the preleucemic dermatoses, the granulomata and the leucemides or lymphadenides of Audry. The latter may be erythrodermas, or resemble prurigo or dermatitis her-

petiformis. They differ from the preleucemic forms clinically similar, in having a leucemic minute structure. The leucemides itch, and are often associated with leucemic granulomata.

Differential Diagnosis. In general the same applies to the diagnosis of leucemia cutis that applies to granuloma fungoides. From granuloma fungoides leucemia is distinguished clinically by its tendency to favor the head and extremities, by the blood picture, larger glands and enlarged spleen. However, granuloma fungoides sometimes has a leucemic blood picture, and aleucemic leucemia, if there is such a thing, has no leucocytosis. Under the last conditions, a differentiation is impossible. Pseudo-leucemia is distinguished from leucemia by the blood picture, except in the aleucemic stage of leucemia, and from both leucemia and mycosis by the histology of the glands. The structure of the cutaneous lesions is lymphogranulomatous. This excludes everything diagnostically except lymphosarcoma which cannot always be differentiated. In children chloroma gives the cutaneous picture of other forms of leucemia.

The leucemides resemble prurigo, dermatitis herpetiformis, etc., as already stated. They are distinguished from these by the blood picture, and may be further differentiated histologically if they too have the structure of lymphogranulomatosis.

Mouth lesions in acute leucemia resemble those of syphilis, tuberculosis and Vincent's angina. The Wassermann test, search for tubercle bacilli and Vincent's organisms, as well as the blood count, should prevent error.

Etiology. Nothing is known of the cause of the disease.

Treatment. This is identical with the treatment of mycosis.

Prognosis. The prognosis is bad.

Rare Lymphatic Diseases of the Skin. It follows from what has been stated that mycosis, sarcoma, leucemia and pseudoleucemia have many points of contrast. At times one cannot be differentiated from the other. It is only in typical cases that these rare diseases are interdistinguishable. Still rarer ones have been described undoubtedly belonging in this group.

Leucosarcomatosis Cutis is characterized by lymphogranulomata in the skin with a peculiar mononuclear leucocytosis. It is closely related to chloroma. *Lymphosarcomatosis Cutis* consists of more or less localized lymphatic skin proliferations similar to those of leucosarcomatosis, but the blood picture is normal. *Lymphogranulomatosis Cutis* is a disease so closely related to pseudoleucemia cutis that it is probably a variant of the same. *Lymphoderma perniciosa Cutis* (Kaposi) is a subvariant of mycosis. It follows the same course, but the erythrodermal stage is more marked. Kaposi's case is the only one on record, and he described it at a time when the various forms of mycosis were less well known than now.

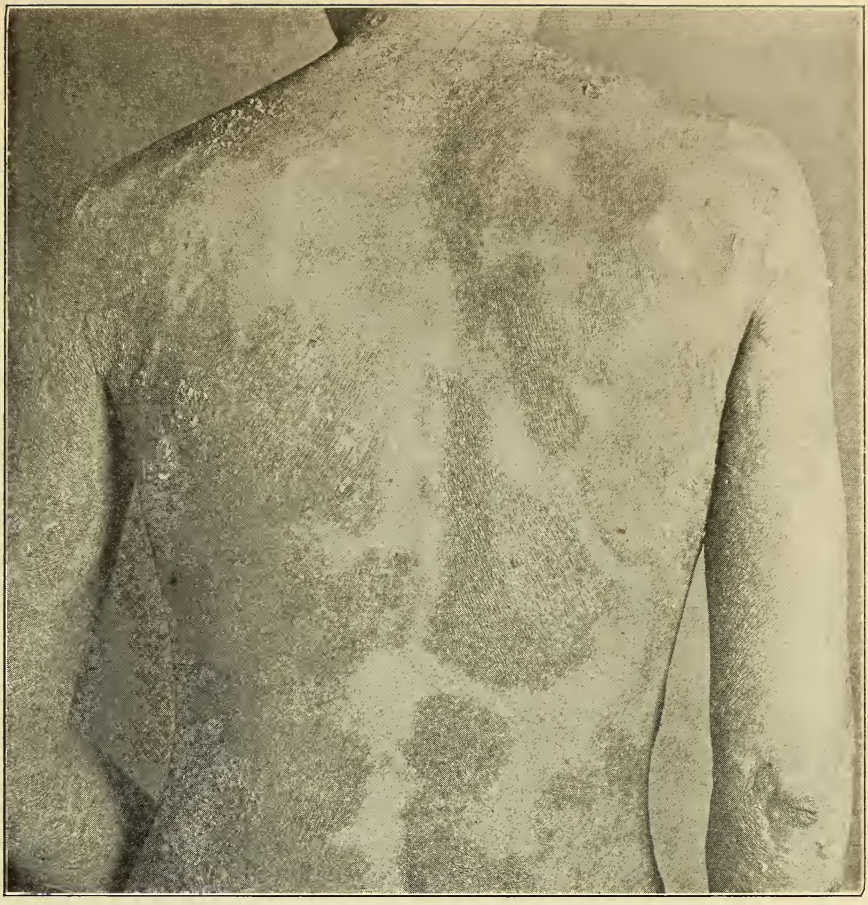

FIG. 67. GRANULOMA FUNGOIDES

The early manifestations of the disease are scaling erythroderma which gradually gives rise to nodules or tumors. Both stages may coexist. Scaling is shown in many areas. The scaling lesions are red and sharply circumscribed. Tumors are found near the elbows in this picture.

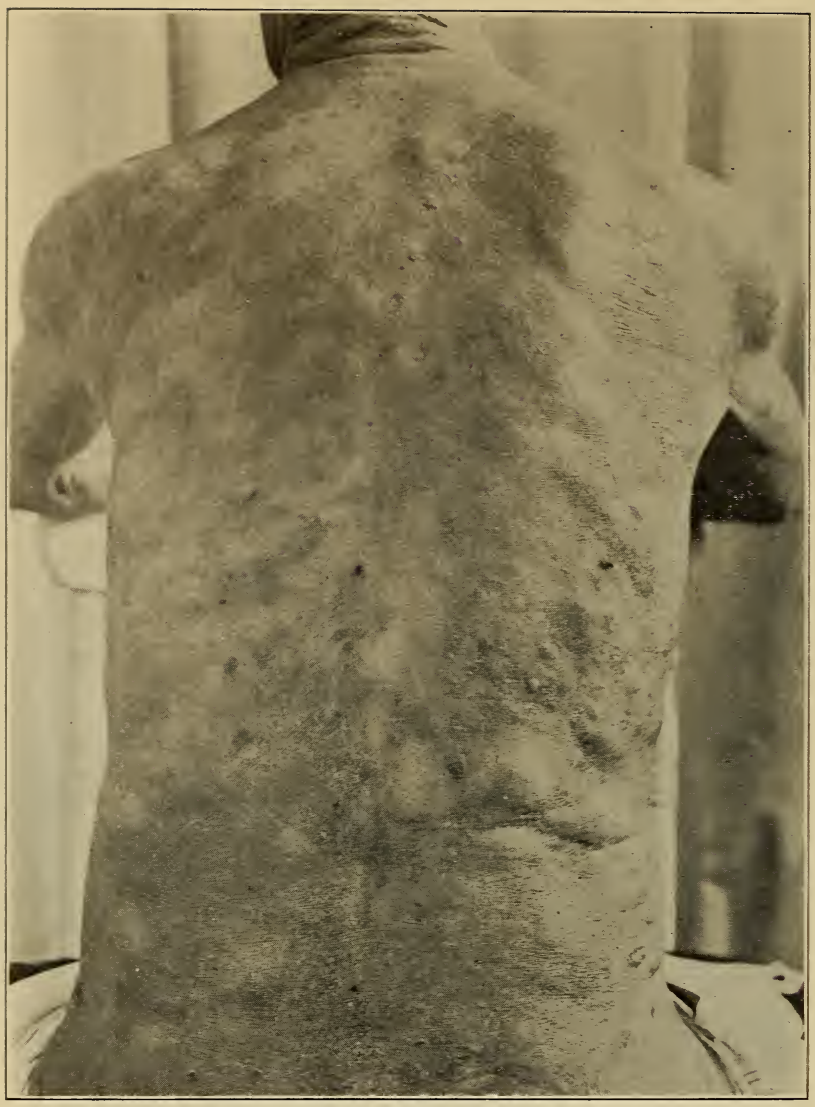

FIG. 68. LEUCEMIA CUTIS

In lymphatic leucemia two types of lesions are found. Non-specific general reactions, called leucemides (Audry) and leucemic infiltrations forming tumors or nodules, as here seen. The latter are composed of lymphocytes. As in mycosis, a preliminary exfoliative stage is often found. Myelogenous leucemia very rarely, and Hodgkin's disease sometimes, produce this picture.

IODODERMA AND BROMODERMA TUBEROSA

The various dermatoses caused by iodides and bromides have already been described (Chapter VII). The tuberous forms merit further attention at this point because in nature's laboratory they represent a nice illustration of non-infectious granulomata. They are due to overdosage or susceptibility. They occur on the face and shins chiefly, and are condylomatous, cribriform, papillomatous or granulomatous lesions resembling the various infectious and non-infectious granulomata. They vary in size up to an inch or two in diameter, are brownish red or purple, often have punctiform abscesses on their flat upper surface and are recognized by the history of drug ingestion and the conventional methods of excluding the fungus granulomata, tuberculosis, syphilis, lepra, mycosis, leucemia and sarcoma. Bromodermata are found in epileptics, children with pertussis, and in nursing infants whose mothers take bromides. Iododermata are most frequently seen in syphilitics, which augments the difficulty of excluding gummata.

The condition is redescribed at this point because iodides and bromides are capable of provoking nongranulomatous dermatoses, an analogy to the state of affairs in mycosis and leucemia. This is suggestive for those of us who seek to explain the causation of the major non-infectious granulomata on a toxic basis. The treatment of the halogen dermatoses is to stop the ingestion of the offending drug. Arsenic hastens involution, and the prognosis is good.

SECTION D. NEOPLASMS AND NEVI

NEOPLASMS AND NEVI

Neoplasms and nevi may with more or less justification be included under one heading. Many nevi are neoplastic; many neoplasms arise from both neoplastic and non-neoplastic nevi. Many neoplasms, however, as will be seen, arise from previously inflamed or normal tissue. Thus it cannot be stated that the relation of neoplasms to nevi is so close as to exclude the origin of new growths from all other sources. And yet a certain intimacy exists between the two groups, closer than that of either with any other class of dermatoses. For this reason, even though somewhat arbitrarily, they may be associated.

GROUP VIII. NEOPLASMS

Neoplasms are new growths the etiology of which is not understood. There is no conclusive evidence that they are infectious, and yet it cannot be denied that this possibility is often suggested. They are not due to disturbed metabolism, and yet different types of neoplasms are so much more characteristic to one period of human existence than another that a metabolic basis for their origin cannot be ruled out. They are not definitely hereditary, and yet in a sense, and in so far as they are related to congenital anomalies, they may be. They are not definitely toxic in origin, and yet human life is so greatly a chemical drama that who will deny the possible bearing of metabolic disturbances upon the pathogenesis of neoplasms? They are surely not due to physical causes but physical factors play as large a rôle in life as chemical, and is it not striking that cutaneous cancer so frequently develops on the lips in men, or in the breasts in women, or on the face in both? It is possible that the clinical picture of neoplasms may be an expression of different forces, and that as the various causes are isolated this tumor will be regarded as infectious in origin, that as congenital, the third as toxic, and so on.

Neoplasms are benign or malignant and these factors depend arbitrarily upon their curability, and the amount of harm they do to the patient either locally, by extension, or remotely, by metatasis. They are of connective tissue origin or epithelial origin, according to whether they arise in the mesoblastic elements of the skin or in one of the skin's many ectoblastic structures. There is another group, the melanomata, sometimes showing characteristics of mesoblastic origin, sometimes of ectoblastic, sometimes of both, but always characterized by a great number of pigment cells and nearly always by great malignancy. In a sense the infectious granulomata are neoplasms. We may exclude them, however, because we know their etiology.

CHAPTER XXVII

BENIGN EPITHELIAL NEOPLASMS

Benign epithelial neoplasms are new growths arising in the epidermis and devoid of harmful potentialities. These new growths may be single or multiple, and there are several varieties, namely the different warts. Warts are also called verrucae, and in French verrue, and German, Wartzen.

Verruca vulgaris or simple wart is a hypertrophic epithelial lesion appearing chiefly on the hands, about the nail bed, and sometimes on the face, genitalia, or finally, anywhere else. The lesions are from two millimeters to a half inch in diameter, single or multiple, and when multiple often grouped or even coalescent. They are yellow, gray or brown, smooth or rough and sometimes cleft, forming digitations giving them the appearance of a stubby brush or coxcomb. They are non-inflammatory but may become inflamed by irritation, unskillful treatment or infection. In this event their base becomes red and they are painful and tender, although ordinarily warts are not sensitive. The cause of the disease is unknown. Certain evidence, chiefly apparent autoinfectability, suggests an infectious origin. At times tuberculosis verrucosa cutis is simulated. In such cases microscopic examination alone would clear up the diagnosis. The youth of the patients excludes the likelihood of epithelioma, but at times skin cancers begin as warts. The treatment consists of removal by means of the knife, curette, high frequency spark or X-rays. The last two methods are the best. Of caustics only trichloracetic acid is to be recommended. About the nail bed or near the eye, X-rays represent the method of choice. The high frequency spark is faster but more painful.

Verruca plana juvenilis is a disease chiefly of infancy and childhood, and rarely seen in adults. Numerous yellow glistening warts appear, chiefly on the face, neck and backs of the hands, although no part of the body is immune. There may be few lesions or hundreds. They are so minute as to be barely visible, and rarely exceed a diameter of two millimeters. They are discrete, clustered or coalescent and no subjective symptoms are present. At times sev-

eral children in a family are affected. They are infectious and Wile has found a filtrable virus. The high frequency spark or the use of trichlor-acetic acid constitute the best therapy. Arsenic internally is also most valuable.

Verruca dorsi manus et pedis are large flat warts on the back of the toes and fingers, particularly the index fingers and thumbs. The warts are found in the middle aged and old. They are treated as verruca vulgaris.

Verrucae or keratoses senilis, also known as seborrhoeic warts, are lesions up to a half inch in diameter. They occur from middle age up, chiefly on the nose, temples and neck, but also anywhere else on the face and often on the body. They are greasy, yellow, gray, brown or slate colored, slightly elevated, scaly lesions. On removing the scales a slightly glistening red serous surface remains. They are often the forerunners of epitheliomata and are easily cured by the use of salicylic acid plaster or trichlor-acetic acid.

Verruca filiformis is a minute brown wart looking like a small thread, a millimeter or two long, and a fraction of a millimeter broad. It occurs in multiple lesions chiefly on the neck, chest and face of women. A similar form which usually disappears spontaneously after confinement, is seen in gravidae. The late Samuel Brickner regarded this type as molluscum molle. As a matter of fact it is hard to tell whether these lesions are really warts, papillomas or fibromas, as they correspond to one papilla covered by epithelium. The best way to cure them is by ablation or the high frequency spark.

A peculiar form is one that might be called verruca multiplex barbae. It occurs in men who shave close. The bearded region is the site of a few or innumerable flat yellowish lesions resembling verrucae planae. They arise near or above a hair follicle and often contain an ingrown hair. The only successful therapy is the use of the high frequency spark.

Treatment. In employing the high frequency spark a spark of ⅛ to ¼ of an inch is used. For small lesions the spark should be allowed to jump from the electrode to the surface to be treated. This swells and turns white in a few seconds. For larger lesions the electrode should be firmly pressed in and the spark allowed to play for about 20 to 30 seconds till the lesion is white and bubbly. It is better to be conservative and repeat the treatment than to be too drastic and leave a depressed scar. After a day or so the treated lesions turn brown, a crust having formed which falls off within ten

days. Warts of the nail bed should be treated carefully by this method, as too destructive an exposure causes disfiguration of the nail. Some warts recur no matter what treatment is used. In these varieties, as well as in warts so situated as to make other treatment unfeasible, X-rays are indicated. A single exposure of four Holzknecht units often suffices to cure.

Connected with verrucae, as with so many other dermatological matters, there is some technical jargon that must be mentioned. Verruca acquisita means a wart acquired after birth. Verruca congenita is a variety of nevus (Chapter XXXII). Verruca digitata indicates a fingerlike proliferation of the papillae composing the wart's structure. Verruca glabra is a smooth wart. In addition to this must be mentioned verruca acuminata already described among the dyskeratoses (Chapter XIV) under the better name condyloma acuminatum; verruca necrogenica or tuberculosis verrucosa cutis (Chapter XXV) and verruca peruana (Chapter XXI), neither of which are warts, but an infective granulomata that looks somewhat like warts.

CHAPTER XXVIII

MALIGNANT EPITHELIAL NEOPLASMS

Malignant cutaneous epithelial neoplasms are new growths which originate in the ectoblastic elements of the skin. Hence they may arise from the epidermis, the pilosebaceous or sweat organs. It is not, however, their embryological source that determines their malignancy, for mesodermal tissues are also capable of producing equally vicious neoplasms. Malignancy is determined by the effect of the growth on the host, and the concept is relative. In other words there are degrees of malignancy. A rodent ulcer is malignant because it does not heal. Neither does it form metastases, however, and in this respect it is less malignant than a prickle cell epithelioma of the lip which metastasizes and kills. A rodent ulcer of the eyelid which ultimately destroys the eye is more malignant, so far as the patient is concerned, than a rodent ulcer of the chest which can do little actual harm. But by destroying the eyelid, and thus causing secondary changes in the eye and orbit, an inflammatory process may arise capable finally of causing meningitis. Thus such a rodent ulcer has incidental malignant attributes which have no direct connection with the epithelioma as such. In the human sense such a growth is malignant, but not in the biological. It is no worse than lupus vulgaris, or a gumma of the nose. On the other hand, a prickle celled epithelioma is worse than either. Its inherent properties are sinister. In forming our ideas of malignancy it is therefore necessary to distinguish between growths, the very nature of which is disastrous to the patient because of certain constant peculiarities, and growths which are disastrous because of incidental peculiarities, such as those of localization or size.

The malignant epitheliomas include, or rather are, that group of neoplasms also called carcinomas. Technically a wart is an epithelioma, though benign, but we eliminate confusion by calling warts vurrucae, and by understanding the important fact that in growth a wart is regular, and that the epithelial cells never become anarchistic as in malignant epithelioma or carcinoma. Another name for mol-

luscum contagiosum is epithelioma molluscum, and in so far as this condition is an epidermal neoplasm it is an epithelioma with the same restrictions as a verruca. Epithelioma adenoides cysticum is no epithelioma at all, but a condition sui generis, and perhaps a nevus (Chapter XXXIII). With these exceptions we understand by the term epithelioma a peculiar growth of epiblastic or ectodermal ancestry, capable of wild proliferation both locally and afar, and harming the individual in whom it develops partly by local destruction, partly by becoming disseminated, and partly by leading to cachexia. In one of these three ways it kills either directly or indirectly.

Among epitheliomata, or closely related to them, are melanomata and endotheliomata. In coming years, when the latter are better understood, they may resume the independent position they once held. They were originally supposed to arise from vascular endothelium, and perhaps, indeed, they do. This has been doubted, and many conditions regarded as endotheliomata have turned out to be baso-cellular carcinomas. Nevertheless enough remains substantiating belief in endotheliomata to warrant retaining the term, while sufficient evidence exists against the belief to warrant including endotheliomata in a chapter upon epithelioma. This would not be justified in a more philosophic work than the present book, and the arrangement is convenient rather than scientifically accurate.

EPITHELIOMA

Synonyms. Cancer, Carcinoma, Cancroid; French Cancer, Carcinome, Epithéliome; German Krebs, Karzinom, Epitheliom.

Definition. Epithelioma or cancer of the skin is a more or less malignant epithelial new growth characterized by a tendency to unrestricted proliferation with the ability, in certain forms, to invade local or remote glands, or to form matastases. There are primary types, of which the above is a definition, and secondary types, which are skin metastases derived from neoplasms elsewhere.

The symptoms and course of epitheliomata differ in the different types so that each variety will be considered individually in these respects, in a manner not conforming with the general arrangement of this book. Many classifications are to be found in the literature, and for the deep student all have interest. For the practical clinician, however, no classification excels that of Darier, which, with some modification, will serve as a model. It will further simplify matters to consider cutaneous and mucous surfaces separately.

Cutaneous Epithelioma

I. Primary.
  1. Prickle Celled.
    A. Deep type.
    B. Superficial vegetating type.
      a. Hyperkeratinized form.
      b. Denuded type.
  2. Basal Celled.
    A. Flat Cicatricial form.
    B. Rodent Ulcer.
    C. Deep ulcerating form.
    D. Cylindroma.

II. Secondary.
  1. Papulo-tubercular.
  2. Cancer en cuirasse.
  3. Paget's disease.
  4. Acanthosis nigricans.

III. Pigmentary epithelioma.
  1. Primary or nevo-epithelioma Melanoma.
  2. Secondary or metastatic melanoma.

IV. Endothelioma.

Mucous and Muco-Cutaneous Epithelioma

1. Buccal and palpebral.
2. Male and female genitalia.
3. Perianal.

primary cutaneous prickle cell epithelioma (figs. 69, 70, 72, 73)

Synonyms. Squamous celled, lobular epithelioma; French Epithéliome Spinocellulaire, Epithéliome Lobulé; German, Fasernepithelkrebs; Latin, Epithelioma spinocellulare.

Definition. Cutaneous prickle celled epithelioma is a malignant neoplasm originating from the prickle layer of the epidermis. It is characterized histologically by the presence of epithelial pearls and other evidences of complete keratinization. Clinically it shows a marked tendency to voluminous growth and lobulation, with or without necrosis, and in its course it tends to be most malignant, usually involving the regional lymph glands, and throwing out metastases, however rarely, to remoter regions of the body.

Symptoms. The disease begins as a small scaling papule, large papule, or abrasion on the normal skin, or upon one of the so-called precancerous lesions. It grows very rapidly or extremely slowly, or at first remains stationary and then grows more or less rapidly. Scales or crusts form which adhere firmly and, when forcibly removed, leave a glazed, oozing or bleeding, red surface surrounded by a more or less dense raised margin which tends to roll and is pink, red or buff. The margin is hard and the entire lesion feels so too, when pinched. As times goes on the surface becomes lobulated (Fig. 69) and the lesion is red, purple or brown. The lobulations are irregular in size and volume, sometimes resembling hypertrophic granulation tissue, sometimes cerebriform, sometimes suggesting cauliflower, but always bleeding easily with the slightest trauma. A serous or sero-purulent substance is secreted which dries easily into yellow, gray or dirty brown adherent crusts. Very often necrosis takes place and a crateriform ulceration develops, the floor of which has the characteristics mentioned above, the margin of which is undermined and vegetating or rolled. Pressure of the mass causes the extrusion of fusiform or caseous wormlike particles consisting of cell detritus or cheesy pus. The extension is not only on the surface but downward, and it involves any deeper tissues, even bone, that seek to obstruct it. Regional glandular involvement usually takes place, and rarely general metastases are encountered. The glands are hard. The growth hurts and pains, but the glands do not, as a rule. Three fourths of these cancers appear on the scalp and face, largely near the lips. The remaining quarter appear anywhere else on the body, but chiefly near the muco-cutaneous junctions. An entire lip or eyelid, the glans or anal margin may be melted away.

Course. The course may cover months or years, but is always very rapid after the vegetating stage has set in.

Varieties. There are two varieties, the papillary or hyperkeratinized form, and the papillary form devoid of epithelium. The former may occur anywhere but usually arises on the face, lips or neck, and at times on the exterior sides of the extremities, hands, feet and digits. Its origin is commonly in a senile wart. It grows with extreme deliberation, crusts, scales, fissures, bleeds, ulcerates, and finally assumes the features of the deep type described above.

The denuded variety is largely confined to the muco-cutaneous junctions; is voluminous, red, velvety, glazed, and finally is converted into the deep variety following the latter's course.

Diagnosis. The diagnosis rests upon clinical and microscopic

data. Clinically the long duration of the disease, the fact that a preceding dermatosis (senile wart, scar, leucoplacia, lupus vulgaris, etc.) existed, the appearance of the growth, its site, and the glandular involvements are the significant points. The histological appearance is characteristic and serves as the proper guide for all differential diagnosis. It must be remembered, however, that the probatory excision is not without danger, as biopsy frequently stimulates growth. Thus small lesions should be excised in toto and examined, the excision being the therapy. The excision of large lesions is a pretentious operation. Thus small pieces should be removed and examined immediately in frozen sections, the patient awaiting the report, and the surgeon prepared forthwith, upon an adverse verdict, to remove the growth and to extirpate the regional glands. Should the findings be negative the growth should nevertheless be entirely removed provided all other confusing conditions have been excluded. Thereupon a most searching histological study should be made, and if a diagnosis of malignancy is reached the local glands should soon be extirpated and examined for metastases.

Early epitheliomata clinically resemble warts, and ulcerating epitheliomata resemble tuberculosis, syphilis, the fungus granulomata and other malignant growths. The histology is characteristic in all instances. Syphilis should be excluded on clinical and sero-logical grounds, but the positive Wassermann test does not exclude the possibility of cancer arising in a syphilitic. The finding of specific pathogenic organisms in the various infections is conclusive.

Etiology and Pathology. To discuss the etiology of epithelioma is futile, as nothing definite is known. Despite an outlay of millions of dollars for cancer research, and an incalculable cost of time and intellectual energy our grandfathers knew as much of the causation as we do. A few facts stand out. Epitheliomata usually arise during and after middle age. Some types, as the labial for example, are commoner in males; others, as the mammary, in females. Men smoke and women suckle, so that the trauma appears to play a rôle. For the most part the growths develop on the face where glandular activity of the seborrhoeal sort is common; thus functionally active epithelium appears to be a favoring item. Certain conditions known as precancerous dermatoses are predisposing causes. This question will be considered in detail in the next chapter. Certain kinds arise in nevi. Followers of Cohnheim's views believe that all epitheliomas, including those of nevus origin, spring from embryonal cell inclusions predestined to cancerous eventualization. Some writers

ascribe the disease to infection. Sponsors of this view have succeeded in proving it about as well as alchemists succeeded in their labors for synthetic gold. That a metastazing epithelioma has many features suggesting infection is true enough, but this attribute is confined to the growth, and the transplantation occurs in the host. There is not an authentic instance recorded of one person's having infected another, nor has the presumptive agent been isolated. Heredity too is invoked on the usual specious grounds, but no true evidence thereof exists save that it has been known to occur in several members of a family. This may also be said of corns, bunions, chronic nephritis, and gout, and yet who ascribes these diseases to heredity? Epithelioma is a common disease and it therefore may be assumed that familial instances need no further explanation than coincidence. Bulkley ascribes cancer to excessive proteid ingestion. This is arrant nonsense.

Treatment. Little need be said about treatment. The method of election is extirpation both of the growth and the invaded glands. Undoubtedly X-rays and radium cure many cases, particularly when the lesions are still young and small. On the other hand, under these conditions there is certainly no good reason to give excision a place of second rate importance. Radiotherapy is to be preferred only when patients decline surgery or when surgery would cause a cosmetic defect, as would often be the case in facial growths. Radiotherapy is always indicated after surgery as a prophylactic measure. When X-rays are employed to destroy a growth a massive dose of eight Holzknecht units should be given. When used prophylactically, after excision, the scar and region of the removed glands should be irradiated two or three times at weekly intervals by the cross fire method and with one or two Holzknecht units at a time. Equivalent doses of radium may be employed, but radium has no essential advantages over the Roentgen ray.

In other times chemical agents, curettage, fulguration and the actual cautery were used. These methods have been, in general, discarded. The only valuable chemical agent is acid nitrate of mercury. The skin is cocoainized, the growth thoroughly curetted, a sixty percent. solution of the mercurial salt is swabbed over the bleeding surface, and after a moment a dry dressing of pure sodium bicarbonate powder, affixed firmly, is applied. The bicarbonate neutralizes the mercury. A scab develops which falls off in about two weeks. This method is useful in people who refuse surgery and fear the X-rays. The actual cautery is as good as the knife when

broadly applied, but the cosmetic result is horrible. Simple curettage and fulguration have been abandoned as they appear to stimulate recurrences.

Prophylaxis is important. People at the age subject to epithelioma should be cautioned to consult reliable physicians for skin lesions that grow or show no tendency to disappear. They should be cautioned not to pick, irritate or treat inexpertly such lesions. Physicians examining such patients should take nothing for granted, but should excise the lesions, and after a microscopic examination has been made should select suitable therapy.

Prognosis. In early lesions the prognosis is good provided the treatment is wise. Excision is the best. Even so, local recurrences may take place or metastases may arise from glands early invaded. Late growths always render the outlook grave because the glands are likely to have become involved. Untreated prickle celled epitheliomata always are fatal.

BASAL CELLED EPITHELIOMA (FIG. 71)

Synonyms. Rodent Ulcer, Noli Me Tangere, Jacob's Ulcer, Ulcus Rodens, Cancroid.

Definition. Basal celled cancer, of which the rodent ulcer is the best known example, is a superficial epithelioma the course of which shows great chronicity. It tends to cicatrize and at times is capable of spontaneous healing. In that it neither involves the lymphatics nor metastasizes it is biologically benign; in that it may cause profound destruction, and in that it may become the gateway of fatal secondary infection it is potentially malignant.

Symptoms. The growth begins slowly, as a rule, on the face, particularly the temples, near the canthi of the eyes, nose, nasolabial fold or on the ears. Its first stage is a papule arising on previously normal skin, or on pre-existing lesions such as abrasions, warts, verrucous nevi, senile warts or in chronically desquamating areas near sebaceous glands. The papules are minute and waxy in appearance while their color is yellow, buff, reddish or a muddy gray. The papule extends circumferentially and breaks down centrally. Its growth is indolent indeed, and may become stationary for months or years at any stage. The central ulceration is covered by a firmly adherent crust, the removal of which discloses a yellowish red, slightly moist, varnished looking and extremely shallow, saucer shaped base, which either rapidly crusts again or even cicatrizes gradually. The margin is flat or rolled and, as a rule, is studded

with elevations looking like the original papules, but tending to resemble pearls.[1] This is the pearly margin of the rodent ulcer and is distinctive thereof. The margin is red, pink or yellow, hard, and shimmers with a waxy lustre. The scar may be smooth or slightly corrugated.

The massiveness, verrucosity and lobulation, and the undermined margin of the prickle celled growths are lacking in the less voluminous and superficial surface neoplasm called basal cell epithelioma. At times the scarring takes place completely within, while only the characteristic margin, extending in festooned, circinate or gyrate manner, indicates peripheral activity. Such lesions suggest the tubero-papular, ulcerating syphilide. No glandular involvement or metastases occur.

Course. The lesions are indolent, their evolution sometimes requiring years, decades or even a generation, during which time the diameter of the growth may not exceed an inch, or it may include half the face. Although spantaneous healing may rarely occur the lesions usually progress indefinitely unless properly treated.

Varieties. The discoid form begins as does the type already mentioned. It often throws out neighboring daughter lesions. Otherwise it behaves as the rodent ulcer. It may, however, when stimulated, become malignant, assuming the attributes of the prickle cell form. A subvariety of the discoid form is the morphoea-like or sclerodermatous epithelioma which looks like a circular disc, the diameter of which may reach an inch, and which suggests cardboard implanted in the cutis. This growth is white or yellow, traversed by dilated vessels, and marginated by "pearls." It too grows indolently and may exulcerate. Hutchinson described a crateriform ulcer in this group, characterized by deeper involvement than that ordinarily found, but with the usual type of margin. A massive variety with little tendency to ulcerate favors the scalp. Microscopically this form consists of tubularly arranged rows of basal cells. This is the cylindroma of Billroth and Melassez often confused with endothelioma or sarcoma. It is benign.

Differential Diagnosis. The slow growth, benignity, tendency

[1] The use of the word pearls in this connection is one of those mishaps of nomenclature so unfortunate in scientific language. It causes confusion with the term epithelial pearls found microscopically in spinocellular epithelioma, while rodent ulcer is never spinocellular. The use of the word in basocellular epithelioma distinguishes a clinical feature. Actually the microscopic epithelial pearls of prickle cell cancer suggests onions, and the clinical pearls of rodent ulcer resemble little globules of wax.

to scarring, and appearance, render the clinical diagnosis easy. The only two conditions with which it might be confused are syphilis and spinocellular epithelioma. The last consideration is academic. A microscopic examination would clear away all doubt.

Etiology and Pathogenesis. There is nothing already stated of prickle celled carcinoma that does not apply to the basal cell type. Nothing is really known of the causation.

Treatment. The therapy corresponds to that of the more malignant form. Recurrences are rarer than in the latter.

Prognosis. With proper therapy the cases are practically always curable.

SECONDARY CUTANEOUS EPITHELIOMA

This group of cutaneous carcinomata arises by involvement of the skin through dissemination of malignant metastases from a primary focus elsewhere in the organism. The daughter growths may be conveyed by means of the lymphatics from great distances to the skin, or may involve the skin by direct extension. A rectal neoplasm, for example, may become cutaneous by downward proliferations. Microscopically the cutaneous lesions exhibit the attributes of the parent neoplasm. The skin metastases may ulcerate or not.

Papulo-tubercular carcinoma is that metastastic form in which malignant deposits are lodged subcutaneously, and grow in situ, forming lesions of all sizes, prevailingly up to a half inch in diameter, although some grow as large as an egg. The lesions are convex, hard papules or nodules. Their color is buff, brown or purple. They lie in the subcutaneous tissue or corium and push up the normal skin which is traversed by dilated vessels, in a manner quite characteristic of the disease. They are the far flung descendants of remote visceral ancestors whose native structure is microscopically revealed in the cutaneous growths. They may also arise as recurrences in scars of extirpated rectal, mammary or cutaneous neoplasms.

A special variety of this form, which may be primary or secondary, is *cancer en cuirasse* or *scirrhous carcinoma.* The lesions develop as shiny buff or red lenticular papules which coalesce over the whole chest or abdomen, into a hard surface. The skin feels leathery and binds down the ribs so that respiration is impeded. The entire growth suggests, in shape and localization, the breastplate of heraldic armor; hence its name. The margin is indistinct and throws out proliferations. Because of lymphatic infiltration and involvement

of the axillary glands and vessels, edema or elephantiasis of the upper limbs is provoked. The skin is shiny, yellow, red, telangiectatic and at times ulcerated. The entire body may be involved. Fortunately the disease is rare, as it is invariably fatal. The only therapy is the use of X-rays.

Paget's disease or eczematous epithelioma of the nipple is rare. It begins, or rather first is observed, as a serous dermatitis of the nipple, and later mammary cancer of a highly malignant type, invading the skin, develops. The exact interrelationship of the two processes is not understood. Women are affected more than men. Extra mammary forms have been described.

Acanthosis nigricans (see description Chapter XV). This disease in adults denotes carcinoma of the abdominal viscera. With removal of the internal growth the cutaneous condition has been known to have disappeared. The disease is in no sense epithelioma itself, but is provoked by visceral tumors of this character. Thus it is mentioned at this point. It is rare, but significant, and its existence should not be forgotten or overlooked by the alert clinician.

PIGMENTARY EPITHELIOMA OR MELANOMA (FIG. 76)

Pigmentary epitheliomata are primary or secondary. The primary forms arise from pigmented moles and are usually single. Any part of the body may be the site. The lesions are slate gray, blue, brown or black and assume the appearance of the deep or prickle celled epithelioma. Thus they are verrucous, papillomatous, cauliflower shaped, or corrugated, and often ulcerate. They are rare, highly malignant, matastasize throughout the body or into the skin, and structurally resemble the nevi from which they spring, plus epithelioma containing large numbers of pigment cells.

The secondary variety arises in much the same way as any other secondary epithelioma, from mother neoplasms of the pigmented type. This form is multiple from the outset starting as tiny dark brown or black dots which grow into the fungating form. Both types are highly malignant. The treatment is the same as that of ordinary epithelioma, but the prognosis is always bad.

ENDOTHELIOMA

Theoretically endotheliomata are not particularly malignant neoplasms, originating from vascular endothelium. Spiegler's cases were probably true specimens of the disease. The lesions vary in size, some reaching the dimensions of a small orange, but the ma-

jority being smaller. They favor head, neck and trunk, and are usually multiple. They are resistant but not stony hard, and they show wide variations in color from orange to blue or purplish. They grow slowly. Often there is a close histological similarity to basal cell epithelioma. The entire subject is still vague.

MUCOUS AND MUCO-CUTANEOUS EPITHELIOMA

Mucous and muco-cutaneous epithelioma are prevailingly of the prickle cell type and thus very malignant. They easily recur or form metastases, and if treatment is begun too late, they kill. In clinical respects they show no difference from the cutaneous growths of like type. Certain peculiar details, however, deserve special mention. Only the commoner forms will be discussed.

Epithelioma of the Lip. This disease is seen much more in men than in women. Thus it is possible that smoking is a causative factor. Regional lymphatics are rapidly involved and the course of the malady is highly malignant. The only proper therapy is radical excision and extirpation of the affected glands. Subsequent X-ray treatments, designed to prevent recurrences, are to be advised.

Epithelioma of the Tongue. This variety is also commoner in men than women, but the ratio between the two sexes is not so great as in the labial form. Whereas the growth is usually single there may be multiple lesions. The site is on the dorsal aspect near the edge of the tongue, opposite the six-year molars. As a rule a crater like ulcer, voluminously infiltrating the tongue, is exhibited. The lesion is extremely hard, often tender and frequently instead of an ulceration a fungating mass is present. A syphilitic leucoplacia often furnishes the starting point. The glands are involved early and rapidly. Ptyalism is present, and a suggestive symptom is severe pain radiating to the ear on the side corresponding to the lingual lesion.

Tuberculosis and syphilis may be confused with cancer of the tongue. Tubercular ulcers are multiple, more superficial, the margins are not rolled, but shallow and delicately grooved, and the site is nearer the tip, on either the upper or lower surface. It is easy to find tubercle bacilli in smears from the ulcers.

Gummata are usually multiple and dorsal, otherwise they closely resemble cancer. Inasmuch as lingual cancer so often arises in specific cicatrices, leucoplacia or from gummata themselves, a positive Wassermann test does not exclude malignancy, and unless this fact is clearly understood serological diagnosis, here applied, is a

menace. Only a negative Wassermann test is of value as favoring cancer. If the serum test is positive arsphenamin should be given diagnostically, and if after two injections at a week's interval the growth has not involuted to half its original volume or less, it ought to be wholly excised, examined at once in frozen section, and upon a diagnosis of malignancy the already anesthetized patient should immediately have his tongue resected and the involved glands extirpated. Roentgen therapy should follow. A physician temporizing in this matter should regard gently Cain, Nero, Attila or Lucretia of Borgia.

Lingual gummata do not cause glandular enlargements as does carcinoma. The prognosis of the disease is always bad, and only intelligence and prompt action on the physician's part furnish to the patient a fighting chance.

Lingual cancer may also simulate a chancre. The same general problems are involved as with gummata. In a chancre there are enlarged glands, no pain in the ear, and the lesion, when it looks like a cancer, is likely to be so advanced that there are already evidences of secondary syphilis. The Wassermann reaction may be present. Spirochaetes may be found, but their identification is difficult because of their resemblance to spirochaeta microdentium. The therapeutic diagnostic test should be made if necessary, the subsequent procedure and therapy being similar to what has been stated above.

Cancer of the penis, aside from melanoma, is usually of the ordinary squamous cell variety. It too resembles gumma and chancre and the differential diagnostic points are the same, and have the same significance, as in linguinal carcinoma. Finding the spirochaetes is easy and is of great value in diagnosis. Malignant neoplasms of the vulva are usually prickle celled and the questions involved differ in no wise from those already discussed in other parts of this chapter.

In conclusion it may be reemphasized that the diagnostic problems connected with cutaneous cancer throw a tremendous responsibility upon the physician. If he is not prepared to meet them intelligently he should not fail to seek proper aid. Early recognition often converts malignant potentialities into benign ones, and there is no excuse for errors, for the matter is simple, straightforward and within easy grasp of average intelligence.

CHAPTER XXIX

THE SO-CALLED PRE-CANCEROUS DERMATOSES

Over twenty years ago Dubreuilh classified into a group certain cutaneous affections which he regarded as forerunners of epithelioma. He designated them as pre-cancerous dermatoses and his views were accepted without critical analysis because in many instances the facts as conceived by him were true enough. In 1912 Bowen of Boston described a condition as the "precancerous dermatosis." This malady was sufficiently characteristic in its objective attributes to warrant isolation as a clinical entity, and cases were described by Darier in Paris and by the author of this book in New York. There are now seven instances of the malady on record. Critical study of the cases, however, indicates clearly that since four were actually cancers they could scarcely be termed precancerous, and since the remainder showed nothing epitheliomatous it was gratuitous to call them precancerous, in that there was no way of foretelling whether they would ever become malignant or not.

Thus it appears absurd to accept the designation "precancerous" into our dermatological vocabulary. It is perhaps meticulous to be a purist as to scientific diction, but the term "precancerous" appears to be peculiarly vicious since it contains implications at variance with fact. Precancerous means a condition invariably terminating in cancer. Applied to dermatoses we know that this is not true. We know that very few lesions of this sort ever become malignant. We know, too, that epitheliomata as frequently arise from apparently normal as from abnormal skin. This being so, why not call skin itself precancerous, or, to bring out the absurdity more clearly why not call the human being precancerous? Why not call smoking a precancerous habit, or lactation a precancerous function, or the fifth decade of life a precancerous decade, or chimney sweeping a precancerous vocation? Or if we wish to apply the principle broadly why not call raw oysters a pretyphoid diet, Peyer's patches pretyphoid structures, or streptococcus tonsillitis a prerheumatic or prenephritic disease? We do not because these concepts are manifest nonsense.

The early stages of a cancer may look like a seborrhoeal wart, but not every seborrhoeal wart becomes a cancer — in fact a very small proportion do. There is nothing in the clinical appearance or minute structure of a given precancerous lesion that enables a justifiable prophecy as to future malignancy to be made, and more than we can select a particular point on the normal skin with reference to such a prediction. A further danger in employing so unsound a term as precancerous is a purely human one. People have a well-grounded horror of the word cancer. The average person collapses when told he has a precancerous lesion. It is impossible for a layman to know what the term really means, and it suggests such things as the guillotine, coffin or eternal damnation. It is enough to advise a patient to have a suspicious lesion removed without frightening him with a meaningless word. The expression "precancerous" has little, either humane or scientific, in its favor.

What little there is of scientific sense must also be mentioned. There are certain skin lesions which more frequently terminate as cancer than other skin lesions. These have been termed precancerous. The expression should not be regarded as implying that they always become malignant, for in comparison with the number that do, infinitely more do not. Thus what we really mean in employing the word is that we are designating a cutaneous manifestation which has, in all human experience, certain well defined malignant propensities. Unquestionably a cancer does not spring up spontaneously, and in retrospect it is perfectly reasonable to believe that a cancer must have had a precancerous phase. But when it was in this phase, or when it ceased to be precancerous and became a cancer, we are unable to state of any growth. There is no name to substitute for precancerous as the concept itself defies intellectual grasp.

It is sufficient to remember that there are skin conditions which seem to be the forerunners of cancer. It is equally important to bear in mind that cancer just as often arises from skin which has previously appeared normal. The practical point is to be suspicious of all neoplasms, regardless of whether they arose upon healthy or unhealthy skin, with a certain conservative bias in the direction of safety, namely that certain lesions favor malignant evolution. It is entirely unnecessary to give such lesions a grotesque and terrifying name. All that a scientist needs is to record his experiences in his memory in order that he may apply them to the welfare of his fellow men. To label experiences, particularly unsoundly, indicates

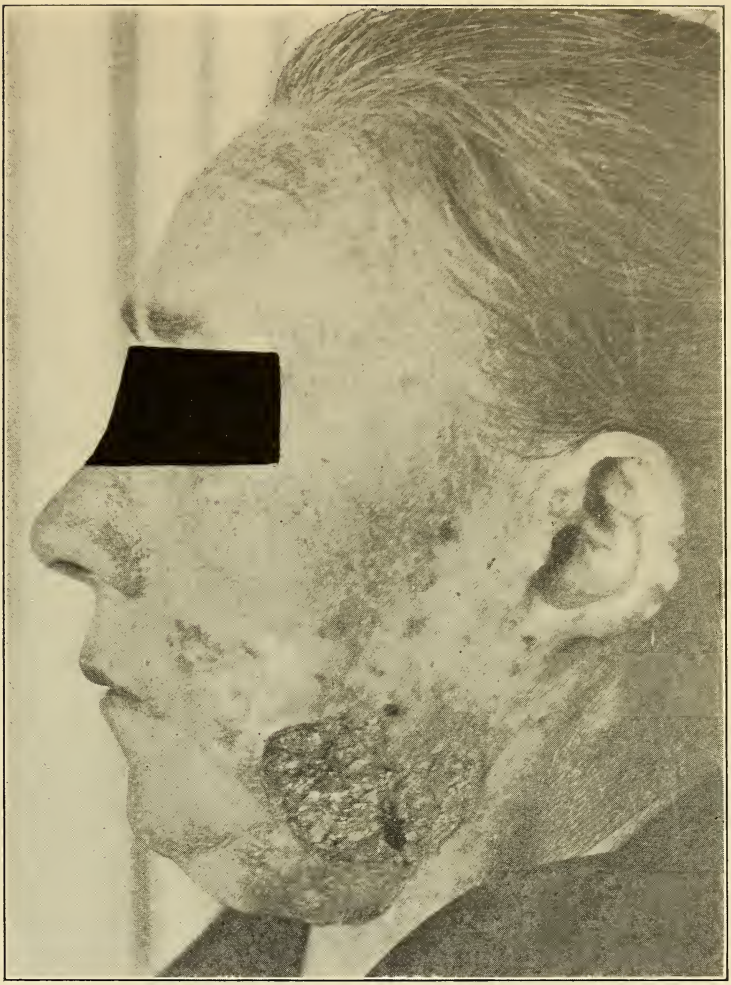

FIG. 69. SQUAMOUS CELL EPITHELIOMA

This lesion is relatively superficial and has a rolled margin, but the floor is more vegetative than would be expected in a basal cell growth.

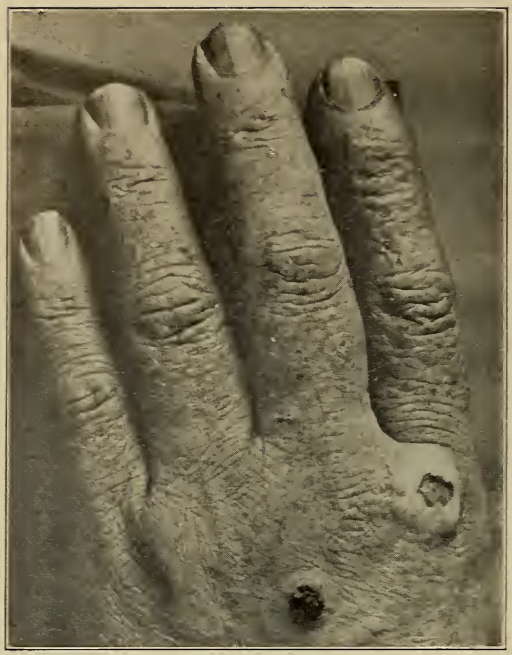

FIG. 70. EPITHELIOMA, SQUAMOUS CELLED TYPE

Note the voluminous character of the lesion.

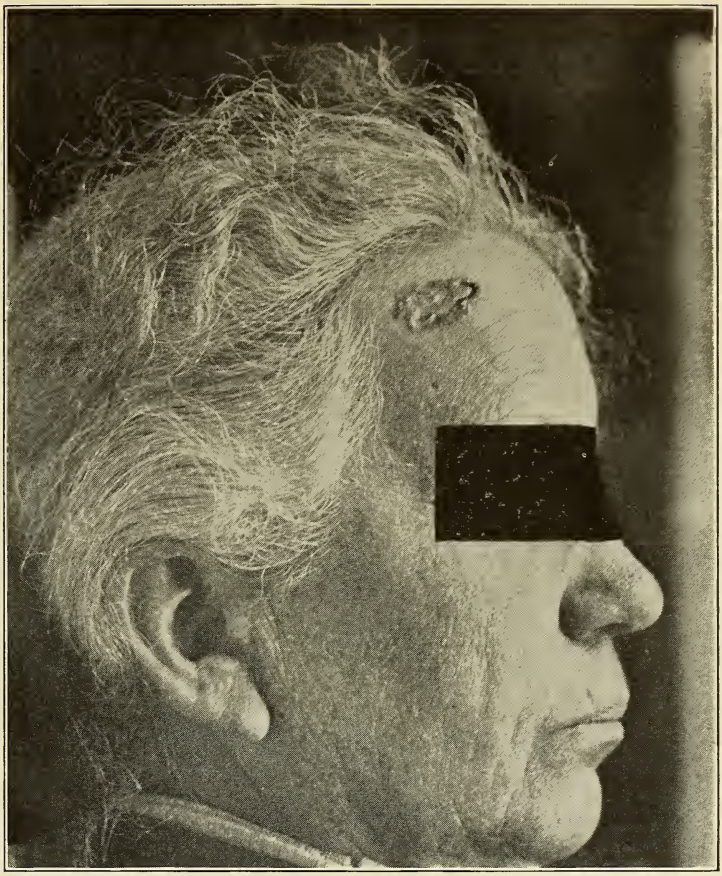

FIG. 71. RODENT ULCER

The site, the rolled pearly margin, contour and superficiality of the lesion are characteristic.

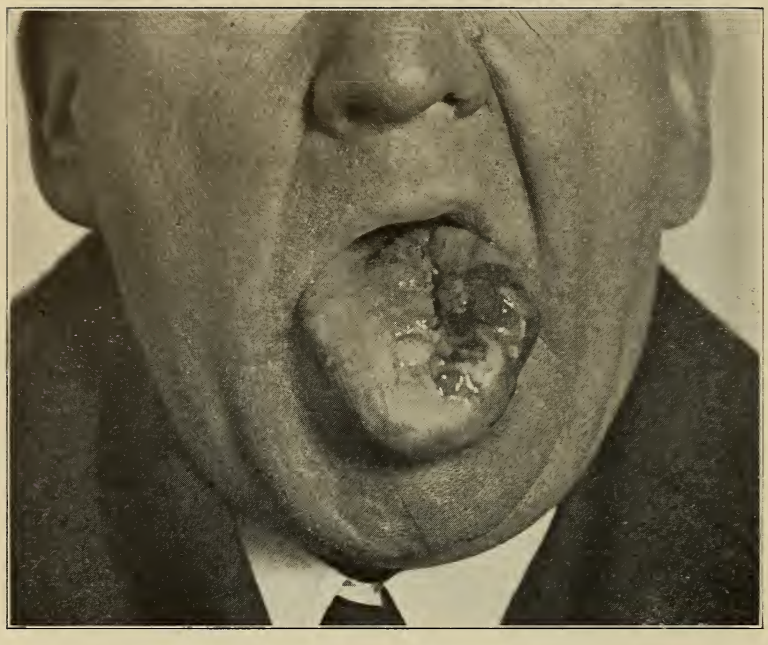

FIG. 72. EPITHELIOMA OF TONGUE

Note the voluminous character of this fungating lesion, and its situation at a point opposite the molars.

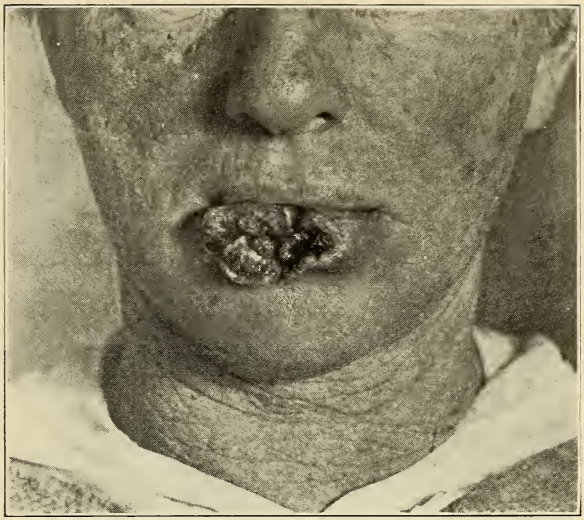

FIG. 73. EPITHELIOMA, LIP

Epithelioma of the lip is less frequent in women than in men. This example is typical of the advanced, fungating variety.

mental limitations to which a true scientist points with anything but pride. The so-called precancerous dermatoses are numerous and are designated in the ensuing table. Of these nevi, xeroderma, Bowen's dermatosis, leucoplacia, horns, lupus vulgaris, Roentgen burns and scars are of unquestionable importance. From the rest epithelioma undoubtedly arises sometimes. Sailor's skin, arsenic, paraffin and soot epitheliomata are well enough known, but extremely rare. Dermoid cysts, and for that matter ordinary sebaceous cysts, sometimes give rise to malignancy but this is exceptional. Paget's disease of the nipple has been discussed (Chapter XXVIII) and indeed it cannot be stated whether the dermatitis is "precancerous" or perhaps secondary to the mammary growth and hence postcancerous. Of the conditions enumerated at the opening of this paragraph xeroderma pigmentosa, Bowen's dermatosis and Roentgen carcinoma form definite pictures.

Seborrhoeal Keratomas (Fig. 37) arise in two ways. They are either exaggerated seborrhoeal plaques, and hence inflammatory in origin; or they are the result of senile regressive skin changes. They look like scaly, yellow, brown, buff or dirty gray papules, single or multiple and of a diameter rarely exceeding a half inch. They arise upon the face, chiefly near the nose or temples, and are roughly circular or oval. The scales are adherent, and when removed leave an oozing surface which first crusts and then scales. They may persist with relatively little change for years, and they develop from middle age on. It is easy to cure most of them with 10 percent. to 20 percent. salicyclic acid plaster, and when there is recurrence or when they are refractory they readily yield to a single X-ray exposure of 4, 6 or 8 Holzknecht units, according to their size and thickness. A small proportion of them go on to the formation of epitheliomata of either the basal or prickle cell type. It might be truer to state that epitheliomata in their early stages clinically resemble some seborrhoeal warts, since only very few of these lesions ever grow malignant. The practical point to remember is that all seborrhoeal keratomas are suspicious and hence should be properly treated.

Leucoplacia Buccalis (Fig. 74) is almost invariably syphilitic. At times these lesions give rise to prickle cell epitheliomata. As already stated (Chapter XXVIII), although nearly all lingual carcinomata arise from leucoplacial soil, the coefficient of this eventuality is small. Nevertheless, conservatism demands due suspicion in the patient's interest, and the slightest sign of activity in leucoplacia

is an indication for imperative measures, as indicated in the previous chapter.

Horns of the skin are hyperkeratoses, the origin of which is similar to that of ordinary warts. They appear wherever senile keratoses may, on the penis, rarely on the trunk and extremities. There is a rough similarity to the horns of herbivorous animals, but the growths are short. They may be conical, cornuate, tortuous and present a yellow to black furrowed, cracked surface. As a rule they do not exceed an inch in length, are situated upon a noninflammatory base and may spring from normal skin, a lupus vulgaris scar, seborrhoeal keratoma, sebaceous cyst, or wart. A very small number become malignant. The treatment is excision followed by X-rays if evidence of carcinoma is found microscopically.

Lupus Vulgaris (Fig. 61) is a common disease. Now and then epithelioma arises in the patches or the lupus scars. The ratio is unknown, but the fact need cause no astonishment for there is nothing in tuberculosis calculated to preclude the development of cancer. Thus the inclusion of lupus among precancerous dermatoses seems particularly forced. Ulcers and fistulae belong in a similar category, and for the same reasons. Inveterate psoriasis and lupus erythematosus even more rarely terminate in malignancy, and the relation to carcinoma of chemical agents such as arsenic, paraffin and soot, considering how great is our exposure to them, is strikingly remote.

Dermoid Cysts and sebaceous cysts rarely are the starting points of malignancy, but should be regarded in their potentialities as suspicious. Unquestionably scars are often the starting point of carcinomas, and particularly the scars of syphilis (lingual, especially), lupus and burns of any kind. It must be recalled, however, that syphilis and burn cicatrices are numerous and common, and that the development of cancer in them is relatively negligible, however great the actual number. Thus their incrimination as precancerous is extravagant. Almost, but not quite so far fetched, is the sailor's skin cancer.

In the last two paragraphs a series of conditions has been enumerated which sometimes precede epithelioma. It is questionable whether their relation to malignancy is causal so much as coincidental. As a mattter of fact they do not give rise to epithelioma nearly so often as does apparently normal skin, and if we were to be panicky whenever one of the above lesions were observed at the cancer age we would do well to subject every individual of forty

or over, even with a healthy integument, to a universal exposure to eight Holzknecht units of Roentgen rays. With all due respect to the terrors of malignancy we must avoid becoming a race of hypochondriacs, for carcinomaphobia is no less crazy than any other form of insanity. If we wish to be sanely careful let us bound our concepts of precancerous dermatoses within reasonable limits, rather than extend them to include all the skin diseases that there are. We should be careful but remain wholesome.

The relationship of nevi, Bowen's disease, and Roentgen dermatitis to cancer is another matter. Pigmented nevi, as already stated (Chapter XXVIII), sometimes become malignant. This is not frequent, but melanomata are so serious that all pigmentary moles deserve earnest consideration. Only seven cases of Bowen's "precancerous dermatoses" have been published. Four were frankly malignant. This indicates the inadequacy of the term, but emphasizes the gravity of the disease. The malady occurs anywhere on the body, but favors the regions peculiar to seborrhoeal warts, which they closely resemble. The lesions are grouped like tubero serpiginous syphilides, and they slowly progress like these, at times leaving an atrophic central scar. Their treatment is that for seborrhoeal warts as mentioned above.

Xeroderma Pigmentosum is an extremely rare familial disease characterized by the presence on the exposed skin of dilated vessels, freckles, atrophies, warts, keratomas resembling the senile variety, and ultimately ulcers and epitheliomata of both varieties. Often the cornea and conjunctiva become involved by these growths, and usually, with or without them, there is photophobia with lacrimation. The disease begins in infancy, progresses until the hands and face or any other exposed areas are involved, and death from carcinomatosis supervenes in adolescence or early adult life. The diagnosis is easy, the prognosis of course always bad, and the disease is extremely rare. Prophylactic treatment consists of minimizing exposure to light, and it does very little good. There is no adequate treatment for the fully developed disease. Actually the condition is premature senility of the skin.

Roentgen Carcinoma is a condition formerly more common than now, for in later years Roentgen operators have taken proper precautions. The general skin reaction produced by the X-rays is an artificial xeroderma (Chapter VII) pigmentosum restricted to the hands. Telangiecstasia, pigmentation, atrophy, ulceration, hyperkeratosis, verrucae and epitheliomata are all present as in xeroderma.

In both conditions epitheliomata invariably arise. Thus we have in xeroderma, Roentgen dermatitis and sailor's skin true precancerous dermatoses, or better dermatoses some element of which eventuate in malignancy. Xeroderma is congenital and precocious senility, Roentgen dermatitis is a regressive process produced by X-rays, sailor's skin is partly due to senile regressive changes and partly to the actinic solar rays.

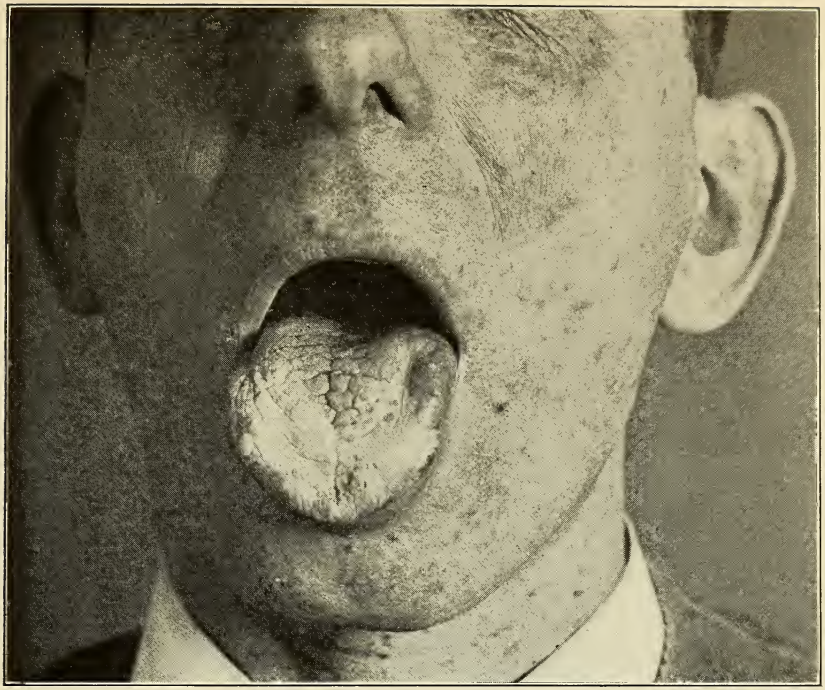

Fig. 74. LEUCOPLACIA BUCCALIS

Leucoplacia, particularly the syphilitic variety, frequently gives rise to epithelioma. Leucoplacia resembles lichen planus, but on study, no papules are seen, and the white is sheerer than in lichen.

CHAPTER XXX

BENIGN CONNECTIVE TISSUE NEOPLASMS

Although neoplasms of this group have no great clinical or anatomical similarity they share a common embryological ancestry. It is questionable whether at times these growths may not be nevi in a sense which will subsequently be discussed (Chapter XXXIII). Excepting fibromata and lipomata the conditions are rare, or, if not rare, of relatively little significance, and save for the sake of scientific completeness they would not need to be mentioned. Fibromata and lipomata are the common varieties.

FIBROMA

Synonyms. Fibroma Durum, Fibroma Molluscum, Molluscum Fibrosum, Neurofibroma, Recklinghausen's Disease.

Definition. A fibroma is a benign new growth formed of fibrous connective tissue cells or fibroblasts, the amount of the cells and fibrous connective tissue or collagen varying in proportion. At times the structure is myxomatous.

Symptoms. The growths may be single or multiple, present at birth or appearing later, and often accompanied by pigmentary macules. They are hard or soft, lenticular, sessile or pedunculated and they vary in size. Some may be huge and pendulous. They are flesh colored, buff, or brownish, and with certain exceptions to be stated below cause no subjective symptoms.

Course. A fibroma practically never goes away by itself. Sometimes the growths undergo malignant change giving rise to fibrosarcomata or, much more rarely, spindle celled sarcomata.

Varieties. Some of the varieties have been suggested among the synonyms. These are Recklinghausen's (Fig. 75) disease, fibroma molle, fibroma durum, myxoma, fibroma pendulum, and keloid, with the latter's subvarieties.

Recklinghausen's disease or multiple fibromata, or neuro-fibromatosis is not common. It begins in early youth, at puberty or rarely in childhood and gradually the body is studded with countless growths, some just visible, others even an inch in diameter.

The growths are soft, sessile or pedunculated, at times painful or tender and circular or oval. They are flesh colored, brownish, buff or red and occasionally traversed by dilated vessels. On pressure some are felt to disappear within a ring but pop out again when released. At times growths may be felt along nerve trunks. Interspersed among the above lesions are lentiginous or chloasmic macules, and some of the growths are bluish or topped in a manner roughly suggesting a vesicle, but containing no fluid. The disease is not unlike dermatolysis (Chapter XVII). A low mental and physical habitus has been noted, but this is neither constant nor diagnostic. The diagnosis is easy, the prognosis good as to life, but the disease is irremediable and no rational treatment exists. The cause is unknown, but in the broad sense, its origin being embryonal, it may be a nevus. Although not rare the condition is uncommon. Sometimes the fibromata arise upon nerve trunks, as indeed elsewhere in the body, but the term neurofibromatosis is thoroughly unjustifiable as the tumors lack nerve fibres.

Fibroma molle not giving the picture of Recklinghausen's disease is common. Usually one or two lesions or a dozen are seen. They vary in size as above and are similar in aspect. They are sessile or pedunculated, at times hanging by a thread of tissue. Their softness is striking. They may be removed by ablation, excision or fulguration. These are the lesions also known as fleshy warts, and their origin and significance are not known.

Among the lesions of Recklinghausen's disease and soft fibromata, some are found microscopically to be entirely or in part myxomatous. Clinically such lesions are not distinguishable from their fellows, and the myxomatous tissue is probably degenerative.

Fibroma pendulum is simply an enormous lesion, the size of which makes it pendulous.

Keloids are hard fibromata which are primary or secondary. The former arise spontaneously, the latter after trauma such as burns, scalds, the accidental or intentional application of caustics, whether acid or alkali, vaccination and the like. Primary keloids are commoner in negroes than in whites, and are elevated, hard, linear masses, red, white or buff in color, and often crossed by dilated veins. They are globular, linear, band shaped, reticular or arborescent and their size varies. They favor the sternum, shoulders, neck, face, back and extremities. The secondary forms, which are also called hypertrophic scars, correspond in general to the spontaneous ones, but their shape is determined by that of the original injury, and

their color is at first bluish, gradually becoming paler. Both types tend to recur after excision, and the secondary form often flattens of itself. The main difference between keloid and hypertrophic scars, as Crocker states, is that the former has no growth limit, while the latter never transcends the size and shape of the determining injury. The best treatment of both kinds is with massive doses of X-rays or radium. Excision, ablation, and burning remove the growth but it is likely to return. Thiosinamin has been used but it is painful and the results are not gratifying.

Hard Fibromata or fibromata dura are pea sized nodules resembling the globular or rounded keloids. They are found scattered upon the trunk or extremities. Structurally they differ from soft forms in their greater content of dense collagen.

Differential Diagnosis. Fibromata of all sorts are easy to recognize. In cases of doubt microscopic examination clears up the problem.

Etiology. Nothing is known of the etiology.

Treatment. This has been described in each condition.

Prognosis. The prognosis is good as to life. In Recklinghausen's disease little can be accomplished because of the number of the lesions. Simple fibromata are easy to cure. Keloids offer a bad prognosis because they are prone to recur.

Neuroma. This condition is closely allied to fibroma. It arises as single or numerous tubercles, usually in old men, and the lesions appear on the shoulders and arms, thighs and buttocks, as nodules or tubercles. They are hard, tender or painful. The tumors appear to arise from the sheaths of cutaneous nerves or larger nerves, from which they invade the skin.

Lipomata are fatty tumors arising singly in the breast, buttocks and neck, rarely on the face, scalp and genitalia. They are soft masses varying greatly in size. At times they are numerous and symmetrical and at times painful and associated with general adiposity (Dercum's Disease). They may undergo calcareous degeneration or liquefy, forming cysts, or they may become infected and give rise to abscesses. Extirpation is the only therapy. They do not recur.

Osteomata are too rare to mention in detail. They are recognized by their hardness, and the final diagnosis is microscopic.

Myoma is a rare skin affection. Muscle tissue is present at times in angioma, sarcoma, keloid, and at times in xanthoma as in the case of Chambard and Guellano. True myomata, however, are very

exceptional. A dartoic variety affecting the nipples, scrotum and labia, or independent of these structures, is known. The tumors are single or multiple, and although seldom larger than an almond sometimes attain the size of a fist. They are sessile or have a pedicle. The multiple type consists of flat papules whose size rarely exceed that of a pea. The lesions are red, buff or brown and translucent. They are disseminated and sharply circumscribed, circular or oval, and flat, and on palpation they are found to be in the cutis. At times they affect only the exterior aspects of the extremities. Often they pain severely and paroxysmally. They are composed of striated or smooth muscle. Only extirpation is of therapeutic avail. There is nothing known of the etiology of the growths.

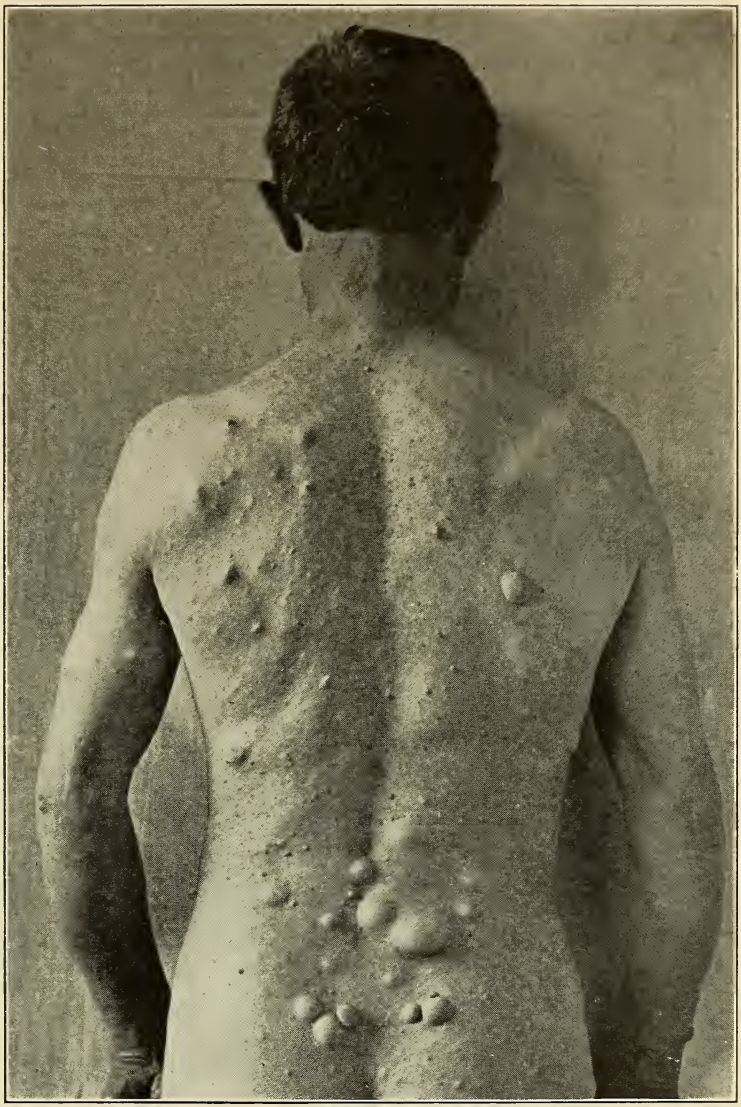

FIG. 75. RECKLINGHAUSEN'S DISEASE

This condition is multiple fibromatosis or perhaps neurofibromatosis. The lesions are minute papules or huge tumors, and are interspersed with hyperpigmented macules.

CHAPTER XXXI

MALIGNANT CONNECTIVE TISSUE NEOPLASMS

Definition. Sarcomata are among the rarest skin neoplasms. They are composed of mesodermal tissue and consist prevailingly of either round, spindle, fibrous connective tissue, lymphatic or giant cells, with or without melanin, and with more or less blood vessels. At times mixtures of the cells are present, and in general their structure is not indicated by their clinical appearance. They recur after extirpation, mestastasize to other parts of the skin and viscera, and some are highly malignant. The growths are primary or secondary. The former are those which arise in the integument,— the latter those which, arising elsewhere, invade or are conveyed to the skin, their source being internal organs or a primary growth in the skin itself. A peculiar primary form is the nevo-sarcoma which is primary as to its sarcomatous character, but secondary in that it originates in a preexisting lesion albeit originally of a totally different character. Primary growths spring either from the cutis or hypoderm and may be single or multiple.

Symptoms, Course and Varieties. Hypodermic sarcoma (Perrin) appears on the trunk, thighs and arms, usually in multiple growths. At first the lesions are invisible, but palpable. Later they become visible and vary in size from a pin head to a pigeon's egg. They are rounded, reddish or violaceous and slightly scaling. They are composed of lobulated islands of round cells. The disease is most malignant, death occurring from metastasis or inanition within a few months to two years.

There are all sorts of single sarcomata, whitish, reddish, red, brown, buff, appearing anywhere on the body and growing, recurring, even metastasizing to other parts of the skin or inner organs. They are small nodules at first, gradually reaching the size of a pigeon's egg or becoming even larger. They tend at times to necrose, rarely to involute spantaneously. Their structure cannot be surmised from their gross appearance, for any anatomical type may be represented. Of all of these the fibro-sarcomata are the least malignant. They may persist and multiply for years doing

relatively little harm, but ultimately they, too, cause death through inanition or by metastasis.

Melanotic sarcomata (Fig. 76) arise from nevi in a manner analogous to melanoma, anatomically resembling epithelioma. They are extremely malignant, having a rapid local growth and throwing out metastases. Structurally they resemble round or pigment cell sarcomata, with great deposits of melanin. They metastasize rapidly and widely both in the skin and internal organs.

Secondary sarcomata arise from internal growths and invade the entire skin as small or large nodules of lenticular shape, and possessing a great play of color. They are hard, rounded and often traversed by telangiectasia and possess the structure of the mother neoplasm. They kill quickly. Chloromata and hypernephromata are of this type.

Differential Diagnosis. Single lesions resemble sarcoids, granuloma fungoides d'emblée, leucemia cutis, epithelioma, fibroma and individual lesions of the various infectious granulomata. The diagnosis can be surmised but not positively established without the microscope.

Multiple sarcomata, whether primary or secondary, resemble granuloma fungoides as well as fibromatous and the infectious granulomata. The clinical diagnosis is easy as a rule except with regard to granuloma fungoides and leucemia, and, with these exceptions, the microscopic diagnosis is always absolute. The clinical differences between sarcoma and mycosis lie in the prefungoid dermatoses, and the rapid evolution and involution of the lesions. The microscopic examination is usually conclusive excepting in so far as round celled and lymphosarcoma are concerned.

It must be remembered that the cell types in sarcoma are, in general, more uniform than in granuloma fungoides, that in the latter there are more metastases, eosinophiles and giant cells. But there is also a giant cell sarcoma to cause confusion, and many lesions of granuloma fungoides have uniform round cells. Lymphosarcoma may cause similar confusion. Thus at times the most expert clinician or microscopist may, without self-humiliation, feel his talents challenged beyond the point of conquest. It is for this reason too that many writers are justified in allying mycosis and sarcomatosis. Whatever is true of mycosis is also true of leucemia versus sarcoma, but the blood picture and other symptoms of leucemia should aid in diagnostic accuracy.

Etiology. To attempt to discuss the cause of sarcoma is absurd.

Treatment. Single lesions may be extirpated or X-Rayed with success. The use of arsenic and Coley's fluid offers nothing. Multiple lesions are at times improved under the X-Rays, but no case of a cure is on record.

KAPOSI'S SARCOMA (FIG. 77)

A special form of sarcoma, Kaposi's multiple hemorrhagic type, represents a clean cut clinical picture and thus merits a special place in the chapter on sarcoma. It is called benign, but the adjective is admissible only in a relative and restricted sense.

Synonyms. Sarcoma idiopathicum multiplex hemorrhagicum.

Definition. This disease is a multiple sarcoma, benign in that its fatal termination is often long deferred, and characterized by multiple growths, widely disseminated, and arising upon skin that is pigmented and hemorrhagic.

Symptoms. The disease begins in the third or fourth decade with the appearance of small nodules or tumors, usually on the feet or hands. These tumors rest in or under the skin and are flat or convex, painless, and rather hard or elastic. Their color is purple, red, violet or a deep blue, at times dulled by a grayish superimposed filmy color which imparts to the underlying tint a peculiar soft quality, such as the peach possesses because of the latter's delicate fur. The skin is often swollen and red, violet or brown, according to the character of the hemoglobin derivatives resulting from hemorrhage. A few growths may be present, or a great many. At first the lower extremities, later the upper, and at times the trunk are involved. Edema is provoked to a high degree through lymphatic stasis due to occlusion of the lymph vessels by metastases. The palms and soles are included. Often, however, only a few blue or purple nodules are present.

Course. As a rule years are required before the disease attains its fastigium. Then the entire body or only the extremities may be involved with growths as described, or with veritable plaques. Some of the growths scale, others involute spontaneously. Still others, but only rarely, ulcerate. Whatever the fate of the individual lesions the disease as a whole is progressive, and if untreated terminates fatally, rather from inanition than internal metastasis. The patient may die of an intercurrent disease, and the end comes within from two to ten years.

Varieties. There are none except in the degree and rapidity with which the disease runs its course.

Diagnosis. Kaposi's sarcoma resembles nothing else. Granuloma fungoides is roughly simulated, but the absence of prefungoid lesions and the absence of a tendency to spontaneous involution rules out mycosis. Anatomically Kaposi's sarcoma is a spindle cell angio-sarcoma with hemorrhage, which differentiates it from anything else.

Etiology. Nothing is known of the causation, but it is more common in Russian Jews than in any other race.

Treatment. Arsenic causes relief in some cases. X-rays are curative in the early stages, and inhibit progress in the later stages, but on the whole no satisfactory therapy is known.

Prognosis. The disease is usually fatal.

A concluding word may not be amiss at this point, on the general problem of tumor diagnosis. Certain epitheliomata, sarcomata, myomata, even fibromata resemble one another clinically. To attempt a diagnosis on clinical data alone is often futile. Hypodermic sarcoma, granuloma fungoides and Kaposi's sarcoma have a few points in common. So have these with Recklinghausen's disease and myomata. So have some of them, indeed, with lepra, syphilis, sarcoid of Darier-Roussey and tuberculous gummata. It is unlikely that one would often be confronted with the clinical problem of such a differentiation, and yet this might occur. The final arbiter is the microscope, for each of the neoplasms has a characteristic anatomy, and the well trained dematologist must know enough of pathological histology to be able at least to distinguish a neoplasm from an inflammatory granuloma. To deride the microscope in its proper sphere is inspired by laziness and ignorance rather than by a keen longing for clinical acumen, for it is easier to make sport of an unacquired attainment than to master it. Sound clinical ability is the most desirable thing in medicine, but there is a broad gulf between this and genius at dialectics (a worthless attribute), and when a diagnosis can be reached only by microscopic study the latter ceases to be an avocation and becomes a duty. Accurate tumor diagnosis is possible only by means of histological investigation, and is here just as useful, as in the diagnosis of pemphigus it is useless. By this means it is possible to determine in five minutes what could never otherwise be determined in endless debate, and upon the correct diagnosis of tumors depends correct therapy and perhaps the only chance for the patient's health — or life.

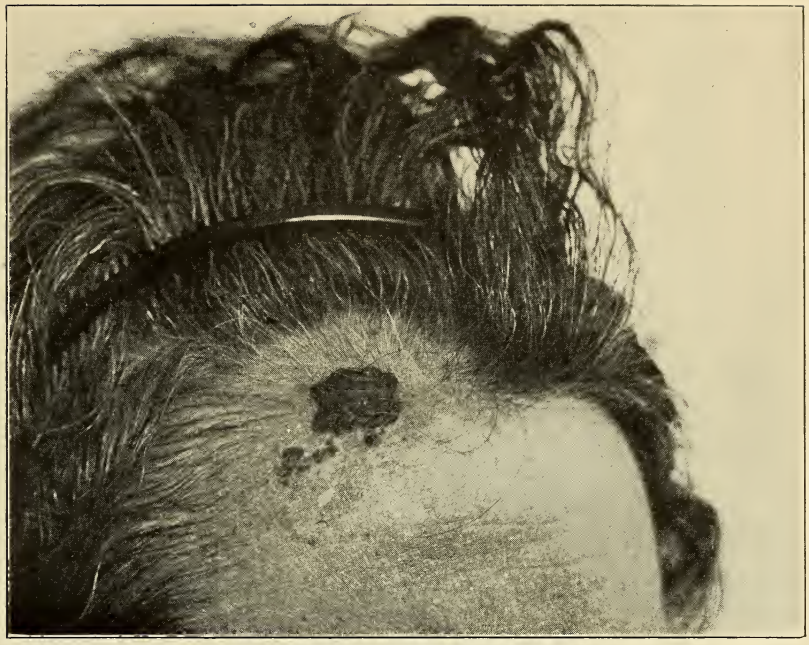

FIG. 76. MELANOMA

This neoplasm usually starts in a pigmented mole and is brown, black or slate-colored. It is highly malignant and breaks down and metestasizes.

FIG. 77. KAPOSI'S SARCOMA

Multiple benign hemorrhagic idiopathic sarcoma is a form of angiofibro-sarcoma in which brown or purple tumors of varying size, and in varying number, rise upon a skin discolored by purpuric macules or sheets. As a rule only the lower extremities are involved.

GROUP IX. NEVI

A nevus etymologically is a spot or blemish. If its use were not restricted in dermatology, about everything could be included. The word has acquired limitations, with the passage of time, and now is employed as a synonym of birth mark. By no means all; not more indeed, than a substantial minority of nevi are congenital. The greater part of them develop within the first year of life. The remainder appear later. A nevus is a skin lesion due to an embryonal tissue disturbance the objective manifestation of which may be present at birth or appear at any time thereafter.

Several types of this disturbance are generally accepted as nevi, others are so regarded by some writers but not by all. A great many attempt to restrict the concept to conditions which microscopically are found to include in their composition the so-called nevus cell. This will presently be further discussed. If this were the accepted point of view a nevus would have to be defined as a skin anomaly, either congenital or appearing after birth, due to an embryonal disturbance, and containing nevus cells. If the broader view were accepted, a nevus would have to be defined as any congenital anomaly in the skin, present at birth or becoming apparent subsequently. Thus a nevus would depend upon springing from embryonally disturbed cells of any origin whatever, and not solely upon the presence of nevus cells. In other words a lesion developing long after birth would still be a nevus because it sprang from *anlagen* deposited in intrauterine existence.

This point of view would appear the more logical, but since it is not yet universal it is better to divide the conditions in question into two groups, nevi and diseases which are probably nevi. It is futile to discuss the origin of these *anlagen.* No one knows whether they represent variations or atavisms; whether they are the result of intrauterine trauma or not; or whether they are merely misplaced cell inclusions. It is of no practical importance to ponder this question, nor whether nevus cells are invariably epiblastic or mesoblastic.

CHAPTER XXXII

NEVI

Nevi may be pigmentary, hairy, vascular, lipomatous or hypertrophic; they may be numerous and roughly grouped, or linear in distribution. They may be of one or more types, and indeed, a single lesion may be composed of several simple forms. The commonest combination is the hairy and pigmented nevus.

NEVUS PIGMENTOSUS

Synonyms. Pigmented Mole, Nevus Spilus; French, Tâche Pigmentaire, Naevus Pigmentaire; German, Fleckenmaal.

Definition. Pigmentary nevi are those which have a greater or smaller content of melanotic cells otherwise known as melanoblasts or chromatophores. They are therefore either light or dark brown, blue-black or black, the shade depending upon the amount of pigment present.

Symptoms. Nevi of this sort favor the face, neck, trunk, thighs, buttocks and genitalia, but they may appear anywhere else. They vary in size from minute dots to several inches in diameter, and may run the entire scale of colors indicated above. They are circular, oval or irregular in outline, flush with, slightly or much higher than the skin surface, smooth, corrugated, verrucous, papillamatous or cauliflower like, and often hairy (nevus pilosus). Only one lesion or many may be present, and they may be grouped when multiple, in irregular patches, lines, bands or in a zosterform arrangement. It is unusual to find them at birth, but they blossom out in infancy, and at puberty some, previously smooth (nevus spilus), become raised and irregular. They provoke no subjective symptoms.

Course. The course has been indicated in the foregoing paragraph. These nevi never disappear and when irritated may become malignant, the malignancy more often taking the form of epithelioma than sarcoma. Either type, however, is serious. Irritation appears to stimulate them to malignancy, of which the first signs are growth, necrosis and a fetid discharge.

Varieties. Varieties may be determined upon a basis of num-

ber, shape, size and distribution. Such distinctions are artificial. Hairy nevi may be regarded as a subvariety of pigmentary ones since one practically never sees non-pigmented hairy forms. All of the features mentioned are present plus hairs, which may be downy or coarse, short or long, sparse or dense, and usually dark. They occur anywhere. Such nevi are most disfiguring. They attain, at times, huge dimensions covering the cheek or chest, back or buttocks, and may be so hairy as to suggest fur. Huge nevi are called giant nevi. When linear in grouping the term nevus linearis, or when on one side, nevus unius lateris, is applied.

Differential Diagnosis. The diagnosis is based upon the history and appearance of the lesions. Nothing else really resembles nevi, but at times pigmentary lesions of other diseases may cause confusion. In such cases the microscope may be enlisted. The structure of the growth is characteristic. Islands of nevus, intermixed with pigmentary cells, are seen.

Etiology. This has been discussed in the opening paragraphs.

Treatment. Pigmentary nevi should not be removed with caustics. They should be excised in order to prevent stimulation to malignancy. When excision is unfeasible carbon dioxide snow is an excellent remedy. Hairy nevi may be similarly treated, or if mere electrolytic epilation is practiced this will often in itself serve to efface the pigmented elements.

Prognosis. When undisturbed the lesions are harmless. When tampered with, irritated or unskilfully treated malignancy may develop.

VASCULAR NEVI

Synonyms. Nevus Vasculosus, Nevus Vascularis, Angioma; French, Nevus Vasculaire; German, Gefässmal.

Definition. A vascular nevus is one due to an anomaly of cutaneous vessel formation resulting in their hyperplasia or absence.

Symptoms. Nevi of vascular origin may be flat or raised, single or multiple, small or large, and their color and appearance depend greatly upon their structure. They may be present at birth or appear shortly thereafter, or small congenital lesions may grow both in diameter and depth within the first few weeks of existence. The growth may be progressive to a certain point and then cease. Rarely spontaneous involution is observed, a delicate superficial scar remaining. The shape of the lesions, their shade and distribution offer a wide range of possibilities. Any part of the body is their site, but the head is the favorite location, particularly near the eyes,

root of the nose, occipito-nuchal area, cheeks, and neck. Crying, laughing or straining tend to deepen their color.

Course. As already stated, the lesions appear at or shortly after birth and are capable of limited growth, a very few involuting completely.

Varieties. Varieties are determined partly by appearance and partly by structure. The following classification is suggested:

A. Nevi due to vascular hyperplasia or hypertrophy.
  I. Flat nevi,
    1. Nevus Simplex,
    2. Nevus Flammeus,
    3. Flat Angioma.
  II. Raised Nevi,
    1. Angioma,
    2. Angioma Cavernosum,
    3. Hemangio-lymphangioma,
    4. Angio-lipoma,
    5. Angio Elephantiasis (Virchow).
B. Ischemic Nevi.
  I. Nevus Anemicus (Vörner).

Nevus Simplex is seen at the root of the nose, near the occiput or finally anywhere else. It is a pale pink to marked red lesion from a half inch to two inches in diameter. The borders are rather indistinct and formed of feathery telangiectasia. About sixty per-cent. of all infants present them, and rather more girl babies than boys have them. They tend to fade or disappear in childhood, but a great many persist. Their color deepens with effort or crying.

Nevus Flammeus is an exaggeration of the above. It favors the face, is deep red to purple and occurs in patches from the size of a quarter to half the face and neck. The border is short but beyond it smaller lesions are seem. Some areas are deep, others superficial. Occasionally one sees portions which are raised and have the features of angio-lymphangioma or angio elephantiasis (vide infra). Nevus flammeus, also called port wine mark, nevus or stain, and in German, Feuermal, persists through life.

Flat Angioma occurs as a deep crimson, circumscribed, oval or roughly circular spot, or there may be several. The surface is lustrous and claret colored. Such nevi bleed easily.

Raised Angioma has the features of the above, but the surface is more or less raised and irregular, or even raspberry like or lobulated.

The lesion bleeds easily, sometimes pulsates, and when near the fontanelles may communicate with the cranial sinuses. In middle aged persons small cherry red lesions of this type, varying in size from a pin head to a cherry stone, are studded over the trunk.

Angioma Cavernosum is clinically like the preceding form. It may reach the size of a walnut or bantam's egg and is often purple or blue.

Hemangio-Lymphangioma has the ordinary features of an angioma or angioma cavernosum, but feels denser and harder, and the blood cannot be easily expressed, for the lymphatics too are involved in the hyperplastic process.

Angiolipoma may have the character of any of the foregoing, but the vessels are imbedded in lipomatous tissue. Angio-elephantiasis is closely related to hemangio-lymphangioma, with the added factor of fibromatous masses revealed, upon deep palpation, as hard nodules within the growth. This form is chiefly observed in nevi extending from the skin through to the mucosa of the cheeks or lips or ear lobes, imparting a swollen edematous look to the tissues.

The color of all of these types is red or blue, or midway between these extremes, according to the proportion in which veins, arteries and lymphatics enter into the composition of the growth. At times vascular and pigmented nevi coexist separately or in the same lesion. Anatomically the flat nevi are composed of hyperplastic capillaries; the angiomata of hyperplastic and hypertrophic capillaries in the corium; cavernous angiomata consist of hyperplastic vessels with a structure resembling that of the corpus cavernosum, and situated in the corium and hypoderm. Hemangio-lymphangiomata are composed of hyperplastic and hypertrophic blood and lymph vessels; angio-lipomata, of such blood vessels imbedded in fat, and angio-elephantiasis of blood and lymph vessels surrounded by fibromatous tissue.

Nevus Anemicus is a curious and rare condition seen chiefly on the chest below the clavicles. The lesion consists of a group of pale macules. When rubbed the normal skin turns pink or red, the nevus remaining sharply blanched. This phenomenon is due either to an absence of superficial capillaries or a defect in innervation, the vaso dilators being absent or the vaso constrictors over active, so that the vessels in the involved areas fail to dilate as do those of the normal surrounding skin.

Differential Diagnosis. There is no difficulty in recognizing vascular nevi. Nevus anemicus must be differentiated from leuco-

dermas which get pink when rubbed, and from lichen atrophicus, white spot disease and morphoea, which are atrophic. Other lesions of morphoea, with violet rings, or of lichen atrophicus with lichenous, infiltrated margins, are too unlike the pale nevus to cause confusion.

Etiology. To attempt to elucidate the causation of nevi is futile. They are embryonal anomalies and are present at birth or are latent for varying periods.

Treatment. Small vascular nevi are destroyed by electrolysis, larger ones with carbon dioxide snow, trichloracetic acid or the high frequency spark. Nevus flammeus is best treated with carbon dioxide snow, radium or the Kromayer light. Angiomata may be excised or treated as outlined in the preceding section. Excision is impracticable in very large lesions. Radium or snow are best in lymphangioma. There is no cure for nevus anemicus.

Prognosis. The cosmetic possibilities regarding very large nevi are poor, as with any treatment a scar remains corresponding in size with that of the original defect, but the scar is white and thus constitutes a relative improvement. Nevus flammeus gives fairly good results when treated with radium or the Kromayer light.

CHAPTER XXXIII

DISEASES WHICH ARE PROBABLY NEVI

There is a number of conditions clinically and histologically not closely related, but having the bond of being anomalies. Since all growth disturbances are embryonal such diseases may be called nevi, unless we desire to restrict this term to lesions containing what we call nevus cells. If this is the case the term must be confined to narrow usage; if not we must regard all anomalies as nevi. Perhaps it would be wiser to make the generic disignation anomalies, of which nevi would be a sub class. It makes very little difference whether we adopt this point of view, or whether we make nevus synonymous with anomaly, so that we standardize our terminology. I prefer considering the terms equivalent and the following classification would be suggested.

I. Nevi in the broad sense
   1. Lymphangioma circumscriptum multiplex
   2. Trichoepithelioma
   3. Syringoma
   4. Adenoma Sebaceum
   5. Granulosis Rubra Nasi
   6. Ichthyosis
   7. Monilethrix

II. Probable members of the nevus group
   1. Keratosis suprafollicularis
   2. Erythroderma congenitale icthyosiforme

III. Possible members of the nevus group
   1. Xeroderma pigmentosum
   2. Recklinghausen's disease
   3. All neoplasms benign and malignant

The first group consists of seven members which are obviously anomalies of growth. The two members of the second group may be inflammatory in origin, but it is more likely they too are anomalies. The third group consists of neoplasms, and it is questionable whether Cohnheim's views may or may not be applied to all new

growths. If they may be then all are nevi. On the other hand, the neoplastic tendency, equally likely, has nothing to do with embryonal disturbances, and in this event there would be no connection with nevi. The relation of the third group to nevi must thus still remain an open question.

Lymphangioma Tuberosum Multiplex is rare. The disease consists of numerous, small, yellow or orange vesicles containing lymph. Each vesicle is as large as a pinhead or lentil and it is characteristic for a number of such lesions to group themselves closely, while the groups in turn are numerous. The favorite sites are the arms, shoulders, scapulae, thighs, axillae and buccal mucosa. The disease begins in childhood. Often nevi are associated. It is reasonable to regard the condition as a lymph vascular nevus.

Epithelioma Adenoides Cysticum, or trichoepithelioma papulosum multiplex (Jarisch) (other synonyms are misleading), is an anomaly of the hair follicles, as is shown histologically. The disease begins at or about puberty and is mainly facial. Symmetrically distributed tiny discrete papules arise on the glabella and adjacent areas, the temples, cheeks, brow, chin and rarely, the shoulder girdle, neck, scalp and arms. They are either pearly, yellow or pink, and in size never exceed that of a lentil, the average being much smaller. Only a few, or a great many, may be present, and they feel imbedded within the skin above which they rise a very little. A telangiectatic tendency is noted in some lesions, and rarely ulceration occurs. After first appearing they increase in number and size for a time and then become stationary. Their appearance at puberty makes it difficult at first glance to accept the belief that nevi are embryonal disturbances. Being hair follicle anomalies their expression would obviously not be gained until puberty, when the hair develops. The condition most closely resembles milium (Chapter XXXV) and trichosyringocystoma. Milia can be expressed but trichoepithelioma cannot. The differentiation from syringoma will be discussed below. Aside from electrolysis or the high frequency spark there is no adequate therapy. Thus the prognosis from the cosmetic side is poor.

Syringoma, of which the synonyms are legion, and no name for which is exact, is best designated by the characterization at the head of this sentence. Some of the other names are hydradenoma eruptiva, syringocystadenoma, benign cystic epithelioma, lymphangioma, tuberosum multiplex, syringocystoma, hemangioendothelioma tuberosum multiplex. The disease is rare and often confused

with trichoepithelioma which it closely resembles when the face is involved. Its favorite sites are the trunk from just above the shoulder girdle to pubis, and occasionally the extremities. The lesions are the size of trichoepitheliomata, smooth, discrete or grouped, soft, yellow, red or brown, and often waxy. Microscopically cysts, probably due to a sweat duct anomaly, are seen. These lesions, too, appear at about puberty and run a course similar to that of the foregoing condition. They are due to embryonally misplaced sweat duct cells. There is no adequate therapy save electrolysis or the high frequency spark.

Adenoma Sebaceum (Pringle) is rare. It begins in early childhood as bright red, yellowish or brownish pinhead to lentil sized papules on the cheeks, forehead and nasolabial folds. Low mentality has been noted at times. As a rule no alterations in the sebaceous glands are noted and Reitmann suggested the name of nevus symmetricus facies. Histologically the lesions resemble minute fibromata. The treatment is as above. The prognosis is poor. At times the condition is associated with Recklinghausen's disease.

Granulosis Rubra Nasi (Jadassohn) is extremely rare. The tip of the nose is red and covered by tiny, dark red papules. Local perspiration is present. The entire picture is a sweating granular red tip of the nose. Children from infancy to puberty are affected. Although there are histological evidences of inflammation the disease is closely allied to hydrocystoma, and it is the probable result of a congenital local anomaly of the vasomotor system, causing sweating and cystic changes in the coils. This is analogous to the vasomotor theory in nevus anemicus (Chapter XXXII). There is no satisfactory treatment.

Ichthyosis has been described (Chapter XV). If this and its allied conditions are nevi they belong in the hyperkeratotic or verrucous group. Keratosis follicularis (Chapter XV) is closely related to icthyosis, as is erythroderma congenitale icthyosiforme. Xeroderma pigmentosum (Chapter XXIX) and Recklinghausen's disease (Chapter XXX) are possibly nevi, but this would depend upon further evidence of their being embryonal disturbances.

If it is admitted that the conditions enumerated in this chapter are nevi, these with those included in the preceding one might be classified as follows:

I. Vascular anomalies
  1. Blood vessels
    a. Hypertrophy or hyperplasia of vessels (see table Chapter XXXII)
    b. Absence of vessels. See nevus anemicus.
  2. Lymph vessels
    a. Lymphangioma circumscriptum
    b. Hemangiolymphangioma

II. Pigmentary anomalies
  1. Nevus pigmentosus
  2. Congenital absence of pigment

III. Hair organ anomalies
  1. Nevus spilus
  2. Trichoepithelioma
  3. Monilethrix

IV. Sweat organ anomalies
  1. Syringoma

V. Sebaceous organ anomalies
  1. Some forms of adenoma sebaceum

VI. Anomalies of keratinization
  1. Nevus hypertrophicus or verrucosus
  2. Ichthyosis (all forms)
  3. Erythroderma congenitale ichthyosiforme
  4. Keratosis suprafollicularis

VII. Anomalies of the rete and other epithelial structures
  1. Xeroderma pigmentosum
  2. Epithelioma
  3. Verruca

  } In the sense of Cohnheim

VIII. Anomalies of the connective tissue
  1. Angio elephantiasis
  2. Adenoma sebaceum (Pringle)
  3. Fibroma
  4. Sarcoma

  } See comment, group VII

IX. Anomalies of fatty tissue
  1. Angio lipoma (probably vascular)
  2. Lipoma

X. Anomalies of innervation
  1. Nevus anemicus
  2. Granulosis rubra nasi

SECTION E. DISEASES OF THE ORGANS OF THE SKIN, AND OF THE MUCOUS MEMBRANES

GROUP X. THE SKIN AND ADNEXA

CHAPTER XXXIV

DISEASES OF THE HAIR AND FOLLICLES

Diseases of the hair may be grouped as follicular hyperkeratoses, dystrophies, infections and nervous diseases. The result of these conditions may be an excessive growth of hair or a loss of hair. The shaft or follicle or both may be affected. Different hairy areas of the body are variously influenced by the conditions.

A. Hyperkeratoses and inflammation of the follicle.
   - I. Seborrhoea (Chapter XI)
   - II. Lupus Erythematosus (Chapter XXV)
   - III. Folliculitis decalvans
   - IV. Trichostasis spinulosa (rare and unimportant)

B. Dystrophies
   - I. Hypertrophies, Hirsuties
   - II. Atrophies
     1. Trichonodosis
     2. Fragilitas crinium
     3. Trichorhexis nodosa
     4. Monilethrix
   - III. Pigmentary dystrophies
     1. Canities
     2. Ringed hairs

C. Infections
   - I. Bacterial
     1. Staphylogenic folliculitis (Chapter XXIV)
     2. Folliculitis decalvans
     3. Impetigo of Bockhardt (Chapter XXIV)
   - II. Fungi (Chapter XXVI)
     1. Affecting the hair
        - a. Trichomycosis
        - b. Leptothrix
        - c. Piedra

2. Affecting the hair follicle (Chapter XXII)
    1. Microsporosis
    2. Tinea
    3. Favus

III. Pediculosis (Chapter XX)

D. Neuroses

I. Organic
    1. Alopecia areata
    2. Alopecia generalisata

II. Functional
    Trichotilomania

Baldness may be primary or secondary.

A. Primary

I. Dystrophic
    1. Congenital
    2. Premature
    3. Senile

II. Nervous
    1. Alopecia areata (Chapter XXXIV)
    2. Alopecia generalisata (Chapter XXXIV)

B. Secondary

I. Traumatic
    1. Injuries

II. Inflammation
    1. Seborrhoea (Chapter XXV)
    2. Lupus erythematous, ulerythema ophryogenes (Chapter XXV)
    3. Folliculitis decalvans, folliculitis staphylogenes, Impetigo of Bockhardt (Chapter XXXIV)
    4. The various fungus diseases (Chapter XXVI)

Regionally hair diseases may differ.

1. Scalp. All the diseases above enumerated and pediculosis capitis.

II. Beard. Mostly staphylogenic diseases, alopecia areata and lupus erythematosus, and pediculosis capitis.

III. Eyebrows. Alopecia areata and ulerythema ophryogenes, lupus erythematosus, and pediculosis capitis.

IV. Eyelashes. Folliculitis staphylogenes, pediculosis pubis.

V. Body. Mostly pediculosis pubis and alopecia generalisata.

The foregoing tables simply indicate the commonest disturbances in the areas under discussion, and are not exhaustive. No great emphasis will be laid on any but the more usual diseases. Conditions already described are so indicated in the first table by the suffixed chapter numbers. Cross reference is suggested to the student. The following descriptions will be limited to diseases not yet dealt with.

FOLLICULITIS DECALVANS (QUINQUAUD) AND ALOPECIA CICATRISATA

Synonyms. Pseudopélade, Alopecia Circumscripta, Acne Decalvante.

Definition. These two, possibly identical, diseases present an atrophying folliculitis of the scalp. In Quinquaud's disease a few minute pustules are at the margin of the plaques, in pseudopelade there is absolutely no suppuration. The disease is, on the whole, rare and unimportant, but often resembles other commoner conditions and must therefore be described.

Symptoms. Baldness arises in round, angulated, or slightly irregular patches on the scalp. These patches vary in diameter from one-half to two inches and may be single or multiple. Their margins are sharp, ending abruptly in a line of normal hair growth. At first the bald area is pink, often smooth, sometimes containing follicles with hyperkeratotic plugs, but totally denuded of all hair. As time goes on a wrinkled atrophic skin remains. In folliculitis decalvans, near the margin of the patches, where the hairs are seen, minute pustules are situated, each pierced by a hair. The two processes lead to identical end results.

Course. The disease runs the course outlined above and terminates in atrophy with permanent baldness.

Varieties. Two varieties are known, viz., those described above. Their relationship is not universally admitted.

Differential Diagnosis. Pseudopélade resembles favus, alopecia areata lupus erythematous and the alopecia of secondary syphilis. Favus is excluded by the absence of spores. Alopecia areata is not inflammatory, stumps of hairs are present, and recovery usually takes place. In syphilis there are evidences of other secondary lesions, and the Wassermann reaction is positive. Lupus erythematosus cannot be excluded and, indeed, many writers consider the processes identical, but absence of hyperkeratosis, marked plugs and

absent signs of lupus erythematosus elsewhere exclude the latter. Folliculitis decalvans is differentiated from all of the preceding, and along the lines mentioned, but impetigo of Bockhardt, staphylogenic folliculitis and pustular ringworm must also be excluded. The first two resemble folliculitis decalvans in the character of the pustules, but the latter are more numerous and larger. Nevertheless it is to be remembered that some writers consider folliculitis decalvans identical with staphylococcus folliculitis and impetigo of Bockhardt, so that it is impossible to state precisely where this disease belongs. In ulerythema sycosiforme, which is merely a subvariant of staphylogenic folliculitis or sycosis of the beard, an atrophy remains very similar to that of folliculitis decalvans, so that after all the latter may very well be closely akin to Bockhardt's impetigo which is conceded to be only staphylogenic folliculitis of the scalp.

Etiology. Pseudopélade is either related to lupus erythematous or staphylogenic folliculitis. In the former event its etiology would be that of the related disease (Chapter XXV). In the latter event it would be the staphylococcus (Chapter XXIV). The same is true of folliculitis decalvans, the precipitating factor of which is the staphylococcus.

Treatment. This is parallel to that of lupus erythematosus and staphylogenic folliculitis. Since, however, the end result of the malady is atrophy and alopecia, cosmetic cures are impossible.

Prognosis. As a corollary to the preceding, the prognosis is poor.

HIRSUTIES

Hypertrophy of the hair, hypertrichosis, superfluous hair, etc., as its name implies is a condition in which an excess growth of hair is present where it should not be, or one in which the hair grows with unusual vigor. It may be general or partial and present at birth or appear later. In some instances, such as dog-faced men, or in women with hirsutes the condition, always pathological, has practical significance. In suitable cases the only treatment is removal by electrolysis. Women are often depressed or even become melancholy from the defect. Certain cases may be ascribed to pituitary deficiency and suitable gland therapy is said to cure the condition. The use of depilatories is to be condemned. A razor is better. The common superstition that soap or grease augment or cause hirsutes, is one of which women should be disabused.

Fragilitis Crinium indicates increased breakability of the hair. This may occur by splitting of the ends, fractures in the shaft, or either, in or above the follicle. Hot air drying, lack of grease, etc., are supposed to cause the condition which can in part be remedied by the use of oils and creams.

Trichorhexis Nodosa is a condition seen chiefly in the beard. The disease consists of ravelling of the hair, at node like points, causing moniliform projections found to consist of the disintegrated strands. The hair is seen to be studded at regular intervals with swellings at the sites of which the shaft easily breaks. The cause is unknown and there is no adequate treatment.

Trichonodosis. A similar appearance exists in trichonodosis, but the nodes are due to the spontaneous knotting of the frayed hair.

Monilethrix is a family disease seen mainly in women. It occurs chiefly over the occiput, but may be found elsewhere. The hair shaft is alternately constricted and swollen so that it resembles a chain of minute beads. MacKee ascribes the disease to a congenital anomaly of the follicle.

None of these conditions is curable or of great practical importance. To all of them is applied the generic term of atrophia pilorum propria.

Pigmentary disturbances of the hair take the form of patches of hair of different colors, ringed hair, or canites. In ringed hairs alternate bands of white and pigmentation are seen. Canites is whiteness of the hair. It normally appears in the prime of life or at the onset of old age. It may appear prematurely. It may be partial or complete. Its only cure lies in the use of dyes.

alopecia areata (fig. 78)

Synonyms. Area Celsi, Alopecia Areolaris, Pélade.

Definition. Alopecia areata is characterized by the sudden loss of hair in slightly inflamed disc like surfaces, which may be single or multiple. The disease tends to recur.

Symptoms. The disease begins suddenly with the appearance of bald spots. These are usually circular, oval, or irregular, and single or multiple, varying in size from one-half an inch to two inches in diameter. The denuded surface is lax, white and shows patulous hair follicles. Here and there a hair stump one-fourth of an inch or so long, and shaped like an exclamation point, is seen. The periphery of the patch abuts sharply against the normal hair. No subjective symptoms are noted. The scalp, beard and, at times, the body are affected.

Course. After a few weeks or months the denuded area is covered with fine hair which in a year or two is normal. At first the hair is often white, resuming its proper tinge gradually if ever. At

times the hair never returns, or at times only partially. Recurrences are common.

Varieties. In youth asymmetrical patches are usually observed, in middle age symmetrical ones. At times baldness appears in a band from the occiput to above the ears. This band is an inch broad, and this form of the disease is restricted to children. Otherwise it closely resembles alopecia areata. At times the eyebrows, moustache or lashes are the site of pélade. A generalized form of alopecia areata (the name is a contradiction of terms) is seen. Alopecia generalisata causes total loss of hair.

Differential Diagnosis. Alopecia areata must be differentiated from ringworm, favus, pseudopélade, lupus erythematosus, folliculitis decalvans and all other types of baldness.

A patch of alopecia areata is sharply circumscribed, the skin is lax, dead white and contains a few exclamation point hairs. At the periphery the apparently unaffected hair is easily withdrawn. No atrophy exists. In pseudopélade, lupus erythematosus and folliculitis decalvans, atrophy exists, and the other evidences of alopecia areata are lacking. In favus there is atrophy, and in this disease, as well as in ringworm, there are scales, fragile hairs and spores which are microscopically demonstrable. In all other forms of baldness the signs already mentioned of alopecia areata are lacking.

Etiology. Alopecia areata is commonly considered a nervous disturbance. So far as can be judged by circumstantial evidence this appears to be likely. All efforts to prove it the result of a local or general infection have failed. Undoubtedly the effects of shock, emotional strain, and the like, are responsible in some instances. In the majority, however, no such cause is ascertainable, nor is there any ground to assume ill health or a neuropathic habitus.

Treatment. The affected surface should be painted every five to seven days with pure phenol which is immediately neutralized with absolute alcohol, or the patient may be given equal parts methyl salicylate in chloroform to apply daily. Capsicum and cantharides lotions are also valuable. So is the Kromayer light. In the author's experience the first two methods have afforded the best results. Favorable cases are self limited, but with suitable treatment the course can be shortened. In unfavorable cases no known therapy is of avail. When the new hair remains white, dyes may be used.

Prognosis. Asymmetrical forms in the young have a favorable

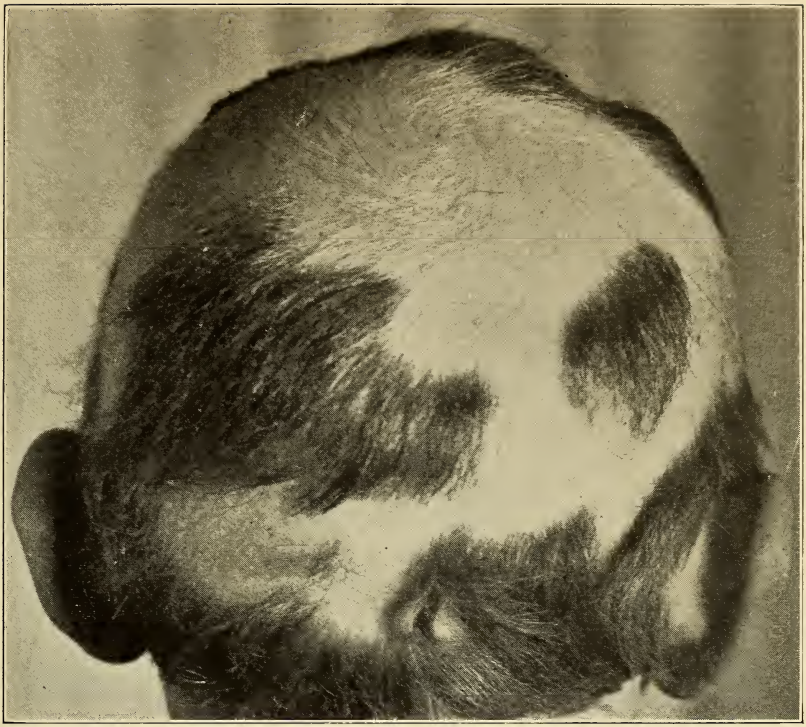

FIG. 78. ALOPECIA AREATA

This condition, also known as pélade, occurs as one or more circular, bald patches which by coalescence. give the picture here shown. The hairs usually return and may be white

prognosis. Symmetrical cases in the middle aged and old have not. In many instances recurrences take place.

A word as to baldness in general. The forms of baldness have been tabulated early in this chapter and the terms are self-explanatory. Cross reference to the respective underlying diseases will disclose both causes and therapy. Traumatic forms are due to scars following injury or disease, as furuncles, cuts, burns and the like.

CHAPTER XXXV

SEBACEOUS DISEASES

This group of conditions represents disturbances of the sebaceous glands determined by excessive function. This in turn is the cause or effect of hyperkeratosis of the follicles, as the case may be; and subject to circumstances, inflammations or mechanical abnormalities, or both, arise.

Seborrhoea of the psoriasiform type has been described among the psoriasiform dermatoses (Chapter XI) and seborrhoeal dermatitis has been mentioned among the vesicular diseases (Chapter IX). Whether these are primarily seborrhoeal in the etymological sense is debatable. That group of conditions about to be described is definitely restricted to morbidity of the sebaceous glands and it includes five entities, i. e. acne, sebaceous cysts, milium, dermatitis papillaris capillitii and rosacea. The first four have close bonds.

Acne is a corruption of the Greek word acme meaning a point. Accordingly the term has been widely employed in other senses than the one to which we apply it restrictedly. Among these are acne artificialis, caused by tar, iodides and bromides; acne cachecticorum, due to tuberculosis; acne urticata, a form of chronic urticaria; and acne keratosa, a rare disease. Here we shall deal only with simple acne.

ACNE

Synonyms. Pimples, Acne Vulgaris, Varus; French, Acné; German, Hautfinne.

Definition. Acne vulgaris is a chronic disease of the sebaceous glands, characterized by an occlusion of their apertures with a greasy, horny plug, and an associated suppuration of the periglandular tissue.

Symptoms. The disease begins at or about puberty with the gradual development of plugs in the sebaceous follicles. These plugs are called comedones and they are black at the skin surface, above which they just perceptibly project. When expressed they are found to vary in appearance. Some are hard elongated casts of the hair follicle above the level of the sebaceous emunctory. These are a

millimeter or two long, gray and waxy. Others are the size and shape of millet seeds. Others have a black tip, but when expressed come out in a curled vermiform shape, as oil paint emerges from a tube. At times they are combined, the first part expressed resembling the first variety, the second part, the second. Some are hard to express, others easy. At times two follicles unite and contain a single comedone with two heads. These are called bridge comedones. Huge comedones, an eighth inch in diameter, are sometimes seen, and these are known as giant comedones.

The sites affected are the forehead, temples, nose, chin, ears, angles of the jaws, chest and back. Any part of the face may be involved and often the shaft of the penis, scrotum and pubis are also. Thus the affected skin is studded with minute black points, and a sensation of roughness is imparted to the palpating finger. These plugs dam back the sebaceous secretion to such an extent that, in extreme cases, the sebaceous gland becomes prominent because it is choked with its own product. In this picture a pearly papule is present surmounted by a black dot. In other words many minute sebaceous cysts are formed. The mechanism of the process is a hyperkeratosis of the follicle mouth. The exfoliated cells are not extruded and become saturated with sebum. The surface portion turns black because it contains substances reducible by oxygen. When the hyperkeratosis is so situated that it cuts the comedone off from the air no oxidation occurs. Hence no black dot forms, but a comedone is present, as expression will prove. When minute cyst formation occurs white seed-like dots appear, particularly on the cheeks and temples. These are milia. They are chalky in color, inexpressible, and when removed by curetting off the surface cuticle. a small glistening spherical mass may be pressed out.

The skin in the involved areas is usually oily, more or less muddy and thick. Either the disease remains limited to the comedone stage, in which a lustreless complexion is seen, studded with black dots; or suppuration sets in. The abscesses or acne pustules vary in size from minute dimensions to a diameter of half an inch. At first a papule arises, in the centre of which is usually observed a comedone. Gradually pointing develops, then fluctuation. The abscess bursts, crusts and heals, leaving a red spot which very gradually fades. On expression a drop of pus and a comedone come out. The life history of a pustule is from four to ten days. If the papular stage persists the condition is called acne indurata, when there is great induration. Otherwise it is called acne papulosa. Acne punc-

tata is the designation for the condition when numerous minute pustules are seen. The color of the pustules and papules ranges from pink to purple. At times deep cutaneous abscesses are intermingled with the pustules. The pustules, comedones, dull skin and oiliness, together form a varied picture.

When the process is over, the skin remains somewhat thickened, the follicles are patulous, and numerous pocklike scars are seen, so that the smoothness of the integument is lost. Coincident with acne one often finds seborrhoea of the scalp, furunculosis, and a peculiar, fine, pale line just under the vermilion border of the lower lip, and a brownish discoloration of the chin and temples. The pustules often come in crops particularly in relation to the menses; and in men, at times, in relation to coitus. Often hyperglycemia, anemia, acid stoods, gastric hyperacidity, and starch indigestion are associated.

Course. The disease starts at or about puberty and lasts as a rule well into the third decade, but it may end sooner or later, rarely persisting until thirty-five or forty.

Varieties. The varieties of acne vulgaris have been included in the symptomatology. These are purely artificial and are senseless distinctions. Neither is there any justification for distinguishing comedones from acne so far as the underlying cause is concerned.

Differential Diagnosis. Acne differs from staphylococcus folliculitis in that in the latter the pustules are pierced by hairs, and no comedones are present. Bromide and iodide acne are also distinguished by absence of comedones, but bromides and iodides may cause an eruption in people suffering with acne vulgaris, and in this event only the history will facilitate the diagnosis. Miliary lupus and acne necrotica are also recognized by the character of the lesions and absence of comedones. The positive signs of acne vulgaris are the comedones, pustules and the age at which the disease occurs.

Etiology. The etiology of acne vulgaris represents a wide field for speculation. Before going into details it may be well to recall some of the broader features of the malady. It is a condition arising at or about puberty, tending to spontaneous termination at the end of adolescence. It is often increased in severity at the menses, or in males after coitus. These facts strongly suggest its relationship to metabolic changes peculiar to the period of maturing. In the last analysis this indicates dependence upon endocrinous gland alterations. Such evidence is significant but of course only circumstantial.

Further elements in the causation of acne may be divided into two groups, internal and external. The internal causes are digestive and evidently sexual. Thus constipation, carbohydrate indigestion and fermentation, gastric hyperacidity, and even gastric ulcer may be found associated with acne. Excessive ingestion of sugars and starch alone, often exaggerate attacks. Allied with acne, hyperglycemia is fairly constant. It may be alimentary or perhaps depending upon hyperthyroidism, a frequent feature of puberty. The sexual elements in its causation are indicated by the influence of menstruation and coitus.

The external causes are irritation and bacteria. In one sense this statement is untrue. Acne is obviously a disease of hyperkeratinization, and sebaceous hypersecretion, inasmuch as the cuticle forms excessively and the glands secrete excessively, producing a mass of greasy scales which fill the follicles. This mass acts as a foreign body and suppuration takes place. To all intents and purposes the disease is present when comedones form. Suppuration is a superadded feature depending upon irritation and pathogenic bacteria of many varieties; staphylococci, bottle or acne bacilli, related to the Friedlander group. The pathogenicity of the latter group is by no means proved.

Massage, rough handling of the skin with coarse towels, unskilful expression of comedones and pustules, all make the disease worse, and often convert a simple pustule into a persistent papule or sluggish abscess or furuncle.

Treatment. The indications are clear. A test meal indicates the special digestive regimen to be followed. In general a diet designed to overcome constipation, lessen hyperacidity, and diminish the starch intake should be prescribed. Such medication as cathartics, antacids, digestive enzymes should be added as indications arise. Surgical conditions predisposing to the above should receive their special operative relief. Gynecological disturbances supply their particular indications. Obviously in weak, undernourished, atonic or asthenic individuals suitable methods of life should be prescribed, but this all constitutes a generalization applicable to all illness and requires no further emphasis. Arsenic is of no use in acne; neither is calcium sulphide, and in fact most tonics and alteratives are of little service outside of the imagination.

The local indications for treatment are twofold: first, to prevent hyperkeratosis of the follicles and hypersecretion of the sebaceous glands; and, second, to cure the condition if already established, as

well as to cure pustulation. The prophylactic treatment consists in the use of Roentgen rays, one Holzknecht unit weekly to a given area, which overcome exfoliation and hypersecretion. Medicaments also serve this purpose. Sulphur, resorcin, or salicylic acid lotions applied every night, after washing gently with soap and hot water, are the best methods. The following are types of these lotions:

℞ Potass. sulphurat.
Zinci sulphat. .....*aa* 6.0 to 10.0
Aquae Rosarum qs. ad. 100.0
Sig.

℞ Rosorcini ... 3.0–5.0 or acidi salycilici 2.0
Glycerini 5.0
Alcoholis q.s. ad. 100.0
Sig.

The first should be shaken thoroughly before applying. When resorcin is employed, the patient should be warned as to his eyes, for resorcin may cause conjunctivitis. These preparations cause exfoliation which favors the emptying of follicles and pustules, a mechanical task to be done by the physician, with proper comedone extractors. Vaccines have been of little value so far as the author's experience goes.

Prognosis. The majority of cases of acne are curable, but at times the utmost persistance is needed. The cosmetic result depends upon the type of the pustules which, when large, leave permanent scars.

Milium is a disease which may occur at any site involved in acne. The lesions are minute, white semiglobules. They may be sparse or numerous and can be cured only by removal with a sharp scalpel or bistoury. They are essentially tiny sebaceous cysts, and are seen chiefly near the eyelids and on the cheeks.

Sebaceous Cysts, wens, atheromas, or steatomas occur also at the sites favored by acne. They are deep seated tumors with a black point (comedone) at the site of which they are attached to the cutis, elsewhere being free. They are moveable at all points but the one mentioned. They may be infected and become abscesses, or rarely undergo malignant alteration giving rise to epithelioma. Their treatment is surgical. When infected they are to be managed as any abscess. As non-infected, growing cysts they must be carefully dissected out.

Dermatitis Papillaris Capillitii, also called acne keloid, and dermatitis sclerotisans nuchae, is a disease in males, and located

over the back of the neck from about the collar line to the occiput. The clinical picture consists of pustules, comedones, and broken off stubbly hairs emerging singly or in brushes from patulous follicles. The feature giving the disease its name is the presence of hypertrophic or pseudokeloidal cicatrices at the site of former pustules. These in turn further predispose to distorted follicles, occlusion cysts and comedones which suppurate. The only efficacious therapy lies in the employment of Roentgen rays. In certain cases electrolysis and local treatment, as in acne, avail.

ROSACEA (FIG. 79)

Synonyms. Acne Rosacea; French, Couperose; German, Kupferrose.

Definition. Rosacea is a chronic disease of the face depending upon vascular dilatation with the formation of telangiectasia. As the result of hypernutrition of the sebaceous glands the skin is oily, comedones form, often with pustulation, and there is a marked tendency to hyperplasia of the pilosebaceous follicles and connective tissues.

Symptoms. At first the face flushes easily upon exertion, irritation, exposure to extremes of climate and the ingestion of hot or highly seasoned food. Gradually the vessels dilate, telangiectasia and blotches forming, and the skin grows oily. The disease has its ups and downs, at times disappearing entirely, then after a dietetic indiscretion, after an attack of dyspepsia, with the menses or at the menopause, it gets worse. At times the patient complains of a sensation of facial heat. The onset of the disease occurs in adolescence or in the third or fourth decade.

After persisting in the stages mentioned for months or years, the flush becomes permanent, many vascular blotches or coarsely dilated veins are seen, papules and pustules develop and the skin is oilier. Admixed with this picture is that of acne. Still later hypertrophy of the cutis causes fibromatous masses, particularly on the nose. This provokes a grotesque swelling that may increase the dimensions of the organ three- to five-fold. This is rhinophyma, and the nose is a huge, red or purple fibromatous mass, cribriform with patulous follicles containing inspissated sebum, traversed by engorged red or purple vessels and covered with pustules and papules.

The sites involved in the process are the nose, chin, cheeks and temples. In the picture one discerns a composite of scaly and oily seborrhoea and telangiectasia, acne vulgaris and connective tissue

hypertrophy. Usually there is an associated gastric or pelvic malady. In fact these probably bear an etiological relationship to the disease.

Course. The disease is eminently chronic, beginning in adolescence or adult life and following a course of years. It may be arrested and remain stationary in its earlier stages, or continue to rhinophyma or to similar hypertrophy elsewhere than on the nose.

Diagnosis. Only lupus erythematosus discoides is simulated. In this condition there is scaling and atrophy, however, and no pustules are seen. Thus confusion can arise only during the early stages of rosacea, and here it is possible to exclude error only through observation.

Etiology. Aside from the fact that puberty plays no rôle in the causation of rosacea the etiology of the disease is identical with that of acne vulgaris. On the other hand the menopause certainly is the starting point of many cases. More specifically alcoholism, constipation, gastritis, the use of spicy, over-hot foods all play a distinct causative rôle.

Treatment. The general and local treatment are as in acne vulgaris. X-rays are harmful however. Scarification of the face is useful. Proper treatment of the digestive tract, as suggested by a consideration of the etiology, often works wonders. Rhinophyma responds only to surgical treatment. The patient is anesthetized, and the redundant tissue ablated and covered with a tight dry dressing.

Prognosis. Many early cases may be arrested by proper general and local treatment. Many late cases can be improved, and a few cured. A large number in all stages are obstinate.

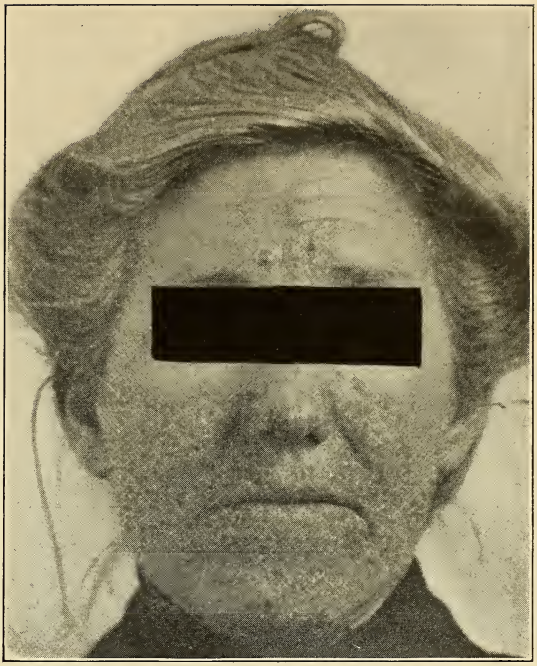

FIG. 79. ROSACEA

At first, the face flushes, then the vessels are permanently dilated and pustules form. The nose may hypertrophy, giving the picture of rhinophyma.

CHAPTER XXXVI

DISEASES OF THE SWEAT APPARATUS

Diseases of the sweat apparatus are secretory and organic.

I. Secretory Disturbances
   1. Disturbances of amount of secretion
      A. Excessive secretion, hyperhidrosis
      B. Diminished secretion, or hypohidrosis
      C. Absence of secretion, or anhidrosis
   2. Disturbances of character of sweat
      A. Colored sweat or chromidrosis
      B. Malodorus sweat or bromidrosis
      C. Sweat containing abnormal substances, as
         (a) uridrosis
         (b) phosphoridrosis
         (c) hematidrosis

II. Organic Disturbances
   1. Inflammatory
      A. Sudamina
      B. Miliaria rubra
      C. Miliary fever
      D. Pompholyx
   2. Infectious, staphylogenic infections of the sweat glands or hydradenitis suppurativa staphylogenes
   3. Nevi
      A. Granulosis Rubra Nasi
      B. Syringocystoma
   4. Unclassified. Hydrocystoma

Thus it will be seen from the above table that diseases of this system are neither numerous nor important. Indeed the majority of them are rare. The practical student of dermatology need bestow no more than a passing regard upon the subject.

Hyperhidrosis, called hydrosis and by several other names, is an increased flow of sweat. It may be local, general, transitory or chronic, and slight or marked. It is physiological or pathological. Some people perspire more than others. Likewise some body areas

are moister than others, as the armpits, groins, perineum, palms, soles and spaces between the fingers and toes. The gradations from the normal to abnormal are difficult to trace. Cold weather inhibits perspiration. Emotional strain and excitement increase it. The flow may be so great as to cause drops to form on the skin even in cold weather. The disadvantages of excessive sweat flow are obvious. Localized hyperhidrosis, symmetrical or asymmetrical, sweating of the nose, forehead or chin are occasionally seen. Spicy foods cause hyperhidrosis in some people, excitement does so in others. Drugs such as salicylates, quinine and pilocarpine are sudorifics.

Tuberculosis causes the well known sweats. The end of severe fevers as in malaria, pneumonia, typhoid, are attended by perspiration. In cachexia of any origin sweats are common. This is also true in certain central and peripheral nervous maladies. The precise mechanism in these processes is not understood. Thus etiologically there is very little to be stated.

There is very little to be accomplished therapeutically. Atropin is the only rational drug and this is of little value because the condition recurs when the drug is stopped. Various local remedies, powders, lotions and the like, are employed. Their number testifies to their inefficiency. Perhaps weak formalin, five percent. in alcohol, and salicylated powders do most good. The former is unpleasant and prone to cause dermatitis. Furthermore the damming back of the secretion predisposes to furunculosis. Diachylon ointment is of some value. Roentgen rays, in a dose of one Holzknecht unit, weekly, for ten or twelve exposures, causes partial atrophy of the coil glands. This is of most value. The prognosis is bad.

Anhidrosis and Hypohidrosis are rare conditions. Their characteristics and symptoms are obvious. People with psoriasis, ichthyosis, and nephritis are prone to the condition. Pilocarpine is the only useful drug but it is dangerous. A practical point is to note that anhidrosis predisposes to sun and heat stroke.

Chromidrosis is rare. The sweat is colored blue, brown or yellow and each form is suitably named. The pigments are phosphorus, hemoglobin derivatives, indican, cyanogen, etc. Trichomycosis palmellina causes red sweat, and there is also a black sweat. No valuable therapy is known.

Uridrosis is due to urea in the sweat. Phosphoridrosis is occasionally seen in cachexias. Hematidrosis is seen in the hemorrhagic diseases. All are unimportant.

Bromidrosis or malodorous sweat is usually allied with hyper-

hidrosis. Any site may be affected. Various drugs, foods and diseases cause it in a passing form. The odor is most offensive and the treatment is that of hyperhidrosis.

Sudamina or miliaria crystallina is a condition in which, because of occlusion of the pores, the sweat is temporarily dammed back so that a sweat cyst is formed. The cysts are crystalline vesicles. As a rule the torso is involved, and is studded with lesions resembling tiny limpid dew drops. They are very delicate structures, the obstruction being but a few layers of horny cells easily removed, so that in bathing they vanish. This fact indicates the proper treatment. After the bath talcum powder is used. Excessive sweating whether due to exercise, heat or fever, causes the rash, the predisposition to which is twofold, overacting glands and hyperkeratosis.

Miliaria rubra, lichen tropicus, prickly heat, is a vesiculo papular disease frequently observed in hot weather. Itching or burning accompany it. The same factors and significance are peculiar to it as to the preceding condition. It differs from the latter in that it is inflammatory. It is peculiar to infants and children, but also afflicts adults. At times it predisposes to dermatitis and, in the adipose, to intertrigo. It is cured by bathing and powdering.

Miliary fever is a rare exanthem, if it is indeed, a clinical entity, and requires no further mention.

Pompholyx has already been described (Chapter IX). Infections of the sweat glands are included in furunculosis (Chapter XXIV). Granulosis rubra nasi and syringocystoma have been dealt with among conditions probably nevi (Chapter XXXIII).

Hydrocystoma is a papulo vesicular disease of the face seen in middle aged women. It is not known whether it is inflammatory or not. At any rate the coil glands are converted into transitory cysts. Thus the lesions are deep seated. They are white or bluish papules containing a neutral or faintly acid fluid, and are the size of millet seeds. The face perspires easily and the sites involved are the forehead, cheeks and nose. Kitchen and laundry work are etiological factors. To cure the condition the predisposing cause must be eliminated, the cysts punctured, and a weak salicylated alcohol applied several times daily.

CHAPTER XXXVII

DISEASES OF THE NAILS

Disturbances of the nails are congenital or acquired. Congenitally the nails may be absent (anonychia) or nails may be abnormal in that they participate in such anomalies as supernumerary, fused or wrongly implanted fingers or toes. These conditions are rare and unimportant. Acquired disturbances of the nails are numerous and the following classification is suggested:

I. Anomalies
  1. Hypertrophies
    a. Onychauxis
    b. Pterygium
  2. Atrophies
    a. Onychatrophia
    b. Hapalonychia
    c. Koilonychia
    d. Onycholysis
    e. Onychomadesis
    f. Leuconychia
    g. Onychorhexis
    h. Furrows

II. Dystrophies (due to disease)
  1. Systemic
    a. Syphilis
    b. Tuberculosis
    c. Acute fevers
    d. Cachexias
  2. Local
    a. Psoriasis
    b. Dermatitis
    c. Erythrodermas, etc.

III. Inflammations of the nail bed
  1. Onychia and onychia maligna
  2. Paronychia
  3. Ingrown nail

IV. Injuries
  1. Hangnails
  2. Subungual hemorrhage
  3. Traumatic furrowing
V. Tumors
  1. Subungual
  2. Nail bed
VI. Infections
  1. Bacterial
    a. Pyogenic
    b. Tubercle bacillus (Tuberculosis verrucosa cutis)
  2. Fungus
    a. Trichophytosis
    b. Favus
    C. Blastomycosis, sporotrichosis, etc.
  3. Syphilis

Onychauxis or hypertrophy of the nails, also called onychogryphosis, may involve the nails in any combination from one to more, even including all the digits. Thus the nails may be thickened, lengthened, distorted and discolored. They are yellow, brown or black, hard or friable, and look like lustreless claws or talons. The surface is rough or rigid, and below the nail is a hyperkeratotic plug. Trauma, pressure, neglect, filth or systemic disease such as lepra may produce the condition, for which no cure exists.

Pterygium is a disease of the fold covering the base of the nail. The fold hypertrophies and covers the nail.

Onychatrophia is an atrophy of the nails, congenital or acquired through disease. The nail is defective, thin, breaks easily either longitudinally or transversely, is lusterless and ridged. In *hapalonychia* the nails are thin and brittle. *Koilonychia* is a dystrophy in which the dorsal surface of the thin nail is concave instead of convex. *Onychonychia* or *onycholysis* is a condition in which the nail easily separates from its bed. *Onychomadesis* is a condition in which the nail easily falls off. These are all due to systemic or trophic diseases. In *leuconychia* white spots form on the nails. In *onychorhexis* the nails split longitudinally, the split starting in longitudinal furrows or flutings. *Transverse furrows* are the result of injury from manicuring, local or general diseases, and are often transitory. The treatment in all of these conditions is unsatisfactory and the cosmetic outlook poor.

Dystrophies. Many of the above conditions are the result of central nervous, systemic or local diseases. They are seen in

syringomyelia, tabes, syphilis, the cachexias, acute febrile diseases, tuberculosis, psoriasis and dermatitis. After fevers transverse furrows develop and have a meaning similar to post-febrile alopecia. Each point corresponds to an original psoriasis lesion of the nail bed. Also in psoriasis (Chapter XI) scaling occurs under the free margin extending down and lifting off the nail. The treatment is to put antipsoriatic substances on cotton underneath the nail, previously having softened the scale with soap and hot water. Fractional doses of X-rays are also indicated. In dermatitis of the nail (Chapter IX) vesicles develop at the base causing dystrophies of the mature nail similar to those observed in psoriasis. In squamous dermatitis psoriatic nails are closely simulated. In both diseases the nails become lustreless and brittle. In psoriasis they tend to become thin. In chronic dermatitis they become thick, friable and suggest mild forms of onychogryphosis. In the erythrodermas the nails atrophy.

Onychonychia is an inflammation of the nail matrix due to pyogenic infections, dermatitis or trauma. The nail bed is thickened and red. Onychia maligna is due, as a rule, to syphilis, tuberculosis or any infective ulcers at the nail border. In paronychia (pararitium or whitlow) streptococci (see impetigo contagiosa, Chapter XXIV) cause suppuration of the nail fold with the local symptoms of abscess formation. Wet dressings, and, if needed, surgical treatment produce a cure. Hangnails predispose to this. They are exfoliated or injured patches of cuticle.

Unguis incarnatus or ingrown nail is usually a disease of the great toe but may occur, albeit rarely, on any digit. Through careless paring, neglect, or injury a sharp spike of nail at the side of the bed grows and pierces the cutis, continuing to grow within the puncture thus made. It acts as a foreign body and at first causes inflammation with swelling, redness and pain. Infection may take place increasing these signs, and it is possible for systemic infection or phlegmon to ensue, although this is happily rare. The treatment is preventive and symptomatic. Prevention resides in careful hygiene and cutting. When the disease begins a small pad of cotton inserted between the nail and the flesh suffices. This should remain until the nail is long enough to be cut transversely from corner to corner. When inflammation occurs wet dressings should be applied, followed by the above. With infection the same treatment is indicated unless an abscess forms. Then surgery is indicated. At times it becomes necessary to remove part of, or

the whole, nail. This must be done under local or general anesthesia.

Hangnail is due to careless manicuring. In removing cuticle little fragments of epidermis may be partly lifted off with the implements. Injuries, or nail or cuticle biting produce the same effect. The fragment tends to tear back down to the corium and hangnails easily become infected and form abscesses, or suppurating paronychia. Likewise digital chancres begin in such lesions. The proper treatment is careful toilet of the hands and cutting away the hangnails down to their bases.

Subungual hemorrhages are due to injuries, mainly blows, or bruises to the nail or bed. A small suggulation develops (at first red, then brown), under the nail. This gradually grows out with the nail growth. In extreme cases the whole nail may fall, but it grows in again. Similar injuries, careless manicuring, occupation, etc., predispose to transverse or longitudinal furrows.

Tumors, chiefly epitheliomata, develop either in the nail bed or under the nail. All forms are rare. The treatment is X-rays or surgery, according to indications (Chapter XXVIII).

Pyogenic infections may occur in the nail bed or under the nail, giving the picture of suppurative paronychia. Redness, swelling or bleb formation (the so-called run-round) appear. The picture is seen in impetigo contagiosa, when it is caused by streptococci; or after puncture wounds, hangnails, ingrown nails, etc., when it is due to either type of pyogenic cocci. Rarely Klebs-Loeffler bacilli or Bacillus pyocyaneus are responsible. The treatment consists in removing the underlying cause of the impetigo, and the local application of wet dressings, or antiseptic creams, chiefly the conventional mild mercurials.

Tuberculosis verrucosa cutis may arise about the nail (Chapter XXV). Fungous diseases have already been described (Chapter XXVI).

SYPHILIS OF THE NAIL

Syphilis, either congenital or acquired, involves the nail in all stages by attacking either the bed or nail substance itself. Primary syphilis usually is seen near the fold of cuticle on the one side of the nail, the starting point as a rule being a hangnail or some other injury. Thus in primary syphilis only one nail is diseased. In other forms more than one nail is affected. In syphilitic periostitis or dactylitis, either in the secondary or tertiary stages, dystrophy of the nails may be produced because of secondary involvement from the

subjacent tissue. Thus a furrowed or distorted nail is seen, with a swollen, red, infiltrated phalanx. In tertiary syphilis there may be a gumma of the nail bed or folds, or syphilitic onychia or paronychia.

In onychia syphilitica the nail becomes dull red, yellow or brown, is striated, furrowed, friable and often fissured. The cuticle scales, and the plate pits, as in psoriasis. The nail is shed and replaced repeatedly. Paronychia is evidenced by a dull red, infiltrated, scaly fold looking like chronic paronychia. Usually several nails are involved, or all are, and rarely ulcers or gummata develop.

Congenital lues infrequently attacks these organs, but when it does the various forms described are seen just as in the acquired condition. The treatment is that of systemic syphilis.

GROUP XI. SKIN DISEASES RELATED TO NERVE DISTURBANCES

CHAPTER XXXVIII

CUTANEOUS DISTURBANCES DEPENDENT UPON OR ASSOCIATED WITH NERVOUS DISORDERS

Central sympathetic peripheral and functional nervous and mental diseases may produce, or be associated with, cutaneous manifestations.

I. Central Nervous Diseases
   1. Epidemic cerebrospinal meningitis, spotted fever (Chapter VII)
   2. Tabes dorsalis, malum perforans pedis (Chapter XLIV)
   3. Syringomyelia
   4. Morvan's Disease
   5. Herpes. Ganglion hemorrhages (Chapter IX)

II. Sympathetic Nervous Disturbances
   1. Addison's Disease, secondary to diseases of the chromaffin system (Chapter XVIII)
   2. Acanthosis nigricans, abdominal neoplasms, and relative to abdominal sympathetic system (Chapter XV)

III. Peripheral Nervous System
   1. Lepra, neurolepra and neuroleprides (Chapter XXV)

IV. Trophoneuroses
   1. The gangrenes and decubitus (Chapter VIII)
   2. Glossy skin (Chapter XVII)

V. Angioneuroses
   1. Angioneurotic oedema (Chapter VII)

VI. Mental Disease and Deficiency. Functional Diseases
   1. Recklinghausen's Disease (Chapter XXX)
   2. Granulosis rubra nasi (Chapter XXXIII)
   3. Analgesias — parasthesias, etc., of hysteria

VII. Metabolic Diseases
   1. Uremia; pruritus, formication
   2. Diabetes, pruritus, formication
   3. Arteriosclerosis, pruritis, numbness, tingling

VIII. Unclassified Conditions

1. Pruritus hiemalis and paririe itch
2. Dermatalgia
3. Meralgia
4. Trichotilomania
5. Dermatochalensia

The cutaneous manifestations of central nervous diseases are probably trophoneuroses and might be classified as such were it not for their known etiology. In tabes a perforating ulcer of the foot known as malum perforans pedis develops. Other causes of this condition are paresis, syringomyelia and various metabolic and infectious diseases involving the cord. Actually lepra and syringomyelia cause more confusion than does either with tabes. Rarely a similar condition appears on the palm. The treatment is that of any ulcer and is unsatisfactory because of the nature of the underlying cause.

In SYRINGOMYELIA and Morvan's disease, because of loss of pain sense, all sorts of injuries and lesions may occur, and since patients are often unaware of them they may become large and obstinate. Ulcers, whitlows, vesicles and bullae form, and as the trophic disturbances become profounder spontaneous amputation of the phalanges may take place. In addition hyperalgesia and hyperhidrosis are occasionally observed. The treatment is symptomatic. All gangrenes, including decubitus (Chapter VII), may be in this group, as may also be glossy skin (Chapter XVII). It is also widely believed that scleroderma (Chapter XVIII) too is thus to be classified.

LEPRA (Chapter XXV) and ANGIONEUROTIC OEDEMA (Chapter VII) have already been dealt with. The relationship of RECKLINGHAUSEN'S DISEASE (Chapter XXX) and GRANULOSIS RUBRA NASI (Chapter XXXIII) to mental deficiency has been alluded to. This association is neither clear, close nor constant. In **hysteria** there may be all sorts of parasthesiae, itching, analgesia and hyperasthesia. There may be habits, such as scratching the skin, or pulling hairs, that may cause traumatic lesions, and **hysterical mutilations** and **feigned eruptions** (Chapter VIII), belong in this group.

In **nephritis**, particularly with chronic uremia, in the acidosis of diabetes, in leucemia, and arteriosclerosis, there may be itching; and in all but the leucemias, tingling, formication, hyperalgesia and analgesia may be present. These may all be regarded as dermatalgias, either essential, or of central nerve origin, unless an effort

is made to determine the underlying cause. In every case with such skin phenomena examinations for the underlying cause should be conducted, and if the grosser metabolic changes are not found an expert neurologist should be consulted, particularly with the idea of determining a factor such as early syringomyelia.

Pruritus or itching is the commonest symptom of skin diseases. Pruritus essentialis is that form in which no cause can be found. Another form is senile. Other varieties still are anal, vulvar, and nasal, the last usually due to hay or rose fever. Anal pruritus is often inexplicable; but often is the earliest sign of tabes, and at times is associated with intestinal parasites, carbohydrate fermentation or hemorrhoids. Vulvar pruritus is usually secondary to the above causes or to vaginitis or diabetes. Only to those cases in which no etiology is ascertainable may temporarily still be applied the term essential pruritus, secondary to scratching which arises dermatitis with or without lichenification.

In this group is winter itch, or pruritus or prurigo hiemalis. It always appears in cold, and leaves in warm, weather. It gets worse when the patient undresses. Prairie itch, a disease of the West, may be similar, but is more likely due to a mite. Seasonal itching is probably the result of hypersusceptibility of the cutaneous nerves to thermal changes. Similar phenomena occur in some people after bathing.

The treatment of pruritus essentialis is varied and unsatisfactory unless the underlying factor is found, and if found, curable. Certain obvious indications are thus furnished. Antipruritics are numerous and on the whole unsatisfactory. Wet dressings, lotions containing one, two or five percent. of menthol or phenol, or sprays of these substances are sometimes useful. Mentholated (two percent.) zinc stearate powder, alone, or after such applications as have been mentioned, is frequently most valuable. Even mentholated or phenolated oils are good. Ointments are poorly borne as a rule. Camphor and cocaine, in various vehicles, are employed, but are rarely valuable. Bran and particularly mentholated starch baths are good. The latter may cause cardiac collapse, and phenol when employed over extensive areas may cause nephritis. Hence care should be employed in using these substances. Internally, morphine and bromides are often of avail, but they themselves may provoke itching. Pilocarpine and sweating have been recommended. The former is dangerous and overstimulation of the sweat glands may increase the pruritus and inflame the skin.

Dermatalgia or painful skin is rare and of many qualities, resembling the feeling or rubbing, cutting, bruising or burning. There are no objective signs. *Meralgia* is pain in the outer anterior surface of the thigh. The sensations are tingling, numbness, pain and other parasthesiae. Effort increases them. Treatment is unsatisfactory. *Trichotilomania* is a compulsive desire to pull at hairs. It is really a neurotic habit or mannerism, and may cause secondary lesions, infections and traumatic alopecia. Scleroderma, erythromelalgia, some of the other atrophies, thromboangitis obliterans, some forms of acroasphyxia are considered neurological in origin. No satisfactory evidence thereof exists.

GROUP XII. DISEASES OF THE VISIBLE MUCOUS MEMBRANES

CHAPTER XXXIX

DISEASES OF THE ANUS, CONJUNCTIVA, BUCCAL AND GENITAL MUCOSA

Cutaneous diseases often involve the visible mucosa, and there are also diseases peculiar to the mucous membrane which do not involve the skin but which are included in the general science of dermatology. It is necessary to know these conditions to be an enlightened practitioner, but since a detailed description of them would of itself double the size of this book, they will be catalogued as briefly as possible.

Conjunctiva. The conjunctiva, as compared with other similar tissues, is subject to relatively few of the disturbances in question. At times erythema multiforme and urticaria may occur at this site, particularly angioneurotic edema (Chapter VI). Many of the purpuras (Chapter VII) are found here, and, above all, the petechiae of sepsis. Both simple herpes and zoster (Chapter IX), the latter also on the cornea, are by no means rare. Pemphigus of the conjunctiva (Chapter X), both as bullae in pemphigus vulgaris, and as a condition called Graefe's essential shrinking, must not be forgotten. The pemphigus bulla easily breaks and the floor remains as a superficial conjunctival ulcer. The shrinkage of Graefe's form resembles cicatricial conjuctivitis. Lesions of dermatitis herpetiformis rarely involve the conjunctiva. The erythrodermas (Chapter XII) cause very little conjunctival trouble save in a secondary sense. All of them, but chiefly pityriasis rubra of Hebra, are capable of producing ectropion with the usual consecutive conjunctival changes thereof. The same is true of scleroderma (Chapter XVII). Pediculosis pubis and pediculosis capitis (Chapter XX), the former by its presence in the lashes, the latter by its irritation from the long infected scalp hairs, cause mild conjunctivitis. Tubercular (Chapter XXVI) conjunctivitis is known in children, and conjunctival lupus vulgaris is not rare. Conjunctival epitheliomata (Chapter XXVIII) are seen, particularly rodent ulcers; and in xeroderma

pigmentosum, on the conjunctiva, together with the cornea it is a usual finding. Vascular nevi (Chapter XXXII) are occasionally seen, as a rule the conjunctival involvement being simply participatory in a process including the whole eyelid. Diseases peculiar to the conjunctiva belong in the province of ophthalmology.

Diseases of the buccal orifice, including the nose, are numerous. Those peculiar to these regions will be dealt with separately after the general conditions have been indicated. Erythema multiforme and urticaria (Chapter VI) are not infrequently seen. Particularly the lips are involved, and in the former are often swollen and crusted, while in the latter (angioneurotic edema) they are greatly distorted. Lead poisoning (Chapter VII) and bismuth poisoning cause characteristic gum lines. In the exanthemata (Chapter VII) definite buccal signs exist. In measles Koplik's spots are found, in scarlatina the strawberry tongue, in German measles petechiae of the soft palate and uvula. The purpuras, too, (Chapter VII) involve buccal structures, and labial and gingival petechiae are common in sepsis. Early in mumps an erythematous ring surrounds the opening of Stensen's duct. After measles and in other wasting diseases (Chapter VIII) noma and nosocomial gangrene develop. Herpes (Chapter IX), and zoster in all parts of the mouth and nose, are by no means rare. Pemphigus vulgaris (Chapter X) is common in the mouth, and even the lesions of dermatitis herpetiformis, bullous erythema and urticaria are not unknown. Psoriasis (Chapter XI) is rare. The erythrodermas (Chapter XII), chiefly pityriasis rubra of Hebra, cause ectropian of the lips. The lips, mucosa of the cheeks and tongue are commonly involved in lichen planus (Chapter XIII). Scleroderma (Chapter XVII) causes changes in the lips similar to those in the eye. Addison's disease (Chapter XVIII) characteristically produces buccal pigmentation. Diphtheria (Chapter XXIII) causes well known mouth lesions, and in typhoid fever ulcers on the anterior faucal pillars are encountered during the prodromal period and first week. Vincent's angina must not be overlooked. Streptococci (Chapter XXIV) cause perlèche. The various forms of tuberculosis and lepra (Chapter XXV) seen in the nose, mouth and larynx need only be mentioned to reveal their importance. Lupus erythematosus (Chapter XXV) is seen on the lips. As to non-infectious granulomas (Chapter XXVI), it is essential to remember that acute leucemia may cause tonsilar changes suggesting diphtheria and Vincent's angina. Warts (Chapter XXVII) occur on the lips, and

lesions resembling multiple warts or pointed condylomas are noted in the mucosa of the lips and cheeks. All sorts of epitheliomata (Chapter XXVIII) are seen on the lips, tongue and elsewhere. In this connection, and particularly with reference to sarcoma (Chapter XXXI) confusion with salivary ranulae and Miculicz's disease is possible. Nevi, particularly the vascular and lymphatic, are common. Syphilis appears in its primary form on the lips, tongue, tonsil and uvula; in its secondary form, throughout the buccal orifice as mucous patches; in its late forms, as gummata of any of the structures but prevailingly of the tongue. Interstitial glossitis is common, and throughout the mouth specific leucoplacia is frequently seen.

A few special points concerning buccal forms of diseases not restricted to the mouth, and a few diseases peculiar thereto require further emphasis. Of the latter some are seen on the lips, some on the tongue, some on the entire mucosa. On the whole these conditions demand no great space.

A. Diseases peculiar to the tongue.
   I. Anomalies
      1. Grooved tongue
      2. Scrotal tongue
   II. Infections
      1. Black hairy tongue
      2. Sprue
   III. Inflammations
      1. Moeller's glossitis
      2. Erythema migrans
B. Diseases peculiar to the lips
   I. Anomalies
      1. Fordyce's disease
   II. Infections
      1. Perlèche (Chapter XXIV)
   III. Inflammations
      1. Cheilitis glandularis exfoliativa
C. Diseases peculiar to the entire buccal mucosa
   I. Inflammations
      1. Ulcerative stomatitis
      2. Foot and mouth disease
D. General diseases involving the buccal mucosa and requiring special mention

I. Inflammations
  1. Leucoplacia buccalis
  2. Lichen planus (Chapter XIII)
  3. Lupus erythematosus
II. Infections
  1. Syphilis
    a. Chancre (Chapter XLI)
    b. Mucous patches (Chapter XLII)
    c. Gummata (Chapter XLIII)
    d. Interstitial glossitis (Chapter XLIII)
  2. Tuberculosis
    a. Miliary ulcers (Chapter XXV)
    b. Tubercular granulomata (Chapter XXV)
    c. Lupus vulgaris (Chapter XXV)
  3. Vincent's angina
III. Neoplasms
  1. Epitheliomata (Chapter XXVIII)

Grooved Tongue is simply a tongue in which the ordinary markings are intensified. When that is so to a marked degree the convolutions suggest the topography of the scrotum. This is the scrotal tongue.

The Hairy Black Tongue is the result of a hypertrophy of the filiform papillae which may reach an inch in length. They form a wedge on the dorsum. Many suppose a yeast fungus causes the condition. *Sprue* is a fungus disease of the tongue, epidemic at times in institutions, and characterized by the presence of white spots, due to a membrane consisting of epithelial detritus and the infective agent, the white oidium. *Moeller's glossitis* is a rare disease of the dorsum and margin of the tongue. The affected areas are glossy, red, swollen and extremely painful. The cause is unknown. *Erthyema migrans* or glossitis areata, or transitory benign plaques of the tongue is characterized by rings, the red margins of which wander, forming gyrate figures suggesting erythema multiforme. Within the red border is a white zone of oedema or exfoliation, and the centre of the large lesions is normal. This condition is also called geographical tongue. *Fordyce's Disease* is an anomaly. Misplaced sebaceous glands are seen on the inner aspect of the lips and cheeks, particularly the lower lips. The affected area is covered with scattered white or bluish white rather hard spots or globes, the size of a pin head. They are minute sebaceous cysts. *Cheilitis glandularis* is a rare exfoliative disease of the lower lip which is swollen, tense and covered with minute elevations corresponding to mucous glands with enlarged openings. The best treatment is with X-rays. Nothing is known of the cause.

Ulcerative stomatitis, canker sore or aphthous stomatitis is a condition in which minute ulcers form on the lower lip, below and

on the tongue. They are grey or yellow and have a narrow inflammatory red zone about them. They are sparse or numerous and painful. They rarely exceed a diameter of two millimeters. They are either infectious or herpes simplex. The best therapy is to paint them with twenty percent. silver nitrate solution. Foot and mouth disease is closely related, and is supposed to be similar to a condition seen in cattle. In man it causes aphthous ulcers and mild fever.

Leucoplacia buccalis is a chronic hypertrophic inflammation. It is pearly white, occurs in thickened, roughly circular or oblate patches which are sparse or numerous. Syphilis is its commonest cause and it predisposes to cancer. Lichen planus closely resembles it, but the patches are not smooth, are striated as on the skin, and most obstinate. Lupus erythematous prefers the lips. A white or gray hypertrophic margin with a red center, similar to that of a skin patch, characterizes the process. Details of syphilis and tuberculosis of the mouth are found in other chapters.

Vincent's angina closely resembles chancre, gumma or diphtheria, particularly when the tonsil is involved. On other areas of the mucosa mucous patches are simulated. The cause is Vincent's spirillum, and the treatment arsphenamin locally and intravenously.

Epithelioma of the lip and tongue is usually squamous and very malignant, involving the glands and metastasizing early. Epithelioma of the tongue is usually single, opposite the molars, often arises from leucoplacia, is painful, and the pain is referred to the ear on the corresponding side. Glandular enlargements develop. The only therapy is radical operation. A cancer may simulate a chancre or gumma. The chancre contains spirochaetes, but it must be remembered that the pallida and microdentium are similar. Other evidences of syphilis must be sought. Gummata are usually multiple, but a gumma and cancer may coexist. In case of diagnostic difficulty a complete excision of the suspected growth should be made, and a microscopic examination should follow at once of frozen sections, the surgeon being ready to proceed with an immediate operation.

Anal diseases within the province of the dermatologist are not numerous. Dermatitis (Chapter IX) pemphigus, particularly pemphigus vegetans (Chapter X), which may be confused with flat condylomata, and psorospermosis (Chapter XIV) are seen more or less, according to the incidence of the respective diseases. Dermatitis is the commonest, particularly with pruritus ani. Pruritus is often inexplicable but may be an early sign of tabes dorsalis, or be

associated with itchy dermatoses, colitis, proctitis, hemorrhoids and starch indigestion with acid stools. Constipation is often responsible. The underlying cause must be removed, and Roentgen therapy is excellent. Secondary to pruritus, lichenification arises. Furuncles are often found near the anus. Tuberculous ulcers and various types of syphilides, particularly the chancre, mucous patches, flat condylomas and gummata are common. Epitheliomata (Chapter XXVIII) are not rare.

Diseases of the glans penis and mucous surface of the prepuce are dermatitis (Chapter X), herpes (Chapter IX) known as herpes progenitalis, balanitis, pemphigus (Chapter X), lichen planus (Chapter XIV), scabies (Chapter XX), erosive balanitis, soft chancres (Chapter XXIII), rarely tuberculosis (Chapter XXV), verrucae and pointed condylomas (Chapter XXVII), epitheliomata (Chapter XXVIII), the primary lesion of syphilis, mucous patches and gummas.

Diseases of the vulva are dermatitis and herpes (Chapter IX), pemphigus vulgaris and vegetans (Chapter X), kraurosis (Chapter XVII), scabies (Chapter XX), furunculosis (Chapter XXII), ulcus molle (Chapter XXIII), tuberculosis (Chapter XXV), verrucae (Chapter XXVII), epithelioma (Chapter XXVII) and syphilis, particularly the chancre, mucous patches, moist papules, flat condylomata and gummata. Pruritus vulvae is analogous in significance and causation to pruritus ani, and is also often secondary to vaginitis both specific and non-specific.

In all of the conditions described in this chapter the therapy has been indicated in other places in connection with descriptions of the individual diseases. The points raised with reference to syphilis in its various mucous manifestations will be elaborated in the description of this disease.

SECTION F. SYPHILIS

CHAPTER XL

GENERAL ASPECTS OF SYPHILIS

Mode of Infection and Infectious Mechanism. To understand syphilis, it is necessary to bear in mind some of the general facts, as they are to-day conceived, of the biology of the spirochete and host in their inter-relation. In other words, a practical understanding of the infectious mechanism constitutes the only basis upon which can be founded a rational insight into the clinical peculiarities and therapeutic problems of the disease. The cause of syphilis is a microörganism, probably a protozoön, possibly a member of the protista, that gains access to the host through an injury to the skin or mucous membrane. After an incubation period of from ten days to six months, the average, however, being three weeks, at the site of inoculation a sore develops. This lesion, if unmodified by treatment, persists for from three to six weeks and spontaneously involutes, leaving a more or less distinct scar which may or may not be pigmented. While this is going on the microörganisms are being disseminated throughout the host, the disease becoming generalized or systematized. The period of dissemination varies in length, usually extending over a number of years, and being characterized by waves of activity and quiescence, but gradually growing more restricted in its objective evidences. Finally, and lasting for years or perhaps through life itself, there are recrudescences of the restricted or localized phenomena. Each of the phases or stages mentioned has its own significance and has been given a special designation. The period elapsing between the exposure and appearance of the first sore is known as the primary incubation period. The first sore, itself, is known as the primary or initial lesion (Chapter XLI). The interval between the onset and general dissemination is called the secondary incubation period. The active era of dissemination is termed the secondary stage (Chapter XLII), or period of early syphilis, and the inactive recesses taken by the malady during this stage are called periods of secondary latency. Late or restricted

manifestations constitute the so-called tertiary stage (Chapter XLIII) and it, too, is characterized by rising and receding tides of activity separated by periods called tertiary latent periods.

Definition. Thus, syphilis is a chronic infectious disease running a course covering a number of years, and obviously having a tendency to self-limitation. This tendency evinces itself not only in the behavior of the malady as a whole, but also at every major and minor step in its progress. Such evidence of this as we possess will be adduced below, when each of these steps is more fully described, but it may here be stated that the peculiarities of the disease must depend either upon phenomena in the life cycle of the parasite, or upon peculiarities of the host's protective mechanism, or, what is more likely, upon both. Before going into this, however, it would be well to become familiar with the parasite itself.

The Spriochete. The organism is a highly motile, flagellated, refractile body, from fifteen to forty microns long, that can best be studied by dark field illumination, and while alive. Its mode of division is not definitely known; it grows anerobically on Noguchi's medium, and it may be capable of entering into resting or encysted stages. In breadth, it is but a fraction of a micron, and it is wound in fine spirals like a spring, perfectly even and from twelve to twenty in number. Its movements are complicated, and consist of a corkscrew forward or backward motion upon the long axis, and a bending motion upon a point at or near the middle, as a fulcrum. At times it is possible to see the flagellae at the delicately tapered tips, lashing about, but these organs can best be demonstrated in dead specimens subjected to the Giemsa stain. Meierowski has found bodies which he takes to be nuclei, and which he regards as connected with the procreative phases of the organism. The spirochete is also called the treponema pallidum, by virtue of its striking palor when stained. In genital lesions, it must be differentiated from a coarser related organism, the spirocheta refringens, which is once or twice its size, and eel-like in contour. In mouth lesions are found, not only the above saprophytic organism, but also another, more capable of causing confusion because more like the syphilis germ in appearance. This is the spirocheta microdenitium, which differs from the spirocheta pallida only in length. It is equally delicate, but very short, showing but six or eight twists, and it is less active. The organism of syphilis was discovered by Schaudin.

Dissemination of the Spirochete. As stated, the portal of entry is a solution in continuity in the skin or mucosa. Because trans-

mission usually takes place during coitus the initial lesions is nearly always situated upon the external genitals. At the moment of infection the spirochetes gain a foothold in the injured tissue, multiplying and invading the local lymphatics, and rapidly entering the lymph vessels. This must occur very quickly, as was illustrated in his lectures by Jacobi of Freiburg. A student with herpes, within twelve hours of intercourse with a woman he discovered immediately post coitum to be actively syphilitic, was circumcised, with the hope of thus warding off infection. Although no primary lesion ever developed, in due time the secondary stage appeared. This gives a good idea of how soon the organisms get far beyond the site of inoculation, and how futile the attempts are to modify the gravity of the disease by excising a fully matured initial lesion, a procedure once widely advocated. At its inception, syphilis is a disease of the lymphatics, and its early avenues of dissemination are restricted to this system. If a chancre be studied histologically by the Levaditi method, the perivascular lymph spaces will be found filled with the microörganisms, and here and there one of the latter will be seen penetrating the wall of a small blood vessel, thus entering the general circulation. In other words, the manner of spreading has now become twofold, hematogenous and lymphatic. This is what goes on during the initial stage after the development of the primary lesion. In the meantime, the chancre has begun to involute. Undoubtedly, this must be the result of a developing local immunity, on the part of the affected tissue. But the host has been completely invaded by multiplying organisms, and the secondary incubation period is over.

The Biology of the Early Stage. With the conclusion of the latter, the disease has reached the secondary or eruptive stage, one characterized by tissue responses, and subjective and objective symptoms referable thereto in all body organs. The host has now become a huge battle field in which the spirochetes, armed with their toxins, wage war against the host's cells defended by immunizing substances, whatever they may be. With that niggardly economy so often witnessed in nature, immunity never seems quite to suffice at a given moment, and whereas the invaders may be stemmed, they are evidently not entirely routed. Instead, they gather their forces, and wave upon wave sweeps over the host, the invaders possibly going into resting or resistant stages, as do malaria plasmodia, until in moments of immune inadvertence, they burst forth anew. This accounts for the periods of secondary latency. Related to the resistance mechanism is the Wassermann reaction, a complement fixation

phenomenon, which albeit not specific, nevertheless must be either an index of immunity, or an index of activity on the part of the spirochetes. At any rate, it is always positive during active syphilis, except in the malignant forms, and under certain other conditions which will be outlined below.

During the secondary stage there is a disseminated, symmetrical, generalized exanthem, capable of assuming many forms; there are general glandular enlargements, mucous lesions of various kinds, headaches showing meningeal involvement, myalgias, neuralgias, arthralgias, and there may even be evidences of involvement of the abdominal and thoracic viscera, and organic changes in the brain, cord, and organs of special sense, particularly the eyes and ears. Fever sometimes occurs. In short, at this period the disease is a generalized infection, varying in intensity according to the virulence of the germ, or the resistance of the host. Should the former be excessive, or the latter too slight, malignant syphilis supervenes, a form in which very early tertianism with rapid death results.

The Tertiary Period. Normally, however, no such dramatic and varied picture occurs. On the contrary, superficially regarded, the disease seems to be mild, and therein resides its insidious peril. Gradually, the host becomes habituated to the prolonged task of resistance, and the virulence of the infection wanes. The transformation from the relatively stormy to the comparatively calm tertiary period has gradually taken place, and finally this period, apparently more innocuous, but actually quite as dangerous, has set in. The lesions are now no longer widely disseminated, but restricted to one or a very few organs or tissues, and recur and wane persistently at given sites. Thus, the skin or bones or heart or vessels or central nervous system, for example, may be the scene of recurrent activity, subject either to tissue affinities of different strains of spirochetes, or to susceptibility on the part of different tissues, or both. The determining factors vary with race, occupation and habits of living. The Chinese, whose mentality is in abeyance, rarely, it is said, get nerve syphilis as compared with Caucasians. Alcoholics are subject to nerve and cardiovascular involvement. Heavy indulgence in tobacco, and severe physical work also seem to favor syphilitic susceptibility of the circulatory system.

The Disease as a Whole. Ultimately, and in spite of everything, a certain number of syphilitics seem to recover completely even without treatment, but this is rare, and negated by the studies of Warthin, who proved that even in clinically apparently cured

cases, spirochetes could be found in the heart without any inflammatory reaction, dwelling there symbiotically and perhaps capable of reactivation to pathogenic potency.

Syphilis thus, throughout its course, is remarkable for its remissions in activity. In other words, the host gradually, as time goes on, tends effectively to limit the virulence of the invading agent. So far as complete elimination of the invader is concerned, this is probably never accomplished. Whether or not the disease assumes serious forms will depend upon the extent to which have been permanently damaged the tissues subjected to the concentrated activities of the spirochetes. This in turn will depend upon tissue peculiarities, or upon peculiarities of the spirochetes themselves. Without doubt the disease must be controlled by the host's ability to generate antibodies sufficiently early to prevent serious inroads upon important structures. The precise mechanism, and the exact nature of the protecting bodies is not known, but roughly the Wassermann reaction seems to be connected with the process. The test is practically always positive during periods of activity.

The Wassermann Test. Without exhaustively entering into the matter, the fixation test is found negative during the first incubation period and in the early days of the chancre, growing more likely to be positive as the secondary period approaches. During activity in the secondary stage, the test is always positive, and it may be so during intervals of latency in this period, but may equally often be negative. The same is not true in the tertiary stage, for here at times it is negative in the presence of clinical activity. When it is negative at such times of obvious activity, the discrepancy is possibly due to a failure in the protective mechanism of the host. This is still speculative, however, for it is not thought that the substances giving the reaction are identical with the immunizing bodies. Nevertheless, there seems to be some parallel between the two. Malignant syphilis, a form running a rapid course, and soon acquiring tertian features, often presents a negative serum test. Since malignancy, or better precocity, depends upon a failure in protection, there is reason to believe that the fixation phenomenon is closely related to immunity.

The test often becomes transiently negative, as shown above, before the patient can possibly be cured. Thus, it requires great experience with the subject properly to interpret the negative reaction. A positive reaction in syphilis invariably indicates activity, but the phenomenon is found also in yaws and nodular lepra, as well as in

some other conditions. Therefore, knowing the rules and exceptions as to the positive test, it is easy enough to interpret it. Not so with the negative! It indicates latency, an inadequate protective mechanism, or cure, according to the stage and character of the disease, and the quality and persistence of the treatment. Since the matter included in the previous sentence is worthy of a treatise in itself, the implied hints will urge conscientious and intelligent practitioners to grasp the truth that there is something to syphilis diagnosis and prognosis besides the serum test; that a positive test does not always indicate syphilis; and that a negative one requires study before it can be applied in practical syphilology.

Treated Syphilis. The foregoing paragraphs outline untreated syphilis. When treated, the course is greatly modified. If treatment is begun in the primary stage, the disease may be aborted, and none of its later manifestations develop. In like manner, progress of the disease, in favorable cases, may be cut short at any of the later stages. In general, the earlier treatment is instituted, and the more intensively it is pursued, the greater the probability of eliminating pictures which formerly were common. Thus, in civilized parts of the world, the late cutaneous manifestations are becoming scarcer, and it is to be confidently expected that modern therapy will completely draw the fangs of the disease. There will then no longer be those remote results of the malady once called para and metasyphilis, but which to-day we know to be syphilis itself, and due just as much to the spirochete as is the chancre. In fact, all syphilitic lesions in all tissues are generically alike in minute structure, the variations being only those determined by the normal anatomical peculiarities of the various tissues themselves. Nor is there any real difference between a secondary papule and a tertiary gumma microscopically, except in degree.

To understand all these facts is to understand syphilis, and if they are grasped, the ensuing chapters will not be difficult. Neither will it be difficult to analyze the objects of therapy. The treatment of syphilis has, for its aim, to destroy the spirochetes before they are inaccessibly intrenched in vital organs, to aid the host when his immunizing resources are inadequate, and as rapidly as possible to limit the process so that the patient may be cured, if not academically, at least to such a degree that in the normal span of human life the disease can never become a menace.

CHAPTER XLI

ACQUIRED SYPHILIS. PRIMARY STAGE

Synonyms. Chancre, Hard Chancre, Hunterian Chancre, Initial or Primary Sore or Lesion, Sclerosis; Fr. Chancre, Sclerose, Accident Primitif; Ger. Schanker, Initial Affekt, Primär Affect, Induration, Sklerose.

The incubation period of syphilis may be ten days or three months, but its fairly uniform time is three weeks. At the expiration of this period the first clinically visible evidence of the disease appears. This is the primary lesion and its site is most often on or near the genital organs. Less frequently it appears elsewhere on the body.

Symptoms. The chancre is a granuloma which may or may not break down. In the former event it is a papule, nodule or tumor, according to its size: in the latter it is an erosion or ulcer. The combinations of size or type of necrosis furnish the possibility of a great variety of clinical pictures. Although the chancre tends to be solitary it is by no means rarely multiple. Multiplicity depends upon either inoculation of multiple injuries, usually herpes progenitalis, or upon infections of contiguous surfaces, the one from the other, as the glans and prepuce. Thus this sort of initial lesion is in a relatively circumscribed area. Very rarely initial lesions may be multiple and widely separated as on the genitals and lips.

When the lesion first appears it looks like a small papule and it may never exceed this size, or it is a nodule or, as has been said, a tumor. The unbroken sores are usually stony hard and dry. The erosions and ulcers are usually found in a hard inflammatory infiltration of variable size. The ulcerative forms are usually saucer shaped with an indurated shallow margin, rarely undermined. These necrotic lesions secrete a serosanguinous, at times purulent and fetid, substance which is highly infectious. The secretion forms a crust the removal of which leaves a slightly bleeding surface.

The primary lesion is coppery, or dusky red or purple, very hard to the touch, in fact usually cartilaginous or stony. But it must be remembered that all of these characteristics may fail and a certain diagnosis be impossible by the unaided eye. The lymph vessels

leading from the chancre to the regional lymph nodes are very often palpable. On the penis the dorsal lymph vessels are easily felt as hard cordlike structures leading to the pubic symphysis. Within five days after the chancre's appearance, and progressively increasing thereafter up to the eruptive stage the local glands enlarge. They are hard, but elastic rather than stony, and give the impression of a chain of beads. They are usually painless, and rarely even slightly tender. The glands immediately draining the infection are earlier involved than those more remote, and the first gland to drain the lesion is the largest of the affected group. This is called the pilot gland by some, and the fact is noted to acquaint the reader with an inapt term which, however, has great vogue.

Course. The chancre ordinarily begins to heal of itself, even when untreated, in from three to six weeks, or at the approach of the period of dissemination, known as the secondary stage. Usually no subjective symptoms accompany the primary lesion, save when associated with the secondary period. The site of the healed chancre is permanently marked by a scar, at first pink and later brown or white. The scar is soft, pliable and covered with a finely wrinkled integument or mucosa, more or less depressed according to the volume of the original infiltrate. When stretched the scar is glossy. Occasionally chancres are phagedenic.

Varieties. The varieties of chancres depend upon various circumstances. As already stated they may be multiple or single, and practically any of the following may in turn enter in the foregoing group. In addition there are the ulcer (Fig. 80), papule, erosion (Fig. 82), plaque and indurated edema. The ulcer or Hunterian chancre (Fig. 80) is a saucer shaped lesion with a slightly granular surface. It is pink or more commonly copper, hard and has a shallow slightly raised hard margin which is rarely undermined. It may reach a diameter of three fourths of an inch. The papule, or if larger, the nodule, is stony hard, coppery and dry. The erosion is a papule with a superficial ulcer which has a serous secretion that forms crusts. The plaque is a thin discoid coppery patch. It is rare and feels like cardboard. The indurative edema is usually on the vulva. The entire involved lip is swollen, hard, and coppery or purplish in hue. Indurative edema is sometimes seen on the prepuce independent of, or associated with other forms of chancres.

Chancres are usually on the genitalia, but often elsewhere. The latter are qualified as extragenital (Fig. 81). On the penis the com-

monest site is the corona, then the prepuce, glans, meatus, shaft and finally the dorsum of the root. In the female the commonest site is the vulva (Fig. 83), at the fourchette, on the lips or near the meatus, and the cervex. At times instead of a visible initial lesion, a general primary syphilitic vulvitis exists. Because so many initial lesions are cervical this stage of the disease is often missed in women, a fact which increases the danger of transmission in coitus, and which exposes unwary physicians and nurses to infection in their work. Extragenital chancres, which also assume any of the forms mentioned, occur most frequently on the lips, tongue, fingers, tonsils, near the genitalia, and finally anywhere else on the body.

We must become familiar with the terms reinfection, superinfection and chancre redux. The reinfection is a primary lesion in a previously cured syphilitic suffering a subsequent inoculation. A superinfection is an initial lesion in a syphilitic whose first infection has so nearly run its course that he is already susceptible to a new infection. The chancre redux is a chancre that periodically returns at the same site without new exposure. It is difficult to say whether this may not be a gumma, save for the fact that motile spirochetes may be demonstrated in it, as well as in the reinfection, superinfection, and ordinary primary lesion. The three types mentioned are rare and the student need concern himself with them no further than to know the terms and their significance.

At times syphilis begins without any demonstrable initial sore. This may be due to the fact that there is none (syphilis d'emblée) or to the fact that the lesion is so situated as to elude the patient or physician. The former circumstance is very rare. The latter group comprises urethral chancres in the male, cervical chancres in the female, rectal chancres, and chancres so deep in the pharynx or larynx, that their presence is unsuspected. Only proper examination with suitable specula or other appliances, reveals their presence. But a word must be said as to the masculine urethral chancre. A serous urethral discharge appears containing no gonococci. Such a discharge should be examined for spirochetes. On palpating along the urethra's course a lump is felt. Endoscopy shows this lump. If it is large enough, symptoms of stricture are present.

Phagedenism in chancres is uncommon, but at times the hard and soft chancres are associated. This is the *chancre mixte,* and usually the symptoms of the soft chancre with large inguinal buboes dominate the picture. The soft chancre is not indurated, is irregular in

outline and has undermined edges and a purulent margin and base. At times pure initial lesions are small, nondescript and soft and their recognition by the eye alone is impossible.

Diagnosis. Aside from the clinical features of the initial sore the diagnosis depends upon a demonstration of the treponemata. This is best done by means of the dark field. Before attempting this procedure a wet dressing of normal saline should be applied for twenty-four hours or longer, partly to soften the crusts and partly to eliminate destruction of the spirochetes by antiseptics used by patients before they decide to consult a physician. In non-ulcerous lesions this is less necessary. The chancre is scratched lightly by a sharp implement, and the serum which oozes forth contains the spirochetes. Often examinations on successive days are required before the organism is found. No matter how distinctive the clinical aspect of a suspected sore may be, microscopic corroboration is urged. Negative findings are only of relative value, as the patient may present himself so late that the lesion is beginning to involute and contain only few if any spirochetes. Lip and mouth chancres contain spirochaeta microdentia as well as pallida, and care must be exerted not to confuse these two, as the former is not pathogenic.

In early chancres there is no Wassermann reaction, but as the secondary period approaches the serum change appears. Thus the incidence of the reaction in primary syphilis is in an ascending curve, starting from zero at the appearane of the lesion and reaching 100% with the onset of the secondary stage. The curve is gradual up to the tenth day of the second incubation period and steep from thence. Obviously the Wassermann test is of no value in primary syphilis unless positive, in which case it heralds approaching dissemination of the disease.

All primary lesions anywhere may clinically resemble certain secondary lesions, gummas, ulcerative tuberculosis, blastomycosis, sporotrichosis and epithelioma. Further, at certain sites chancres may suggest or be inoculated upon non-specific lesions characteristic of these sites.

The secondary lesions simulated by the primary are moist papules (Chapter XLII) whether single or grouped. The latter are not indurated but contain spirochetes and show adenopathies. The glands however are generally swollen in this stage, and the history excludes error save where no chancre has ever been present. The only practical issue is one of prognosis, for, as will be seen, the earlier treatment is begun after the onset, the better the outlook for

FIG. 80. HUNTERIAN CHANCRE

The margin is slightly rolled, the surface of the lesion is glistening, red, and suggests granulation tissue. A soft chancre would show undermined edges and a more irregular margin, but very frequently the hard simulates the soft chancre. Frequently, too, hard chancres are multiple.

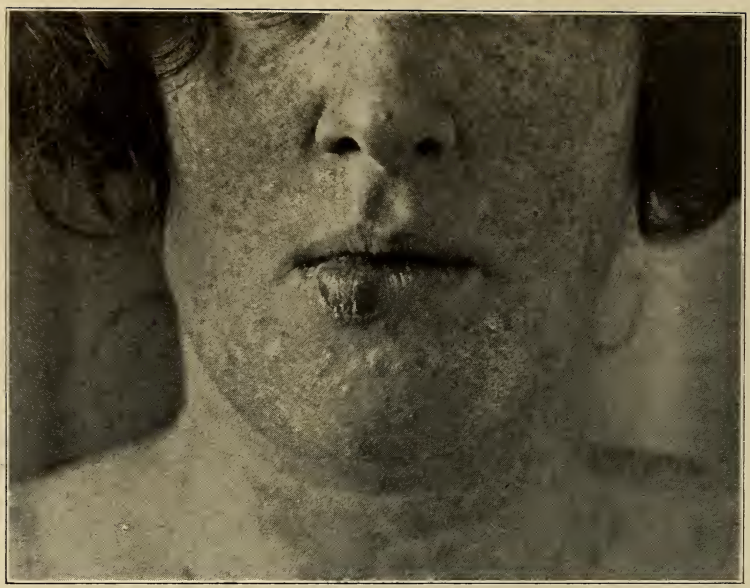

FIG. 81. EXTRAGENITAL CHANCRE

This picture shows the nodular, non-ulcerating initial lesion associated with secondary syphiloderms. On the chest is a macular rash or roseola; on the chin, scaling papules.

FIG. 82. CHANCRE (MEATUS)

A shallow, hard ulceration appears at the penile orifice. The swelling in the right groin indicates the enlargement of the inguinal glands. A maculo-papular rash appears on the belly and thighs.

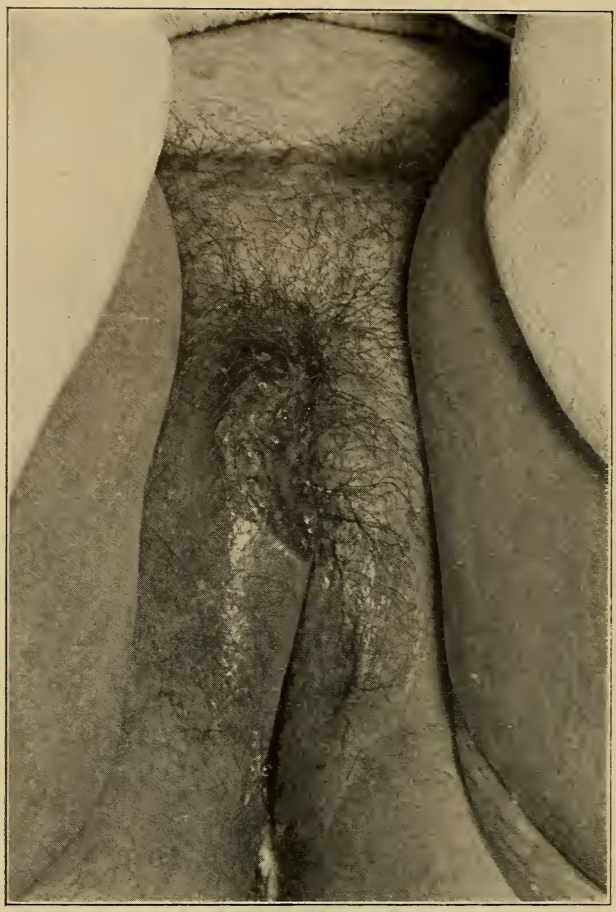

FIG. 83. CHANCRE (VULVA)

A large ulcer is seen on the right larger labium. The margin is elevated and there is a large amount of edema of the labium.

aborting the malady. Gummas often resemble chancres, but spirochetes are absent and there is no regional glandular enlargement. Tuberculosis, blastomycosis and sporotrichosis are differentiated by the absence of spirochetes, by demonstrating the respective organisms, and possibly by the histology of the lesions.

Epitheliomas are recognized by the age at which they occur, although primary lesions often appear at the so-called cancer age: by the absence of spirochetes and histologically (see Chapter XXVIII, Diagnosis of Epitheliomas). In this connection it may be well to add that when clinical and laboratory procedures fail us the only method remaining to establish the diagnosis of syphilis is to await the arrival of the secondary manifestations, a temporizing and dangerous delay, fortunately not often necessary, but which is better than erroneously stigmatizing a human being as syphilitic.

A furuncle of the penis and lip may either resemble or become the site of a chancre. The diagnosis depends upon the clinical and laboratory data already so often adverted to. The same applies to herpes of these sites. Soft chancres and balanitis errosiva et gangrenosa may possibly cause confusion (Chapter XXIII). Lingual chancres, epithelioma and tuberculosis of the tongue are differentiated clinically, bacteriologically and histologically. The spirochaeta microdentia here can cause great confusion. Chancre of the tonsil may be confused with Vincent's angina. The diagnosis often resolves itself into demonstration of the respective organisms.

Prognosis. Chancres always disappear spontaneously, but much more rapidly (sometimes in ten days) under vigorous general treatment. The prognosis in a larger sense is that of syphilis.

CHAPTER XLII

ACQUIRED SYPHILIS. THE SECONDARY STAGE OR PERIOD OF DISSEMINATION AND FLORITION

At about the time that the chancre is almost or completely involuted, or shortly thereafter, clinical evidence asserts itself of the general invasion of the host by the parasite. This evidence consists of symptoms and signs. The secondary stage may be defined as that phase in the development of syphilis in which clinical phenomena appear, dependent upon invasion of all organs by the causative agent.

Symptoms. There may be no subjective phenomena. More commonly there are severe nocturnal general headaches and variable degrees of sore throat, arthralgia, myalgia, and ostalgia, with increased severity of the pain at night. The arthralgia in its intensity may simulate acute multiarticular rheumatism, and the ostalgia osteomyelitis, but swelling and redness of the joints are lacking. Rarely there is low fever, and more rarely still pyrexia with morning remissions and a variable degree of prostration. There may also at times be icterus or jaundice but without the symptoms of cholelithiasis, cholecystitis or gastroduodenitis. Infrequently albuminuria, and, more infrequently still, acute nephritis even of the hemorrhagic type is observed.

Signs. The signs of secondary syphilis are cutaneous, mucous, glandular, visceral, skeletal, circulatory, central or peripheral nervous and humoral. By far the most important are the first three. Like so many infectious diseases the eruptive manifestations of syphilis are exanthematous and enanthematous. To understand the main feature of the secondary rash it is necessary to remember that since this stage is that of dessemination the eruption is both general and symmetrical. Furthermore, it consists of macules, papules and ulcers, or some combination of them, and no part of the body is without them. The sub types of the lesions are numerous and this, together with the combinations they fall into, creates a varied picture. The sites of the eruption are the chest, abdomen, back, face, palms, soles, neck, and flexor surfaces of the extremities rather than the extensor, but by no means exclusively so. There are also pigmentary disturbances and alterations in the hair and nails. The

eruption rarely itches save in negroes, and even among them pruritis is uncommon. In body folds the lesions tend to vegetate.

The mucous lesions (Fig. 88) are seen in the pharynx, on the vulva, in the vagina, on the cervix, the glans, prepuce and at the anus. They are prevailingly superficial, grayish ulcers, and particularly the genital and anal lesions fungate.

The ulcers are called mucous patches, the fungating lesions flat condylomas. Secondary ulcerative proctitis is scarcely rare but is not common. Although not quite in place here, it may be parenthetically stated that in the axillae, under the breasts, the umbilicus and groins and in other folds in the adipose, fungation occurs forming flat condylomas.

The lymph glands all over the body are enlarged. Thus these organs, discrete, somewhat hard, indolent, painless and insensible, are visible or palpable wherever they lie superficially. They are found in the neck, behind the ears, in the armpits, epitrochlear region, groin, and even in the popliteal spaces. They neither break down nor become matted and, precisely like the regional adenopathies satellite to the chancre, they feel like a rosary or string of beads. Their wide dissemination and symmetry reflect that of the rash and are similarly explicable. Fluid aspirated from the glands is often found to contain spirochetes.

The visceral signs are thoracic and abdominal. Endocarditis and myocarditis are rare, and secondary pulmonic changes are not known. The abdominal viscera affected are the liver, spleen and kidneys. With, and sometimes without icterus or jaundice, hepatitis occurs, with enlargement of the liver demonstrable by percussion and palpation. The spleen is frequently enlarged and in febrile cases typhus, typhoid and atypical forms of malaria are simulated. The Widal reaction is absent, blood cultures for the typhoid and typhus bacilli are negative, and plasmodia are not demonstrable. On the other hand, aspirated blood from the spleen contains spirochetes. The signs of syphilitic secondary nephritis are those of any acute or subacute parenchymatous inflammation of the kidney. Whether the alimentary tracts and pancreas may be involved is not yet known.

In syphilitic periostitis or osteitis the bones are tender and the periosteum is roughened. This is particularly the case along the anterior tibial margin, the site of election, but other bones are at times involved, and with fever acute osteomyelitis is simulated. Clinical evidence of peripheral circulatory changes are virtually un-

known although anatomic vascular changes are common in the secondary stage.

Central nerve involvement occurs as mild swelling of the arachnoid causing headache and, in a substantial majority of instances, changes are observed in the cerebrospinal fluid, the Wassermann reaction, pleocytosis and globulin reaction sometimes being present. True meningitis, encephalitis and myelitis are rare indeed. The optic nerve is involved in almost twenty percent. of all cases of secondary syphilis, as ophthalmic examination shows. Early blindness occurs, although rarely, the second nerve either spontaneously or under proper treatment usually showing a perfect recuperative ability. Even more rarely the auditory branch of the eighth nerve is involved, but permanent deafness is most unusual. Bone conductivity is diminished. Seventh nerve palsy too, is rare. The other special nerves are not known to be involved, but mild peripheral neuritis is not entirely uncommon, and this must explain the pain in the extremities, otherwise partly explained by myalgia and arthralgia.

The humoral changes are those already indicated in the lymph glands, spleen and cerebrospinal fluid. More important is the Wassermann reaction in the blood which is present in practically all cases of secondary syphilis. Thus this test is of such great corroborative significance that it may well be regarded as a cardinal sign of syphilis in the invasive stage.

Course. Untreated, the course of secondary syphilis runs from six months to two or three years, spontaneously disappearing as tertianism develops. Rarely the entire disease stops in that stage. Treated cases run atypical courses, depending upon the kind of therapy employed, and the manner of its administration. The untreated secondary stage progresses in augmenting and receding waves of clinical and serological manifestations. At one time the rash has one quality, at another, another. Likewise, the other symptoms and signs vary, depending upon the virulence of the infecting organism, its strain, and the reaction thereto of the host. But the striking feature is remission and recrudescence, the former representing transitory latency, a term more useful than scientific.

Although all the foregoing is absolutely essential to a broad grasp of secondary syphilis, the dermatologist must more minutely master its cutaneous manifestations with especial reference to differential diagnosis. For this reason, under the appended heading of varieties, which applies to the rash on the skin and mucous surfaces, this aspect of the subject will be covered.

VARIETIES OF SECONDARY SYPHILITIC ERUPTIONS

Macular Syphiloderm (Fig. 84). The macular syphiloderm, also called the **syphilitic roseola,** is an eruption consisting of erythematous spots at the sites common to all secondary eruptions. It usually begins on the front or back of the torso as an eruption of delicate rose colored macules about one centimeter in diameter. These are either so pale as to be almost invisible except in side lights, or so dark as to be easily visible; and the lesions may be very few or extremely numerous. The rash lasts from one to five weeks, usually having made its appearance within six weeks to three months after exposure, and within four weeks to two months after the appearance of the chancre, or within four weeks after its disappearance. Occasionally the rash is fugacious and consequently escapes the attention of both physician and patient. It does not itch and so of course there are no scratch marks. It may be associated with other types of secondary lesions but at first is apt to exist alone.

Another form of macular syphilide is the so-called **recurrent roseola** which is a late secondary manifestation, seen as a rule six or eight months after the initial lesion has appeared, and which is the result of inadequate or no treatment. Its characteristic sites are the torso, but it may appear on the extremities, rather favoring the flexor surfaces but not entirely excluding the extensor. These lesions are somewhat larger than those of the early roseola, and inclined to be ring shaped or festooned. Recurrence of this type of roseola may take place at intervals if the disease is unchecked.

There is a form of macular syphiloderm called by Unna **the neurosyphilide** which is extremely rare and which, according to this observer, is analogous to the ring-shaped lepra lesions on an alleged basis of involvement of the cutaneous nerves. It is not purely macular, being somewhat raised and slightly infiltrated, and of a darker, more coppery color than that usually seen in macular syphiloderms. On the other hand, it is by no means so infiltrated as to suggest a papule. All macular syphilides, although prevailingly flat, sometimes give the impression of being raised in the manner of a superficial wheal, when viewed by sidelight. Indeed, even the palpating finger may get a slight impression of elevation.

Macular syphilides sometimes suggest other macular eruptions, notably flat urticarial lesions, the spots of erythema multiforme and toxic erythema, and the rash seen in typhus and typhoid fever, and following the use of certain drugs, notably copaiba. The first three

of this group usually itch; the roseola does not as a rule, though at times it comes as an obstinate urticaria controlled only by anti-specific treatment. The typhus and typhoid rash are accompanied by the characteristic symptoms and serological and bacteriological evidence of these diseases, in contradistinction to the general clinical and serological picture of syphilis. The copaiba rash, associated as it is with gonorrhea, may suggest a syphilitic macular eruption with an intra-urethral chancre. The absence of the Wassermann reaction and general glandular enlargement, the finding of gonococci in the urethral discharge, and the history of drug ingestion, form the only basis of differentiation.

Papular Syphiloderm. Papular lesions are either large or small. The first are called lenticular (Fig. 85) or discoid papules, the second, miliary papules (Fig. 86). The first two are the commonest types of the secondary papular syphilide.

The lenticular papule (Fig. 85) is situated at all the common sites of secondary syphilis. A lenticular papule is from one-half to one and one-half centimeters in diameter. It is shaped like a convex lens; hence its name. It is hard to the touch, pink, buff or coppery in hue, and does not itch or in any other way give subjective symptoms. It is often surmounted by a very superficial, thin, somewhat greasy looking scale, and the lesions may be sparse or very numerous but discrete, or so numerous as to be coalescent or confluent, in which event they form figures of all sorts imparting a variegated appearance to the skin. These lesions may be combined with the early roseola, or with small papules, or both, and they appear at about the same time that the roseola does, and run a similar course. Occasionally it will be found that a lenticular papule of striking dimensions is surrounded by small lenticular papules or miliary papules or both, as the sun is surrounded by its planetary system. These constellations give the picture of the **corymbose** or **corymbiform** syphilide.

Eruptions consisting mainly of lenticular papules simulate papular seborrhea, pityriasis rosea, and psoriasis guttata. The characteristic features of secondary syphilis form the basis of differentiation. Objectively they differ from seborrhea in site and in the character of the scaling and infiltration. The site of seborrhea is in the body folds, over the sternum, between the shoulder blades, and on the scalp. The scales are heavy and greasy, the color is buff. Papular syphilides in a seborrheal patient, however, may assume many of the characteristics of seborrhea, and in these cases, without consid-

ering the general features of syphilis, a differentiation would be hard indeed.

Pityriasis rosea is usually limited to the area between the collar-bones and upper third of the thighs, and is preceded by a characteristic herald spot. Moreover this disease is more or less pruritic. The lesions of psoriasis may be numerous and as infiltrated as those of papular syphilis, but the scales are a shimmering white, the localization on the whole favors the extensor surfaces, and there are no general glandular enlargements or any of the other signs of syphilis. Excepting in acute cases of psoriasis the long history of the disease is of diagnostic value.

In this connection, some emphasis must be laid upon the fact that secondary syphilides have certain characteristic features in certain localities. This is particularly true of the lenticular papular lesion. When the face is involved, although no part is free, the lesions tend to group themselves in a band just across the forehead immediately below the hair line, roughly simulating a crown or diadem. This is called the **corona Veneris.** Dr. Trimble of New York has called attention to the frequency with which lesions on the chin give an impression of mottling. On the palms and soles the papules appear as macules because the epidermis is so thick that the infiltrate is rarely able to develop the elevation seen elsewhere.

The palmar syphilide (Fig. 90) is of great diagnostic importance for none of the lesions simulating this stage of syphilis occur on the palm excepting those of psoriasis, and this so rarely as to be negligible.

The discoid syphilide, better known as the **flat condyloma** or **moist papule** (Fig. 89), has already been mentioned earlier in this chapter. This occurs in all body folds and on the mucous surfaces of the genitalia and anus as fleshy, elevated, gray, discrete lesions, or highly confluent masses which easily bleed, give off a foul secretion, and which are highly infectious. They rarely exist alone, but usually in conjunction with either the macular or papular syphilide, but may come and go throughout the secondary stage. The only condition they simulate is pemphigus vegetans, the lesions of which occur at the same sites and which have the same foul odor. In pemphigus vegetans there is a bullous eruption on the skin, there are no general glandular enlargements, and the Wassermann reaction is absent. In flat condylomata there are no bullae on the skin, but other forms of syphilides, and enlarged glands, and the Wassermann reaction is present. Isolated, or very few flat condylomata may simulate the solitary or multiple chancre. The condyloma is softer than the

chancre, however, and vegetating rather than ulcerating or papular. Spirochetes are found by the ultra microscope, in flat condylomas.

The miliary or acuminate papule (Fig. 86), sometimes called *lichen syphiliticus,* is a comparatively rare lesion. It more frequently itches than any other syphilide, but even here pruritis is rare excepting in negroes. The entire body is studded with minute coppery or ham-colored discrete or grouped papules, one millimeter or two in diameter, sometimes slightly scaling and closely resembling lichen planus or lichen scrofulosorum. They too may be corymbiform in arrangement. Lichen scrofulosorum, however, occurs mostly in the young and is a tuberculous process. Lichen planus itches intensely. In both of these conditions the positive evidences of syphilis, so often emphasized, are absent.

The characteristic features, then, of the papular secondary syphilide are the wide dissemination and symmetry; the infiltration; the constant association with evidences of a disseminated and universal infection; the ability shown to mimic other diseases, which on the whole, however, can be easily differentiated; and the fact that the different types of papules may occur simultaneously and may also be associated with the macular eruption. When this occurs, and the occurrence is very common, the eruption is described as *maculopapular.*

The annular syphilide. At times, and particularly in negroes (Fig. 87), although occasionally in whites, papules arrange themselves in rings on the face, neck, and less commonly elsewhere on the body. These rings may be few in number or numerous and their segments describe arcs of various degrees so that small circles the size of a dime may be seen, or segments which if completed would represent circles ten inches in diameter. At times the circles are concentric. For the most part the papules composing these figures are closely grouped and miliary. It is possible for these lesions to simulate ringworm, but the diagnosis can easily be cleared by a search for fungi versus a search for other evidences of syphilis, and the performance of the Wassermann test.

In discussing seborrhea in a preceding paragraph it was stated that syphilides in seborrheal patients often assume many of the features of the milder disease. This is partially so in connection with lesions appearing in the nasolabial folds. A delicate annular or crescentic arrangement of pink, greasy, scaling papules corresponding to the curve of the nostril is not uncommonly observed, and if it were not for the marked infiltration and the peculiar coppery

hue, familiar to the experienced eye, the condition might easily pass as seborrhea. Patients presenting this picture should be carefully studied from the standpoint of syphilis, a point which is particularly emphasized by *Jadassohn*. Usually the Wassermann reaction will be positive in such people.

The Pustular Syphilides are comparatively rare and are divided into three groups: the acuminate, the obtuse, and the ecthymatous. Before describing them, it might clarify the subject to mention a few of the general characteristics of early, pustular, syphilitic lesions. Actually the syphilitic lesion, however small, is always granulomatous. Sometimes necrosis occurs even in the early stage, and, according to whether this process is chiefly serous or serous with a high content of leucocytes, the lesions become vesicular or pustular. In the latter event, different types of pyogenic lesions are simulated, albeit the contents of an unruptured pustular syphilide are sterile. The ruptured ones become secondarily infected with the ordinary organisms of the skin, but these organisms play no integral part in the causation of the pustules. Actually, then, the vesicular and pustular syphilides may be considered together, the process being one of fluidification of the lesion, the type of fluid depending upon whether there are more or less of the infiltrating leucocytes washed off into the fluid. The pustular lesions may be small, large or ulcerative.

The small lesions correspond to those which were designated acuminate in the preceding paragraph. They are very rare. They develop as a softening stage of the miliary papule otherwise known as lichen syphiliticus, and constitute the picture called acne syphilitica.

Acne syphilitica is seen for the most part in negroes. It occurs wherever lichen syphiliticus may. The lesions are hard and have a papular base surmounted by a white point such as one observes in acne, and if analyzed two elements can be observed, a papular base and a vesicular or pustular apex. In the former event the apex is translucent, yellowish and glistening; in the latter it is opaque and white or cream colored. In either case the apex is minute. The lesions are discrete or coalescent and heal, as a rule, without scar formation, though slight superficial scars may occur. As a rule this type of lesion exists alone, but it may be intermingled with miliary papules particularly, and with any other type of secondary syphilide in any combination. There is no itching, excepting in negroes, and hence in Caucasians there are no scratch marks. The

The other symptoms and signs of secondary syphilis are present and this form of syphilide is peculiarly slow in responding to treatment.

The varicelliform syphilide. One form of the vesicular stage of this condition sometimes resembles varicella and is known as the varicelliform syphilide. It differs from varicella, however, in its slow evolution, in the great number of lesions, much less marked tendency to umbilication and in its association with all the other evidences of early syphilis. Vesicular syphilis may conceivably be confused with universal acute dermatitis. The lack of itching, however, and the marked difference in the symptoms and signs of the two diseases render it only academically necessary to mention the fact.

The pustular phases resemble variola, ordinary acne, suppurative folliculitis and the pustular bromodermas and iododermas. Variola may be ruled out by the fact that the syphiloderms are smaller, the general symptoms not severe, and by the other methods that would be employed in an effort to recognize syphilis. Acne is excluded by the fact that pustular syphilides are distributed all over the body and that there are no comedones, whereas simple acne is limited to the face, chest and interscapular region. Inasmuch, however, as acne may be present in a syphilitic, it is not always easy to determine which disease is represented by a given lesion, nor is it very important to do so, because of the mildness of the ordinary acne. The bromide and iodide eruptions may closely simulate pustular syphilides, and so far as the iodide eruption is concerned the matter is of some importance inasmuch as the iodides are so frequently used in treating syphilis.

The obtuse pustular syphilide is to the large lenticular papule what the acuminate pustular syphilide is to the miliary papule. In other words, the liquification takes place over a somewhat larger surface than in the smaller form and, as in the latter, there is a serous and a purulent phase. The significance of these phases, however, is identical with those in the smaller form. The lesions when analyzed consist of an infiltrative base surmounted by a sac containing serum or pus. The lesion most simulated is the ecthymatous bleb. This form, too, is uncommon, but is not exactly rare. The lesions are seen wherever ordinary papules would appear and variola, ecthyma and the iodide and bromide eruptions are simulated. The differentiation among these conditions is made along the lines indicated above.

Whenever any vesicular or pustular syphiloderm dries a crust is

formed precisely as when any other skin lesion, consisting largely of fluid, dries. Usually the crusts fall off and the integument returns to normal, particularly when treatment has been started early enough.

Sometimes, however, ulceration takes place beneath the crusts, furnishing the picture of **ecthymatous syphilis,** and we find admixed with other syphilitic lesions superficial ulcers varying in size from a millet seed to a dime or perhaps a trifle larger, the bases of which are infiltrated, the infiltration being cupped out by a shallow concavity that is pink, glistening and moist when the crust has been removed. Ecthyma and impetigo contagiosa are thus very closely mimicked, but in these two diseases the infiltration is less marked than in syphilis and the other manifestations of syphilis are wanting. Sometimes, before crusting takes place a veritable bulla forms. In this event pemphigus vulgaris is remotely simulated, but in pemphigus there is no infiltration and the classical concomitant signs of syphilis are absent.

Malignant Syphilis. At times, early syphilis runs a very rapid course and almost before the usual evidences of the secondary syphilitic stage are evolved the condition approximates tertianism. In these cases, fortunately rare because they are justifiably called malignant syphilis, the patient has a widely disseminated pustuloecthymatous eruption, the main features of which are irregular ulcers of various sizes resembling tertiary ulcers and rupial syphilides, which will be described in the next chapter. Malignant syphilis nowadays is getting even less common than formerly because of our ability to control the disease early by adequate treatment. Curiously enough, in malignant syphilis the Wassermann reaction is often absent. Nothing else, excepting perhaps pemphigus, resembles this form, and in pemphigus there is very much less infiltration than one finds in the lesions of syphilis. Without adequate treatment, and in patients who are intolerant of arsphenamin or of mercury or of both, malignant syphilis often ends fatally, the entire course consuming six months or a year. Death is due to cachexia.

Pigmentary Syphilides. Only one more type of early syphilis remains to be described, namely the pigmentary syphilides. With one exception these syphilides are unimportant. This exception is known as the **leucoderma syphilitica.** It is usually situated on the nape of the neck, extending thence down over the shoulder and sometimes even over the breast, or upward over the cheeks and temples, or forward over the throat. It is exceptional to find it in men, and in women it is restricted largely to brunettes. The char-

acteristic feature of these lesions is that they impart a mottled appearance of white macules lying on an otherwise hyperpigmented integument. The hyperpigmentation may merely consist of a slightly increased buff or brown hue exaggerated by the whiteness of the spots within, or the hue may be somewhat like that of milk chocolate. Any shade may exist between the two extremes. This type of lesion appears from four to six months after the original infection and persists for years after all other evidences of the disease have vanished. It may be permanent. As a rule, however, particularly shortly after its appearance, other evidences of syphilis are noted. It is not safe to be arbitrary in diagnosticating the condition, for the curious mottling which characterizes it is sometimes seen in summer in sun tanned women who have worn lace collars or yokes, but who have not syphilis, the configuration being due to the design of the lace. In the majority of instances in which the pigmentary syphilides are found the Wassermann reaction is present. It is possible, too, to confuse the pigmentary syphilide with vitiligo (Chapter XVIII), or with pigmentary lepra (Chapter XXV). In vitiligo the white blotches are enormous, irregular, symmetrical and distributed, in extreme cases, all over the body. In lepra there are other evidences of this disease. It is theoretically possible to confuse the pigmentary syphilide with nevus anemicus and white-spot disease, or with any of the numerous pigmentary atrophies. Though it is scarcely worth while to discuss these matters in great detail, the syphilitic leucoderma represents a disturbance in the pigment forming mechanism, analogous to what occurs in vitiligo, namely an ability on the part of the tissue to produce pigment in certain spots with a real or fancied accumulation of pigment at their periphery. Whether this disturbance is local, or whether the chromaffin system is affected is not known. Treatment does not affect the lesions for, as has been stated, sometimes they are persistent, and at best it takes a number of years for them to disappear.

Regional Peculiarities. A description of secondary syphilides would not be complete without some emphasis being laid on their regional peculiarities. The hair and nails are often affected. When macules or papules involve the scalp the hair often falls out at the site of the lesion and thus small bald patches develop, irregular in size, and imparting a mangy or motheaten appearance to the scalp as a whole. These patches are not as circumscribed as in alopecia areata (Chapter XXXIV) nor do they contain fungi as in ring-

worm of the scalp (Chapter XXII). The nails are either ridged, opaque or brittle and suggest the condition found in ringworm or favus (Chapter XXII), or in psoriasis (Chapter XI), or dermatitis involving the nail bed (Chapter IX). Other evidences of these various diseases being absent syphilis may readily be recognized when concomitant signs of the latter are present. But there are vague nail dystrophies which cannot easily be differentiated from nail syphilis.

The face may be invaded by the macular, papular, serborrheal, psoriasiform, vesicular, pustular and ulcerative syphilides. The most common of these are the simple macular or maculopapular. The former causes a peculiar mottling of the chin, the latter, among other things, tends to form as a band at the hairline of the forehead giving the picture of the syphilitic crown known as the *corona Veneris*. As already mentioned the seborrheal forms favor the nasolabial folds. The torso and extremities may be invaded by any type of syphilide, the most common, of course, being the macular and maculopapular. The palms and soles are peculiarly liable to exhibit syphilitic lesions and these are usually macular or papular and often cause marked scaling, so that sometimes either the picture of palmar and plantar psoriasis, or palmar and plantar scaling dermatitis are simulated. Palmar and plantar psoriasis, however, are so rare that for practical purposes they may be considered as almost non-existent. Scaling dermatitis of these regions is not rare but usually itches intensely, while syphilis does not. Between the toes, particularly in people who perspire freely, maceration of the lesions often leads to their fungation and to the forming of flat condylomas. The various mucous lesions and lesions of mucocutaneous junctions have already been amply described, and it is here necessary only to re-emphasize that they should be carefully studied by the syphilographer because they so often give a clue to the diagnosis.

Secondary syphilis, then, represents a systemic infection in which the pathogenic agents are widely disseminated and in which some of the most characteristic signs of the disease are found in the skin, and mucous membranes. Adequately to describe these objective signs would require a volume in itself and only the salient features have here been touched upon. Actually, there is no skin condition, however common or however rare, that cannot be aped by this versatile disease. It is always possible, nevertheless, by considering the general characteristics of the malady, the Wassermann reaction, etc., to reach a diagnosis.

The treatment of secondary syphilis will be discussed in a separate chapter.

Prognosis. The prognosis is good, as a rule, so far as our ability is concerned to cause the lesions to disappear. Whether the patient shall be cured or not depends particularly upon how early treatment is begun and also upon the tolerance of the patient for antisyphilitic drugs, and probably to a large extent upon which one of the numerous strains of treponema pallidum he has been infected with. Undoubtedly, syphilis in the secondary stage can more often be cured than not if we regard the question of cure not as purists but from the standpoint of the practical needs of society at large.

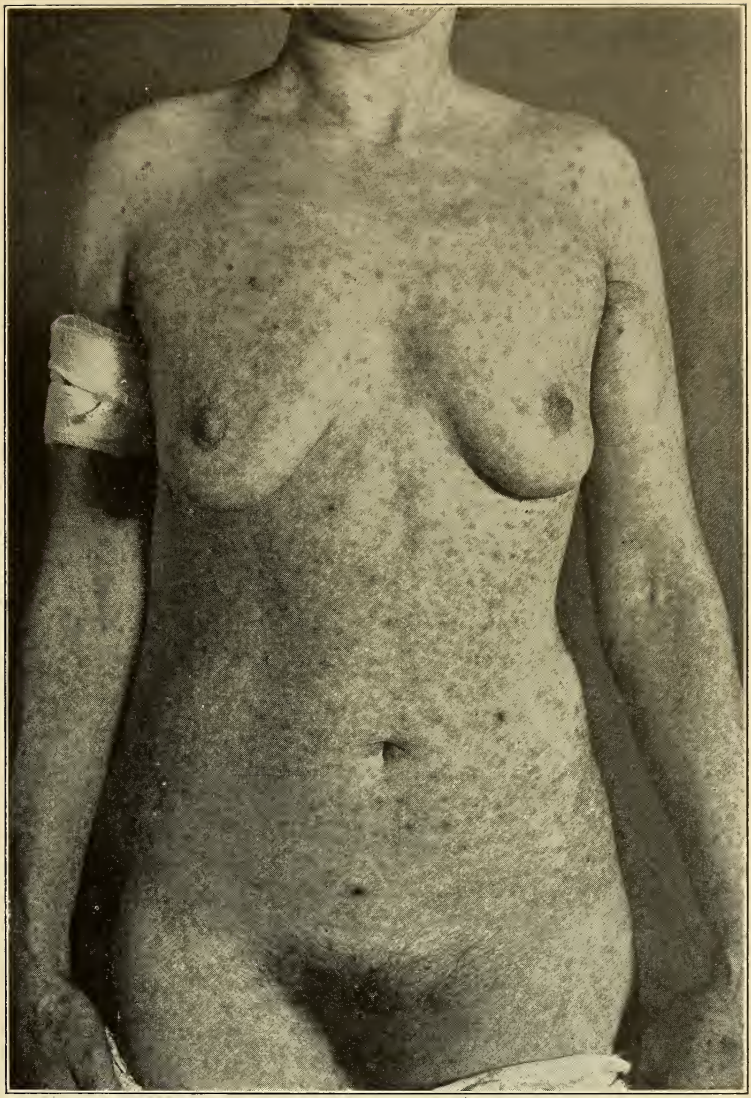

FIG. 84. MACULAR SYPHILODERM

This form is also called the roseola. It consists of more or less numerous, intensely vivid or pale pink, symmetrically distributed macules, a marked example being here shown.

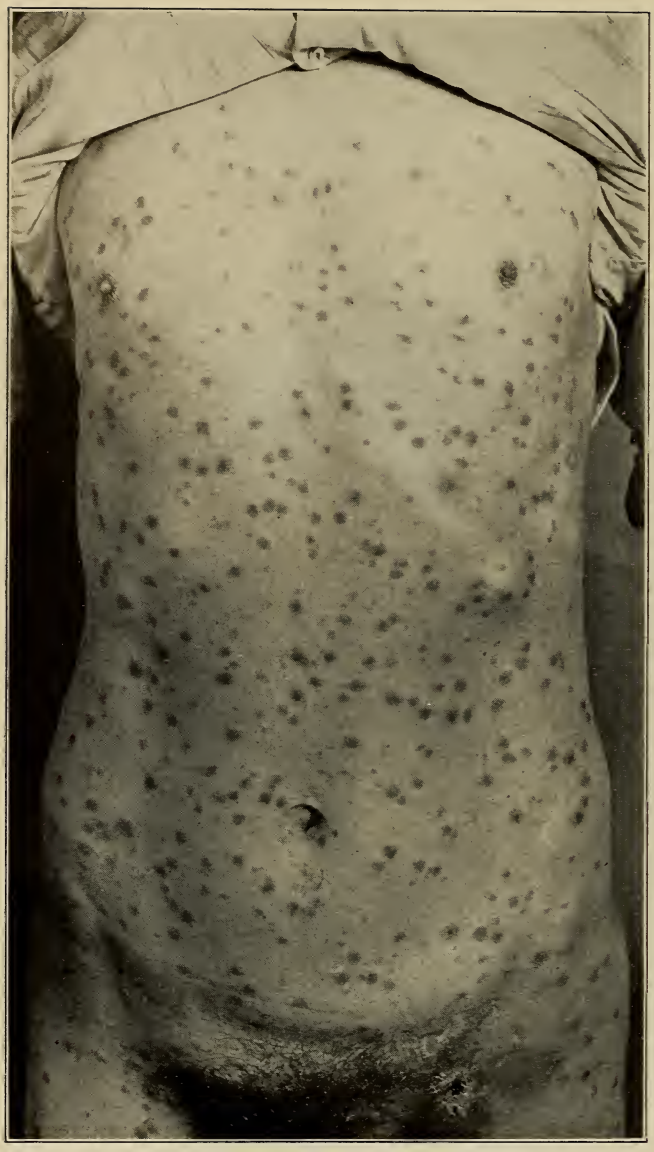

FIG. 85. PAPULAR SYPHILODERM

(Same patient as Fig. 84.)

Note the large papules all over the body, the swollen inguinal glands, the absence of scratch marks and scaling.

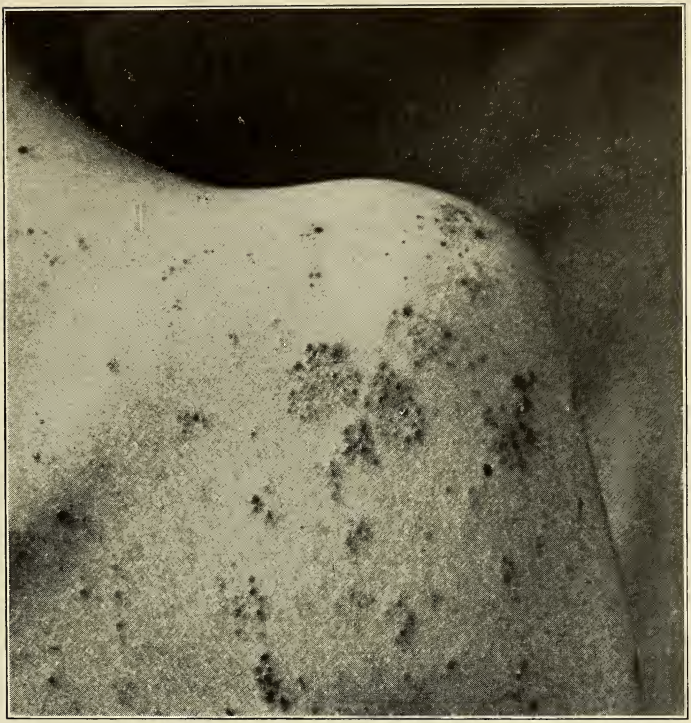

FIG. 86. SMALL PAPULAR SYPHILODERM

Note the wide distribution of the lesions, the minute papules themselves, grouped distinctly.

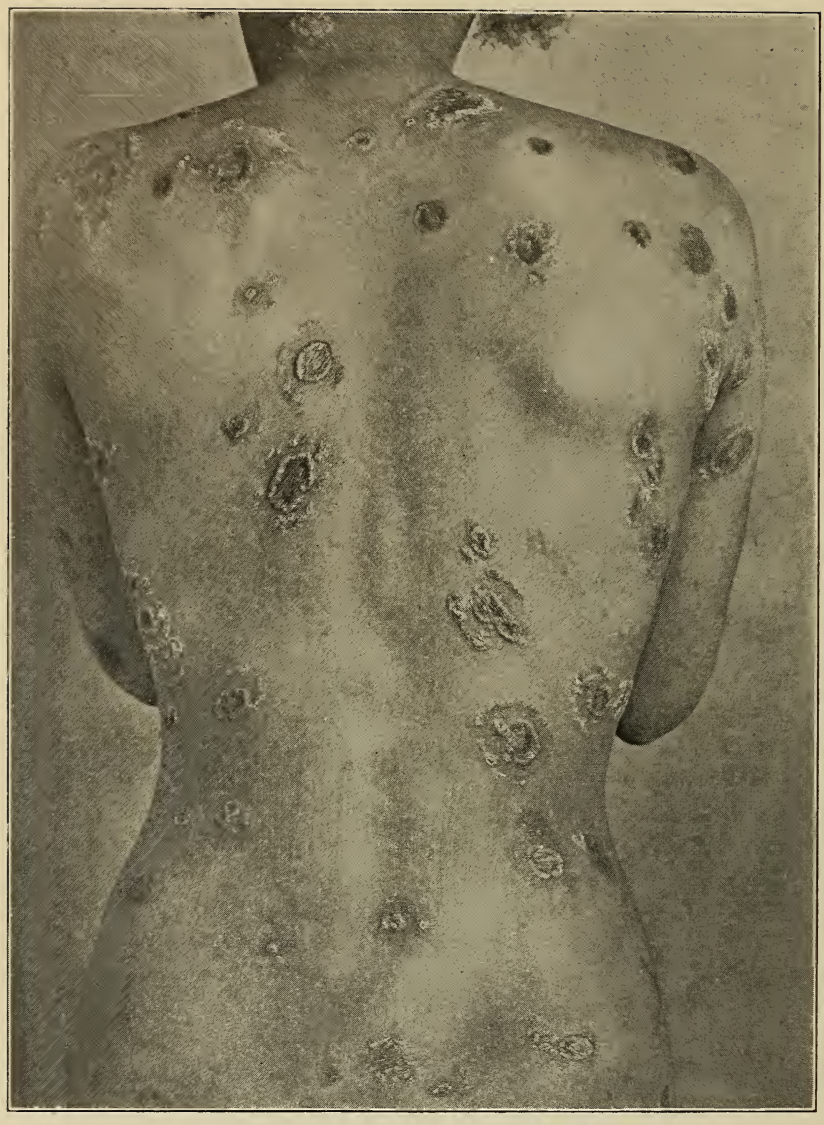

FIG. 87. ANNULAR SYPHILODERM

This unusual type of lesion is less frequently seen in whites than negroes. The rings consist of circles of minute papules. At times, the circles are concentric; at times, segments of circles are seen. This form sometimes itches.

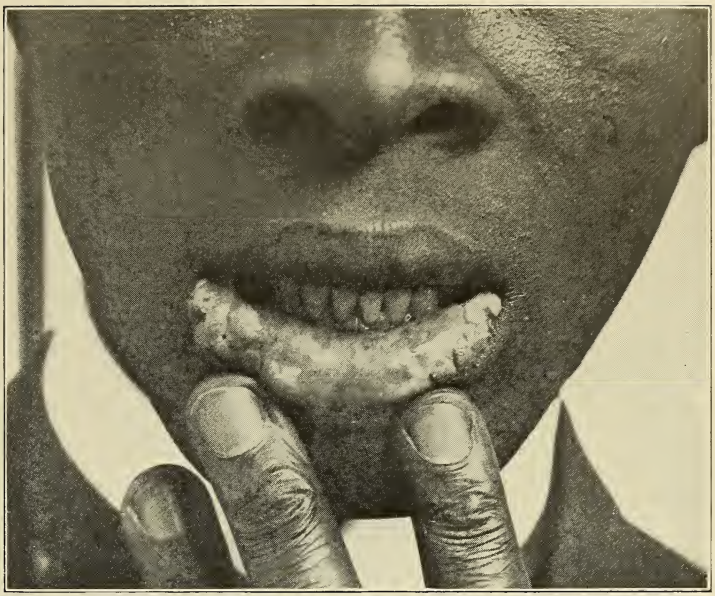

FIG. 88. MUCOUS PATCHES

In the centre there are flat mucous patches, while near the corners of the lips the patches are hypertrophied. A mucous patch is simply a syphilitic papule of the mucosa, its peculiar attributes depending upon maceration incidental to local moisture. When hypertrophic, the lesions are flat condylomata. They are gray or white ulcers or elevations, and those on the genitalia and in the body folds are fetid. All are highly infectious.

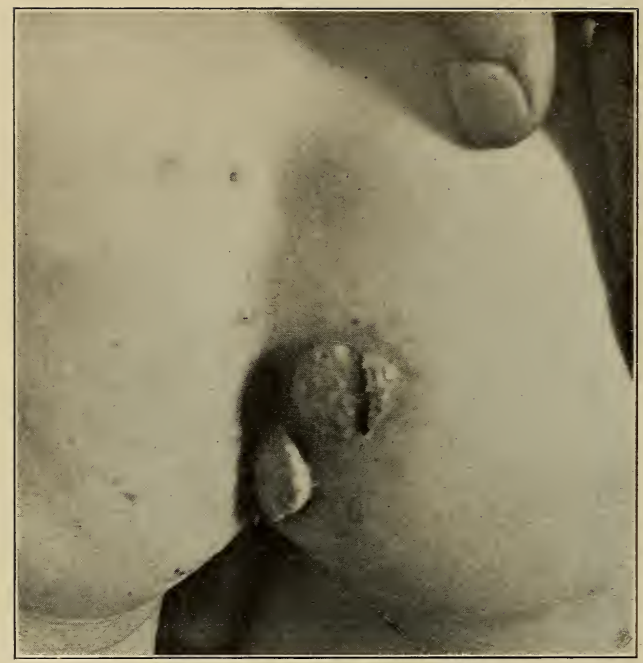

FIG. 89. CONDYLOMATA LATA

These lesions are large papules because they are stimulated to overgrowth by maceration and warmth incidental to the physical peculiarities of the body folds. Often vegetating pemphigus looks like this.

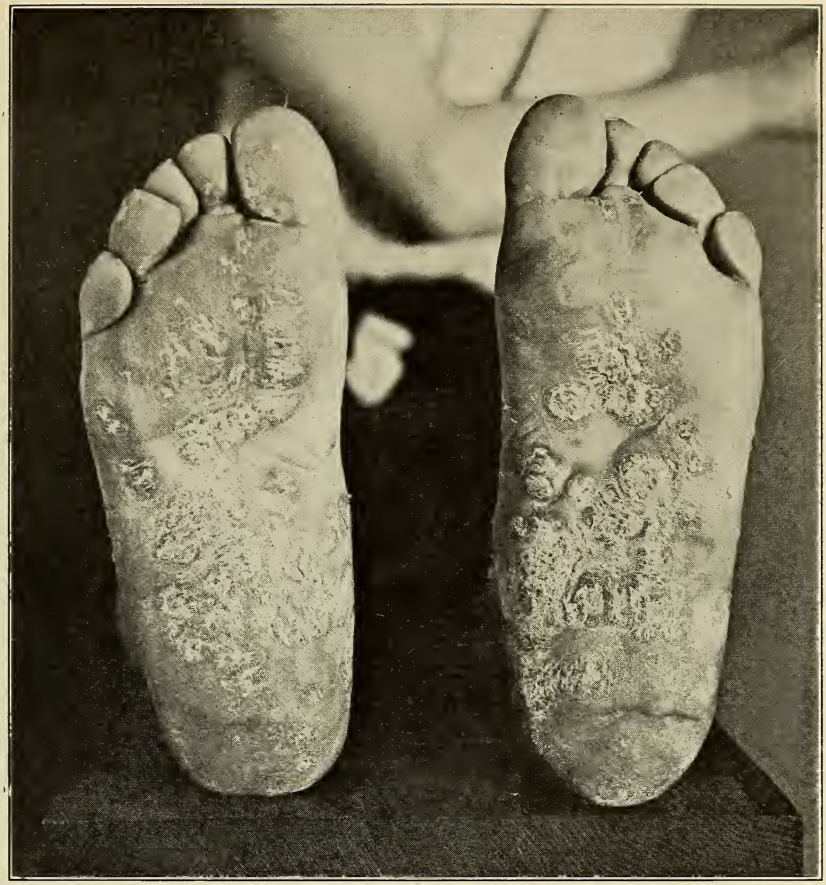

FIG. 90. PLANTAR SYPHILODERM (SECONDARY)

The underlying element in this type is the papule modified by the thick palmar or plantar epidermis. Thus marked scaling occurs. In secondary syphilis the lesions are usually bilaterally symmetrical, and roughly grouped. Arsenical keratosis and squamous dermatitis must be excluded in cases of this sort.

CHAPTER XLIII

ACQUIRED SYPHILIS. THE TERTIARY STAGE OR PERIOD OF ATTEMPTED SELF-LIMITATION

Syphilis must be conceived as a disease with a generous intention not to harm the human body too much. These good intentions, however, are so slow in evincing themselves that permanent harm is often done before self-limitation has become complete.

The recession of the disease occurs in alternating waves of recrudescence, and quiescence. After the secondary period is over, a stage of more or less pronounced latency, sometimes lasting for years, arrives; and then little by little signs of activity may again be noted. No part of the human body may claim exemption, but usually not all parts of the human body are involved. In this chapter, only the cutaneous manifestations of the tertiary stage will be described, a separate chapter having been reserved for conditions affecting other organs and tissues.

Before describing these cutaneous changes, however, it may be well to indicate the general characteristics of teritary syphilis. It has already been stated that the keynote of this phase of the disease is a marked tendency towards localization of the process, in contradistinction to the dissemination thereof, characteristic of the early stage. In other words, the host has tried to wall off the invader and has partly succeeded. At times, for biological reasons which it is impossible here to discuss, the immunizing power of the host is inadequate and the disease lights up again, quickly to be curbed. Whether these phenomena, in the last analysis, depend on a variable degree of virulence of the invader, or on a variable degree of resistance of the host, or both, has not yet been determined. In one place or another in the body, however, the host reacts to the parasite by throwing out an inflammatory tissue wall about it. The lesion thus produced is a granuloma, the exciting cause of which is the treponema, and the significance of which is a successful tissue reaction against the organism. Investigations of Warthin of the University of Michigan have conclusively proven that the treponema is capable of lying dormant in the human tissue without exciting

any inflammatory reaction. As a corollary to this it is presumable that when the tissue reaction is called forth, the host is better able to take care of the invader than when not.

Be all this as it may, the striking feature of tertiary syphilis is the gumma. It is the appearance and disappearance of gummas that give us clinical evidence of tertiary syphilis. Gummas, then, are not likely to be numerous. They are practically never symmetrical, usually there are no general glandular enlargements as in secondary syphilis, but as a rule the Wassermann reaction is positive, although, according to various investigators, in from ten to forty percent. of the cases this may not be so.

Gummas that involve the skin conform roughly to several fairly distinct types, and in general may resemble any other type of infectious or non-infectious granuloma. According to whether they are small lumps or scaling, or more voluminous they are termed nodular, squamous or gummatous.

The Nodular Gumma may be pointed, circinate, or serpiginous. Whatever its sub-type it consists of groups of papules or nodules of a coppery color and a distinctly resistant, if not hard, feel. Some gummas are seen in groups the outlines of which resemble a kidney, fan, or horseshoe, usually indicating by their contour their origin at a fixed point from which they spread forward and laterally. If we regard the syphilitic granuloma as uniform in type, whether it be a chancre, secondary syphilide or a lesion of late syphilis, we realize that the life history of a single lesion in all the stages of syphilis is subject to the same variations. Thus the differentiation among various gummas, as has been done and will be done, is artificial, but for practical purposes necessary, because the technical language of syphilography has been accommodated to our visual impressions.

To digress for a moment, it may well be here further emphasized that any type of syphilitic lesion, including a gumma, is bound to start with the simplest form of inflammatory reaction, namely the red spot, and evolve through all the gradations with which we are already familiar. Thus we observe the papular or nodular gumma, the scaling gumma, and the ulcerative gumma; and among the nodular gummas we find the sub-varieties already mentioned.

To return, now, to the description of nodular gummas for the moment interrupted. When gummas spread peripherally they may or may not heal centrally. These are called **serpiginous** gummas (Figs. 91 and 92). Thus the nodular gumma embraces any or all of the above characteristics. These may consist purely of group

nodules, or of nodules grouped to form a circinate outline, or of nodules not only grouped and circinate but serpiginous. If many such patches should develop in a comparatively restricted area they coalesce, and the resulting plaque possesses a festooned outline. A gumma, according to whether it has destroyed a great deal of tissue or not, leaves scars. The scarring may be preceded by ulceration, or acquired by destruction without visible necrosis. If there has been visible necrosis and ulcers form, the ulcers may be, and usually are crusted. The crusts form as a covering over the floor of the ulcer. If the margin progresses beyond the crust, there is entirely new crust formation, the new crust raising the older crust from below. This may go on in several layers so that the resulting lesion represents an ulcer surmounted by an oystershell-like covering which gives the picture of what is known as the **rupial syphilide.** Gummas form on any part of the body, but the face, the scalp, palm, sole, shin and buttocks are the sites most likely chosen.

When a gumma consists purely of nodules one observes a plaque, with the outlines already described, in the form of elevations on the skin varying in number according to the size of the plaque, and in size from bare perceptibility to the diameter of a dime. According to the diameter and elevation the individual lesions are either pointed or lens shaped, surmounted by scales (squamous), or ulcerative and crustaceous if a crust forms. If the patch heals centrally, scars of various size, shape and density develop, depending upon the degree of preliminary necrosis. As a rule, these scars are thin and delicate with a rather crumpled, pliable surface suggesting paper, and are inclined to be brown but are sometimes devoid of pigment. The total area involved by such groups of lesions may be relatively small or extremely extensive, covering sometimes the entire buttocks, back or thigh. When numerous ulcers develop in such a plaque it may have a honeycombed appearance. If the lesions are vegetating they are called frambesiform. If the lesions are larger than nodules and more like tuberosities they are called tuberous, and according to the shape of the plaque and other circumstances already mentioned they are called **tubero-ulcerative, tubero-serpiginous, tubero-circinate,** or **tubero-squamous.**

The Squamous Gumma may be diffuse or circinate. The basic element here again is the nodule, but the nodules are so compactly grouped, so little prone to necrosis, and with such a marked tendency to scaling that they are justly called squamous gummata. They too appear on any part of the body, are coppery and somewhat waxy

looking agglomerations of elementary lesions, the outline of which may be circinate and the progress of which may be serpiginous. In involuting they leave very slight if any scarring, but show a marked tendency to hyperpigmentation, the pigmentary patches persisting for long after the active process has waned.

The Gummous or Massive Gumma (Figs. 93 and 94). The third major type of cutaneous gumma is the so-called gummous type which might perhaps better be called a syphilitic tumor, or massive granuloma. This sort of lesion is tumor-like in appearance, inflammatory in character, and either copper colored, purple or a dusky red in color. These gummas too may remain intact or necrose, and the necrosis may form a shallow or deep ulcer which in turn may spread in a serpiginous manner.

The type of ulceration common to all gummas is characterized by rather sharply punched out margins, not tending to be undermined, and a flat or slightly concave floor with or without granulation tissue, but always secreting a grumous or serous substance which crusts. These crusts have already been partly described. For the rest, they are adherent to the ulcer, and either a dingy brown or gray in color, depending upon what amount of blood or pus is admixed with extraneous matter entering into the composition of the final product. In elevating the crust bleeding occurs which is by no means profuse.

The Subcutaneous Gumma (Figs. 93 and 94). In addition to cutaneous gummas, subcutaneous gummas are to be mentioned. These are comparatively rare and usually start in the subcutaneous tissue as isolated nodules or in groups of two or three, or sometimes in oval clumps of three or four individual lesions. They are hard and feel somewhat as if enlarged lymph glands were situated in the tissue depth, or like subcutaneous fibromata. They either involute without involving the skin, after having reached the size of a hazelnut or small egg; or the process involves the skin which then breaks down after it has for a time looked like the voluminous gumma above described. It differs from the latter, however, in that it imparts to the palpating finger an obvious sensation of depth, and in that the skin is fixed to the infiltration below. Such gummas sometimes extend to the deeper tissues, involving the muscles or even the bones.

DIFFERENTIAL DIAGNOSIS

In differentiating gummas from simulating lesions, it will be clearer to do so in connection with a description of gummas as modified according to regional distribution.

Scalp. On the scalp the commonest type of gumma is the tubero-serpiginous, or tubero-ulcerative kind, a fan-shaped or horseshoe-form cluster of ulcers, crusted or not, and the hair in the affected area is absent. It is remotely possible to confuse these lesions with favus or deep impetigo. The former is eliminated by the absence of spores; the latter by the presence of other evidence of syphilis, notably the Wassermann reaction. The surviving scars cause permanent baldness, and the areas somewhat resemble the scars left by favus, lupus erythematosus and folliculitis decalvans. It is impossible to tell by looking at the scar which of these processes caused it.

Lupus vulgaris of the scalp is comparatively rare. The differentiation between tuberculosis of the scalp and syphilis requires a differentiation between the lupus nodule and the syphilitic nodule on the clinical grounds already mentioned (Chapter XXV). In addition to this, the Wassermann and tuberculin tests, and microscopic studies may be necessary.

At the back of the neck, we frequently see serpiginous gummas of one kind or another, suggesting perhaps acne keloid (Chapter XXXV). In the latter condition, there are pustules rather than granulomas, and the ulcers are very superficial and undermined. Comedones are admixed with the lesions and the scars are coarse and voluminous as contrasted with the more pliable cicatrices of syphilis.

Face. Gummas of the face are of two types. The first is the tubero-serpiginous or ulcerative sort, already so frequently alluded to, and now familiar in its clinical aspect. It tends to appear on the chin, temples and nasolabial folds but is by no means confined to these areas. The nodular types seen on the face are particularly prone to occur in the nasolabial folds and strongly suggest seborrhea or lupus vulgaris, but are differentiated from the former by the absence of seborrheal elements, and from the latter by the absence of the clinical features of the lupus nodules (Chapter XXV). On the nose and in the neighborhood thereof, the voluminous gumma is very likely to appear. While in the tumor stage it strongly resembles the tumor form of lupus vulgaris, sarcoid, or intumescent lupus erythematosus, as well as rhinoscleroma (Chapter XXIII). The differentiation from lupus vulgaris has already been indicated. Clinically, sarcoid may so closely resemble this type of gumma that only the microscope and the Wassermann test, properly applied, will serve to differentiate the two. Rhinoscleroma is characterized by greater hardness, less evidence of inflammation and its microscopic

structure. Lupus erythematosus is excluded by absence of marked scaling and absence of dilated blood vessels (Chapter XXV). When voluminous gummas of the nose ulcerate they again strongly suggest lupus vulgaris, but on closer study the ulceration of the latter is found to be more irregular, the edges are more undermined at the margin, there are lupus nodules, the tuberculin test is positive, the Wassermann test is negative, and in favorable cases the microscopic picture is distinct. Moreover, lupus vulgaris tends to destroy the tip of the nose while syphilis is prone to destroy the bridge. Thus, in lupus vulgaris the nose ultimately becomes a beak while in syphilis it becomes saddle-shaped.

The Trunk. Gummata of the torso may be of any shape or size and have been described early in this chapter. It will be impossible, without going into too great detail, to differentiate such gummata from the various conditions they simulate. But it may be permissible to catalogue the more important conditions. The squamous variety resembles psoriasis or seborrhea. The nodular variety resembles tuberculides, tuberculous lepra, and sarcoid. The serpiginous variety resembles various forms of tuberculosis. The gummous variety resembles blastomycosis, sporotrichosis, and certain nonspecific ulcers, as well as ecthyma. The student is referred to the various chapters dealing with these conditions for more detailed information.

Palms and Soles (Fig. 95). Gummas of the palms and soles are tubero-squamous in character and hence resemble the various forms of tylosis, particularly squamous dermatitis and arsenical hyperkeratosis. The former represents a practical point to be dwelt upon. Chronic dermatitis of the palms and soles is common and usually associated with evidence of the disease elsewhere. The underlying elements are found to be vesicular and there is itching, whereas in syphilis there are no vesicles and no itching, but rather an underlying infiltration. Moreover there is less tendency to circinate outline in dermatitis than in syphilis, and the Wassermann test is negative in the former.

Mucosa. Gummata occur on the mucous membrane and mucocutaneous surfaces in the various forms, prevailingly the ulcerative. On the lips the commonest type is the voluminous gumma which ulcerates and which most closely resembles the chancre, cancer and tuberculosis. The chancre is ruled out by the lack of regional gland enlargements, and the absence of spirochetes as well as the absence of possible secondary manifestations. Cancer is ruled out

by the clinical appearance (Chapter XXIII), the presence of glands and the microscopic appearances. Tuberculosis of the lips is rare and usually tubercle bacilli are present.

Tongue. The foregoing also applies to gummata of the tongue and tonsil, but tertiary syphilis of the tongue as a whole deserves some special mention because it occurs in various types. In the first place, there is the so-called leucoplacia of the tongue, including the buccal mucosa as a whole. It is characterized by more or less extensive pearly white, thick patches of relatively circumscribed outline resembling lichen planus (Chapter XIII). In lichen planus there are usually, although not always, cutaneous manifestations. Lichen planus of the mucous membrane is furrowed and perhaps not so dead white as syphilitic leucoplacia. At the periphery of the leucoplacial patch one often sees small polygonal papules.

The second form of syphilis of the tongue is a multiple infiltration of the muscle of this organ, with numerous smaller or larger gummas, which become fibrous and which present the picture known as interstitial glossitis. This bears a rough resemblance to the gummatous liver, as seen at autopsy. Such a tongue is irregular as to its surface and the mucosa is smooth and rather glossy, unless covered by leucoplacia. It feels hard and irregular when pressed between the fingers, and is painless.

The third form of lingual tertiary syphilis is the ulcerative gummatous form in which one observes two or three comparatively small syphilitic ulcers such as one sees on the skin. In addition to this it is necessary to remember that any of these three forms may coexist in any combination. There are no glands. Tuberculosis of the tongue is rare; syphilis is comparatively common. In tuberculosis it is usual to find the bacilli; in tertiary syphilitic glossitis no organism is found, and the Wassermann test is positive.

In cancer of the tongue a very confused situation arises inasmuch as lingual syphilis predisposes to lingual cancer. The differential diagnosis, which is extremely important, has been described in connection with malignant epithelial neoplasms (Chapter XXVIII).

As to gumma of the tonsil, the same generalities already mentioned apply, with the further circumstance that Vincent's angina may be simulated. In this condition the causative organisms are readily demonstrated, and the acuteness of the onset and the absence of the Wassermann reaction serve to prevent error.

Genitalia. Gummata of the genital organs and anus are usually of the voluminous type suggesting tuberculosis, epithelioma and

primary syphilis, recognition and differentiation of which follow along the lines indicated in connection with gumma of the buccal orifices.

Summary. To sum up, then, cutaneous and mucous gummata have the following characteristics: they represent granulomas which tend to group and ulcerate. There are no regional adenopathies. They heal after a period of more or less marked necrosis. The Wassermann reaction is present in a substantial majority of instances. They resemble primarily primary syphilis, various forms of tuberculosis, other infectious granulomata and epitheliomata, and must be differentiated from all of these conditions by the clinical and laboratory methods, already alluded to.

Tertiary syphilis as a whole is characterized by the presence of the cutaneous and mucous manifestations already described; by the presence of the Wassermann reaction; by certain general clinical characteristics; by a tendency to the focal appearance of the disease at certain more or less definite points, and by a tendency to recur at these same points; by the absence of general symptoms in the sense employed in connection with secondary syphilis; and by the involvement of the rest of the body in a definite and characteristic manner to be described in the next chapter.

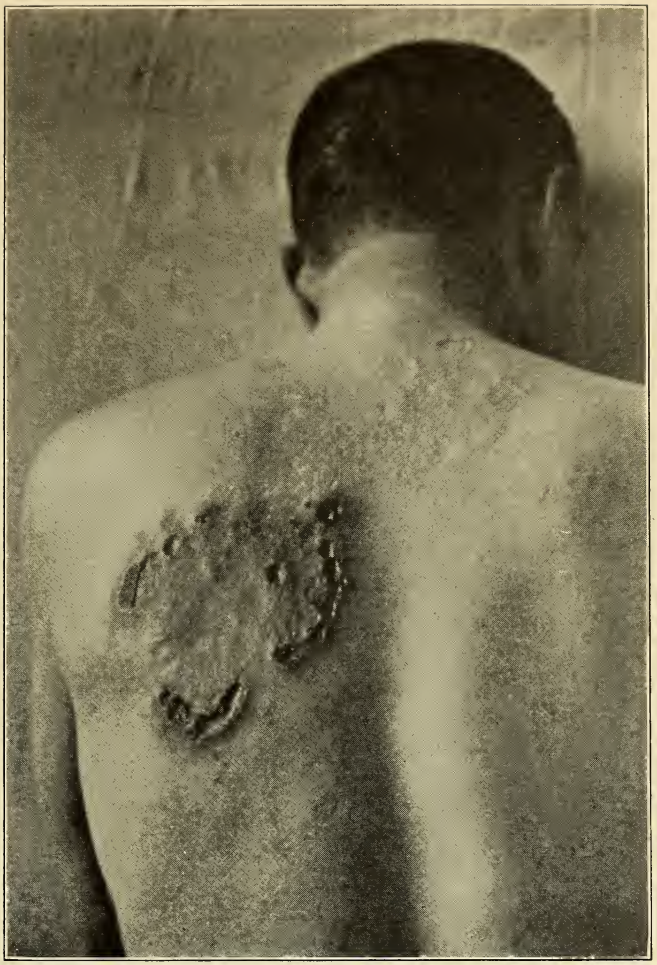

FIG. 91. TUBERO-SERPIGINOUS GUMMA

There is a scar on the back where the advancing lesion has left its traces. At the margin of the scar are seen festooned groups of coalescing lesions which are tuberous and nodular and ulcerous. The progress of the lesions leads to the formation of kidney shaped segments.

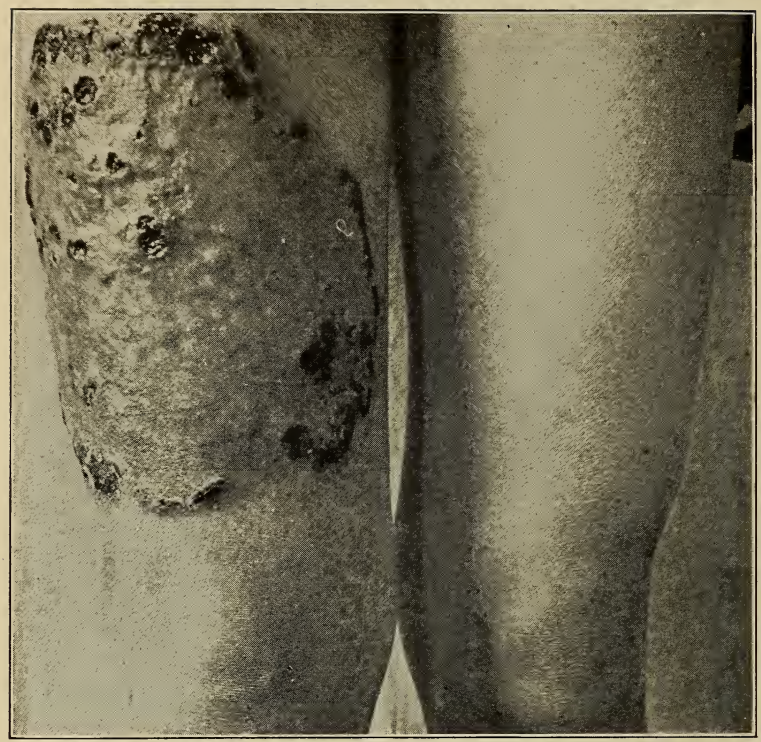

FIG. 92. TUBERO-ULCEROUS GUMMA

The progress of this lesion has been centrifugal. The entire area is scarred. The margin consists of ulcers which, on the inner aspect of the thigh, show kidney shaped grouping. The centre of the lesion shows renewed activity. Lesions of this sort often mimic tuberculosis.

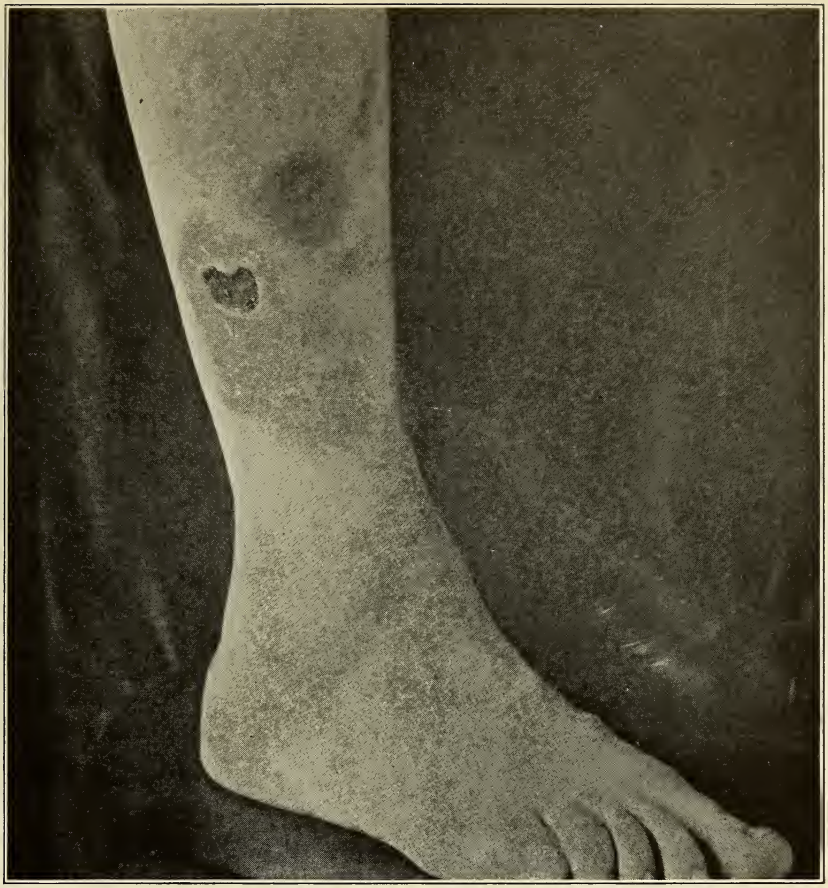

FIG. 93. GUMMA OF THE SHIN

Note the reniform contour of the ulcerating gumma, and the other unbroken lesions resembling erythema nodosum.

FIG. 94. SOLID GUMMA

Note the furuncular aspect of the lesions. Some ulcerate and crust, as on the left arm. Others resemble healing furuncles, still others are small nodules.

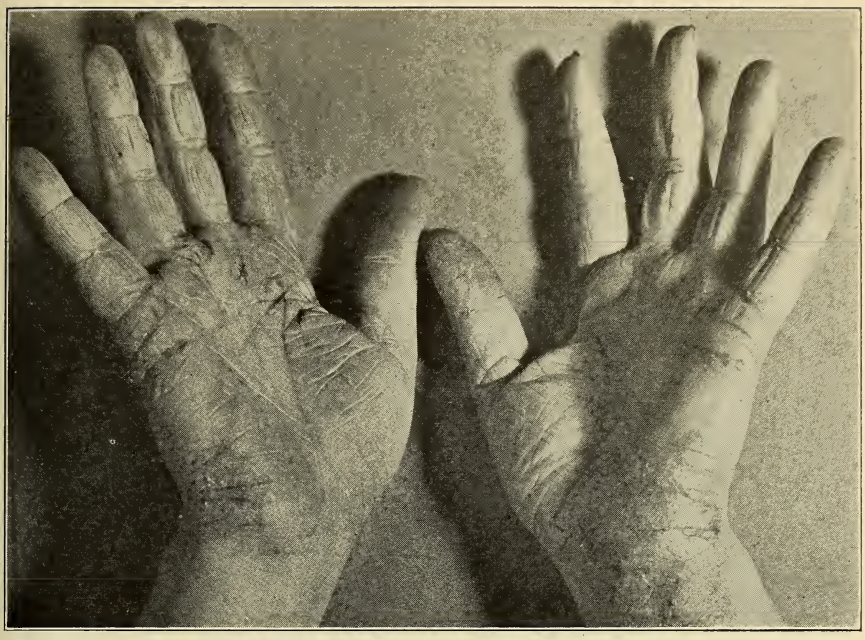

FIG. 95. PALMAR SYPHILODERM (TERTIARY)

Although usually unilateral, tertiary palmar and plantar syphilis may be bilateral. There is often distinct festooning or grouping of the lesions; at times they are discrete, however, and almost non-specific in aspect. At times there is simply scaling, as on the right thumb.

CHAPTER XLIV

ACQUIRED SYPHILIS IN ITS GENERAL NON-CUTANEOUS MANIFESTATIONS

Although perhaps in a work of dermatology the phases of syphilis herein to be discussed have no place in the academic sense, they must, however, be dwelt upon to give a broad view of the disease as a whole. Before the discovery of the Wassermann test, and before we were able to apply other refinements to our study of this malady it became customary to regard syphilis without objective manifestations as latent. To this day we are forced to apply this term to the disease when no morbid evidence exists save the presence of the Wassermann reaction. It is conceivable that syphilis may be dormant and the Wassermann reaction be negative, so that to all intents and purposes the disease appears to be cured. But experience teaches that in a substantial proportion of cases in which all clinical and serological evidence is lacking, the disease may at a later date flare up. For this reason it is necessary for the syphilographer to know syphilis, not only in its cutaneous, but in its general aspects.

The work of Warthin, already several times alluded to, indicates that from the standpoint of the general pathologist syphilis is an incurable disease. In a practical sense it is not necessary to be so pessimistic, inasmuch as we all die of something and if a man succumbs to the inevitable at the age of seventy-eight, say from an aortic insufficiency, it actually makes very little difference whether this cardiac condition was syphilitic in origin or not. Thirty years earlier in the individual's life this view could not be entertained and it would be the duty of the syphilographer to treat the patient's syphilis energetically. Further, it is clear from Warthin's work that syphilis may lie dormant in so insidious a manner as not to provoke any tissue reaction. In other words, the parasite is concealed within the host, ready to become active when the latter's resistance for some reason or other diminishes. There is no organ or system of the human body that cannot be a prey to the spirochete, but it is the central nervous, the cardio-vascular and the skeletal systems that appear to be the most vulnerable. In describing these com-

moner as well as the rarer syphilitic diseases, it will be possible only to offer the briefest sketches, for the symptomatology is so complex and so extensive that this phase in itself would require an encyclopedia to do it justice.

Nervous Syphilis is characterized by a group of conditions which depend upon whether the meninges or the central nervous axis be more greatly involved. If the meninges play the greater rôle we get symptoms of chronic meningitis or pachymeningitis which vary in intensity and character according to the localization of the maximum degree of the process. Thus, symptoms of syphilitic spinal meningitis, or cerebral meningitis, or a combination thereof, will be produced. Closely related to the spinal forms is tabes dorsalis, or locomotor ataxia, which begins as a rule in the posterior roots, finally involving the cord more or less, particularly the tracts controlling locomotion and muscular sense.

If the central nervous axis is involved one gets the picture of encephalitis or myelitis or general paresis. It is possible too for the syphilitic process to localize itself completely, either at one point in the cord or brain, a gumma forming giving the symptoms of brain or cord tumor; or the process may run its major course in the arteries of the brain and cord, giving the picture of cerebral or cerbro-spinal endarteritis. The clean cut clinical entities are tabes and paresis, but these are so often associated with each other, or with vascular syphilis of the central nervous system, or with meningitis that it is almost unphilosophical to endeavor artificially to establish definite clinical entities. The recognition of these conditions depends partly upon clinical and partly upon laboratory evidence.

It is possible to enumerate these features only briefly. In general it may be said that all central nerve syphilis tends to present the following symptoms, viz.: the Argyll-Robertson pupil, ptosis, diplopia, diminution of the deep tendon reflexes, particularly the patellar, Romberg's sign, and various sensory disturbances. In paresis mental symptoms are superadded to these, namely, lapses of memory, disturbances of speech, and attacks of unconsciousness. In tabes lightening pains, and rheumatoid pains in the feet and calves are additional symptoms. In meningitis as well as in paresis there are intense headaches, and in the former the classical signs of that condition appear, particularly the Babinski reaction.

In vascular syphilis a condition closely simulating paresis, if not identical, arises, and if the larger vessels are involved there may be apoplexy. It is possible in syphilitic periosteitis of the cranial

bones forming the cerebral cavity to have the picture of epilepsy. In gummata, as already stated, brain or cord tumors are simulated.

By means of special examinations further evidence of central nerve syphilis is detectable. Ophthalmoscopic examination often shows edema, inflammation, or atrophy of the 2nd nerve, and if the atrophy is complete there is absolute blindness. One of the early signs of involvement of the disc is a diminution in the color field. Deafness may be caused by involvement of the auditory branch of the 8th nerve with diminished bone conductivity. The 7th nerve is rarely involved, but either semilateral facial palsy or Bell's palsy may be encountered. Particularly in tabes, control of the bladder and rectum may be impaired or lost so that there is incontinence, and long before this stage has been reached cystoscopy may show a trabeculated bladder, or a loss of bladder tone with urinary retention.

In all of the conditions enumerated the blood Wassermann reaction is likely to be positive; but if it is negative an examination of the cerebrospinal fluid will usually show evidence of the disease excepting when the process has become complete, either through self-limitation or therapy, in which case, although the scar tissue formed can never be rejuvenated, there is no clinical evidence of advancement and the cerebrospinal fluid becomes normal.

The changes in the cerebrospinal fluid are as follows: increased pressure, an increase in the globulin content, the presence of the Wassermann reaction, increased number of leucocytes, and the presence of the Nonne gold chloride reaction. Without entering too much into the refinements of this matter, be it said that the pleocytosis is likely to be smallest in tabes and greatest in syphilitic meningitis. The same applies to the globulin test. The gold chloride test is most characteristic in paresis, yielding what is known as the paretic curve, but this phenomenon is not universally accepted as being of invariable value. The Wassermann test, as well as those other reactions with the exceptions above stated, always indicate an active process in the central nervous system. It is not fair, however, to imagine that the diagnosis can be labelled by a test tube reaction, for correct conclusions can be reached only by a proper and intelligent correlation of the laboratory and clinical findings.

Syphilis of the peripheral and special nerves, excepting insofar as some of the nerves of special sense are concerned, is not very well understood. Undoubtedly, pressure at the point of emergence of nerves may determine various forms of peripheral neuritis.

Ocular syphilis has already been touched upon. It is usually associated with other conditions already mentioned, but it is possible for optic atrophy to be the only manifestation of central nerve syphilis. There may also be syphilitic retinitis. There is nothing further to add regarding auditory syphilis or syphilis involving the 7th nerve. It is conceivable that syphilis may cause mental deterioration or insanity, simulating the various classical types of psychic disturbances, just as in paresis there are all sorts of aberrations even other than the classical disturbances of mentality, grandiose delusions and the like. Secondary to more important conditions, trophic disturbances may arise such as bed sores and perforating ulcers of the foot in tabes.

Although in the main nerve syphilis chronologically is a late manifestation of the disease, it is not yet known whether its inception may not be placed early in the course. As a matter of fact, in a certain proportion of early cases changes in the cerebrospinal fluid have been noted, particularly the presence of the Wassermann reaction, the globulin reaction, and pleocytosis. Whether this may not be really a transitory change in the pia and arachnoid is not yet definitely settled, but it would seem as though, even at this early stage, substantial changes occur in the nervous system due to the presence of spirochetes causing symptoms chiefly referable to meningeal inflammation, and changes perhaps permanent. In support of this point of view it has been noted that the optic nerve is swollen in about two percent. of all cases of secondary syphilis, and in a fair proportion of cases there is transitory and rarely ever permanent deafness due to auditory nerve involvement. The author has observed an intractable meningitis develop in a patient while under intensive modern treatment for secondary syphilis, and a second case in which Bell's palsy developed during this stage.

Cardiovascular syphilis may arbitrarily be divided into syphilis of the heart and of the blood vessels. Myocarditis and aortic insufficiency are the commonest of these conditions, but there may be gummas of the heart wall and, occasionally, if such lesion localizes itself unfortunately, arrhythmia is noted; or if the bundle of His is involved symptoms referable thereto are observed. Nearly all cases of aortic insufficiency are syphilitic in origin, and the majority of cases of chronic myocarditis in the adult are due to the same cause. The vascular disturbances, aortitis, aortic aneurysm and arteriosclerosis or atheroma, particularly in the young, are almost entirely of syphilitic origin. The coronary arteries are peculiarly vulnerable,

and angina pectoris is nearly always syphilitic. It is unnecessary to enter into the symptomatology of these conditions. Conceivably, many forms of acroasphyxia may be syphilitic, but so far as the author is concerned, he has never in a single instance been able to prove the fact. The work of John Stokes indicates that at least some forms of telangiectasia have a syphilitic basis.

The Kidneys. Unquestionably, early syphilis may cause a parenchymatous nephritis, but the majority of renal changes referable to specific infection are secondary to the mechanical influence of cardiovacular disturbances themselves syphilitic in origin. Gummata of the kidney are rare. In all of the above conditions the Wassermann reaction is likely to be present, and if it should not be, the diagnosis may be corroborated by symptomatic improvement under antispecific therapy. In applying this form of reasoning, however, it is necessary to avoid the *post hoc propter hoc* fallacy.

Syphilis of the skeletal system involves the bones and joints either by producing periosteitis with osteitis or osteomyelitis, or by producing gummata, or arthritis or joint disturbance not due to local involvement. Periosteitis is commonest on the shin, sternum and bones of the cranial vault, particularly the parietal bones. Here painful swellings, hurting most intensely at night and very tender, develop. The skin above them becomes reddened, and sometimes a picture resembling erythema induratum is produced. The last remark applies chiefly to the shins. Osteomyelitis develops mostly in the long bones, and mainly in the tibiae. Usually in the tertiary period, but sometimes in the secondary, as the result of an extinct local process, roughening of the anterior margin of the tibia is found, due to productive periosteitis. Gummata, as has been stated, develop mainly on the sternum and the bones of the cranial vault. They are often painless, and are indolent, fluctuating masses, the area of fluctuation being limited by a hard ring of bone. Another site of election for this process is at the sterno-clavicular junction. It must be remembered that any of the bones of the body may be affected. Gummata may develop at any of the large joints, and possibly those of the sterno-clavicular type actually have their starting point at this joint rather than in either the shaft of the clavicle, or in the sternum itself. The epiphyses of long bones are peculiarly liable to be the starting point of this process.

When joints are involved, and particularly when the synovial membrane is the starting point, an effusion takes place into the articulation. Thus the joint swells hugely, its function is disturbed, it

becomes distorted according to the extent of the hydrarthria. This process must be distinguished from the Charcot joint, or the second type of joint disturbance alluded to in the introductory sentence. The Charcot joint is a trophic disturbance, observed in tabes, in which the leg swings like a flail on the thigh, and the explanation of which is a disturbance of nutrition referable to the disease of the cord. This condition was purposely not included in the description of tabes above. At times syphilis of the joints may resemble acute polyarticular rheumatism; this is particularly the case in joint disturbances during the early stages.

The rarer general disturbances due to syphilis will be briefly enumerated. Syphilis of the **lungs** is difficult to recognize and gives the picture of chronic pneumonia, or of cavity formation similar to that observed in advanced pulmonary tuberculosis. It may be distinguished from the latter by routine diagnostic procedures including the performance of the Wassermann test. **Mediastinal syphilis** is almost impossible to differentiate from mediastinal tumor save by means of the serum test.

Syphilis of the abdominal viscera has not yet been closely studied. In the early stages the liver may be enlarged and a mild, acute hepatitis provoked, sometimes with jaundice and fever; while in the later stages multiple gummata associated with interstitial hepatitis may develop, or there may be syphilitic cirrhosis. Syphilis of the spleen in the early stages causes a soft enlargement of this organ as Udo Wile points out in an excellent article written several years ago. In later syphilis **splenetic gummata** have been observed. Pancreatic changes are rare. **The hollow viscera** present changes in the stomach and rectum due to scarification obviously following ulcers of syphilitic nature. All kinds of deformities of the stomach have been described with their obvious symptoms and signs, and syphilitic proctititis of the ulcerative variety is uncommon but not rare. The renal changes have already been mentioned. Excepting for one condition, **syphilis of the generative organs** in both sexes has not yet been closely studied. This one exception is almost as important as central nervous and cardiovascular syphilis. **The testes** are commonly involved, either by the presence of gummata or there are gummata of the epididymis and rarely of the cord. It is possible, however, and this will be re-emphasized with congenital syphilis, that the testes harbor spirochetes without provoking any gross lesion clinically recognizable. A testicular gumma is a large, hard, somewhat egg-shaped mass, not particularly sensitive, which, under favor-

able circumstances, may give rise to hydrocele of variable extent; and if the effusion is sufficient, the latter may conceal the former until after tapping. The Wassermann reaction is practically always present and the condition responds quite readily to treatment. It must be differentiated from tuberculosis of the testes and malignancy by the conventional methods employed for this purpose.

Although syphilis may involve, as has been stated, any organ in the body, and simulate almost any disease, it is unnecessary to go beyond the facts mentioned for the purpose of this book. It is necessary, however, to emphasize once again that the astute physician should be on his guard to recognize syphilis even though the Wassermann reaction be absent; or, when present, earnestly to look for syphilis somewhere within the body. Sometimes a single enlarged gland will be found, and although glandular syphilis itself is rare and has not here been mentioned, it must be borne in mind that these organs too may harbor the elusive parasite.

CHAPTER XLV

THE MANAGEMENT OF SYPHILIS

The treatment of syphilis to-day depends upon proper use of the newer synthetic arsenic compounds, in general termed arsphenamine; mercury, and the iodides. The three drugs therapeutically show their specific properties in distinct ways.

ARSPHENAMIN

Arsphenamin should not be regarded purely as an arsenical compound, but roughly as analogous to an artificially produced antibody capable of destroying spirochetes. Hence, it is distinctly a spirocheticide, the molecule of which is bound to the microorganism by the benzol ring, as an antibody is bound to antigen by amboceptor. What little tonic effect the arsenic might have on the host is negligible.

Mercury may be a parasiticide, or it may stimulate immune body formation in the host, or it may do both. In any case there is reason to believe that its effect in syphilis is due as much to its influence on the host as on the invader.

The mechanism by which the *iodides* work has not been sufficiently studied, but there is no doubt that the iodides are of value in the treatment of late syphilis.

Arsphenamine is now represented in the market by several different brands made both here and abroad, and it is safe to use any variety licensed by the Hygienic Laboratory. It is procured from the manufacturer in ampules containing different amounts, and it should be dissolved and prepared very carefully according to the following method.

Technic of Arsphenamin Administration. Freshly distilled and boiled warm water should be used. The entire contents of the ampule should be dissolved in this, without too violent shaking or manipulation. The clear yellow or brownish yellow solution is acid. It is dangerous to inject acid arsphenamin into the human being; consequently, it is necessary to neutralize the solution. For this purpose a fifteen percent. sodium hydrate solution, never kept for

longer than two weeks, is employed. With a sterile medicine dropper ten to fifteen drops are quickly put in the acid solution. A heavy lemon yellow precipitate forms which, upon further addition of the alkali, clears up. Then ten drops more of the alkali are added, turning the drug into a disodium salt. The solution is now ready for use. This first stage of preparation has been in a sterile beaker or Florentine flask.

We now come to the apparatus for injection. This should consist of a graduated tube, with a two to three hundred centimeter capacity. To the distal end of the container is attached rubber tubing, sterile, and terminating in an adapter that fits the needle to be used. Near the distal end of the rubber tube there should be a good clamp. The best type of needle, which should be sharp, sterile and patent, is that which is known in the trade as the Fordyce needle. About thirty cubic centimeters of sterile water are put into the container, and allowed to run through the rubber tubing, partly to flush it out and partly to exhaust the air, so that no bubbles get into the patient. About five cubic centimeters of water are allowed to remain in the container. A sterile glass funnel containing a sterile gauze, or cotton, or paper filter should be placed in the top of the container, and the arsphenamin solution should be poured from the beaker, through the filter into the container. Then, sterile distilled water should be added so that the final bulk of the solution will be twenty cubic centimeters to the decigram of arsphenamine. Thus, if four decigrams are used, there should be eighty centimeters of fluid, etc. The patient is now placed recumbent on a treatment table, his arm extended so as to flatten out the cubital fossa as much as possible, and a tourniquet placed about his biceps snugly enough to dilate the veins, but not so tight as to obstruct arterial circulation. The arm is then cleansed with ether or alcohol, the needle inserted, and the tourniquet removed. The connection with the arsphenamine apparatus is completed, and the fluid is allowed slowly and steadily to flow into the veins.

Neo-arsphenamine may be employed in a dosage half again as great as that of the arsphenamine. This substance is neutral and needs no alkalinization. It can be used in greater concentration; for example, four decigrams might be dissolved in fifteen or twenty cubic centimeters of sterile water. The question of the relative value of the two varieties of arsphenamine will be discussed below.

Toxicity of Arsphenamin. As a matter of fact arsphenamin, if properly employed, is rarely toxic. All apparatus should be

thoroughly sterilized; the water should be freshly distilled and sterile. As mentioned, the sodium hydrate solution should never be more than two weeks old, and the solution should flow slowly. If these rules are observed, it is simple to employ the medicament. When properly tolerated, it works purely as a spirocheticide, and does not harm the patient. Its harmful effects, if any do appear, come either immediately or shortly after the injection. When they come immediately after the injection the symptoms roughly resemble those of mild anaphylactic shock, and were originally termed by *Homer Swift* "anaphylactoid reactions" and more recently nitritoid reactions. The patient complains of a sensation of pressure in the epigastrium and chest, the sclera becomes red, the face first red, then bluish, and then swollen, the respirations become shallow and rapid, the pulse very rapid and soft. The attack usually disappears within half an hour. It it a very dramatic syndrome, and one almost imagines that the patient cannot survive. Fortunately the phenomenon is rare. It usually occurs after the third, fourth, or fifth injection in the series. It can be controlled by a hypodermic injection of seven to ten minims of suprarenal gland extract, and can be prevented, as *Stokes* pointed out, by injecting 1-50 of a grain of atropin hypodermatically five or ten minutes before the arsphenamin injection, in patients in whom previous similar reactions have occurred.

The delayed type of reaction takes one of several forms. The mildest is a sense of malaise with pain in the bones and joints suggesting influenza; or nausea and vomiting with diarrhoea, or when more severe, albuminuria and suppression of urine. The last is extremely rare. Another form of reaction is characterized by the appearance of an exanthem which may be transitory and looks like measles or scarlet fever, or there may be a universal toxic rash terminating in an exfoliative dermatitis that persists for weeks, prostrates the patient, is accompanied by suppression of urine, or even with the symptoms of acute hemorrhagic nephritis, and which usually terminates in recovery. Hemorrhagic encephalitis has also been reported. The mortality after arsphenamin reactions is very low and, on whole, the harmful effects on the kidneys, as *Schamberg* has shown, are much less serious and much less frequent than after mercury injudiciously used. Serious reactions in this group develop anywhere from six hours to a week after the injection, and serve as a contra-indication to the further use of the substance. Occasionally after arsphenamin acute yellow atrophy of the liver develops

which is fatal. Occasionally, too, icterus which usually passes off within two or three weeks, is seen. Cases of pneumonia have been reported, after the use of arsphenamin, but the reports are not clear and I question whether the drug ever produces this disease. Considering the hundreds of thousands of arsphenamin injections administered during the past ten years, the untoward effects form so small a total, that the danger may be regarded as negligible.

The cause of the reactions may be either intolerance to arsenic itself, or to some unknown substance in the compound, as *Schamberg* believes, or to various impurities introduced into the substance through careless technic, or to hypersusceptibility on the part of the patient to the drug. A detailed description of this matter is unnecessary. Neo-arsphenamin is supposed to be less toxic, and no less efficacious than arsphenamin. So far as my experience goes, I can subscribe to neither view. Injections should be made on an empty stomach. This in itself may prevent many reactions.

One more type of reaction must be mentioned which has nothing to do with the drug, but which is elicited after any antispecific remedy, whether it be mercury or arsenic. This is the so-called Herxheimer reaction. It was noticed by this writer that in secondary syphilis, after the use of mercury, (this observation antedates the era of arsphenamin) the cutaneous lesions stood out more distinctly before they faded. This was explained as the result of a local liberation of toxins from the spirochetes following the destruction of the latter, which increased the local hyperemia, making the lesions look redder. Be this as it may, antispecific remedies are capable of producing this phenomenon wherever there is a syphilitic lesion. Thus, if the phenomenon develops in or near the auditory nerve, or the second nerve, for example, the patient will suffer transitory partial deafness or blindness, which disappear when the treatment is continued, but which may become permanent and complete if the physician is frightened and interrupts treatment. It was this, and not the effects of arsphenamine, that produced what were formerly called neurorecedives.

Recently there has been placed on the market a permanent solution of arsphenamin, licensed by the Hygienic Laboratory at Washington. According to a report of *Stetson* at the last session of the American Medical Association, this substance is no more toxic, no less efficacious, and infinitely less troublesome to employ than the home made solution. Its use certainly will simplify the therapy of syphilis, should the hopes of its manufacturers be realized.

MERCURY

Mercury may be used by mouth, by inunction, or by injection. The last method is the best. It is less efficacious by mouth than in any other way, and it is more irritating to the alimentary tract. The amount absorbed through the alimentary tract is small, and there is danger in making the spirochetes resistant to the drug, and rendering the syphilitic recalcitrant to treatment. At times there are special indications for its use by ingestion, and if it must be so employed, the best salts are the biniodides or protiodides, grains ¼ to ½, three to four times a day. Calomel may be used in ⅛ to ¼ grain doses, three times a day, or bichloride of mercury, grains 1-50 to 1-10 three times a day. On the whole the last salts should be discarded because of either their cathartic or toxic effect. The best preparation for inunction is the United States Pharmacopoeia mercury ointment, of which two to four grams should be rubbed in daily for six successive days, each half dozen rubbings being considered a series, and one-half dozen series constituting a course. The surface of the skin should be cleansed with alcohol, and the rubbing should last one-half hour. Six surfaces of the body should be selected,— one-half of the chest one day, the other half the next day; one-half of the abdomen the third day, the other the fourth; the inner surface of one upper extremity the fifth day, and of the other the sixth day. After each six rubbings the patient takes a hot bath. This method takes times, is disagreeable to the patient and, although efficacious, is no better than injections. *Schamberg* has shown that mercury is absorbed through the skin. The great advantage of the method lies in the fact that, if the patient is intolerant to mercury, its use can be abandoned without any accumulation having occurred in the body, such as results from injections. It should never be rubbed in in hairy parts of the body, as it tends to cause dermatitis in these situations.

Injections. When injected, mercury may be used in solution or in suspension, insoluble salts being employed for the latter. The soluble salts are the bichloride, cyanide compounds, etc. They are efficacious but must be used daily, or at the least every other day. They are painful, and my feeling is that they are less valuable than the insoluble salts. The insoluble salts are calomel, gray oil and mercury salicylate. Inasmuch as my experience has led me to believe that calomel and the gray oil hurt more, are less easily absorbed, and no more efficacious than mercury salicylate, I no longer use any

but the last substance. *Cole,* at the last session of the American Medical Association, presented X-Ray photographs illustrating the absorption of the various insoluble salts, and proved that only the salicylate was freely, easily, and regularly taken up by the body. The injections, which should be ten to twelve in number, should be begun with a one-half grain dose, which should be increased up to the toleration point, the intervals between the administration being from five to seven days.

Toxic Effects of Mercury. The first symptom of mercury poisoning is ptyalism, with swelling of the gums, and pain in the teeth. The next is diarrhoea. In some intolerant people, preceding either of these, there is albuminuria. Unless this is discovered, acute parenchymatous nephritis may develop. There is no reason for any of these things ever to happen. At the first sign of salivation the drug should be discontinued, or its dosage reduced. The teeth should be brushed with some good tooth paste or powder three times a day, as a matter of routine, and the mouth rinsed with some suitable mouth wash as often. If these measures are followed, there is very little danger in the use of mercury.

IODIDES

The use of the iodides is restricted to the treatment of tertiary syphilis in all its phases. From ten to one hundred grains of the salt should be given three times daily, in water, after meals. The drug seems to have a selective action on late syphilitic lesions. In my experience the drug is not necessary, arsphenamin and mercury filling all therapeutic requirements. This view, however, is by no means that of the majority of syphilologists, and is presented merely for what it is worth. As a matter of fact, very few people tolerate iodides well. Most of them at least develop coryza and lacrimation after the use of very small quantities of the drug, and nearly everybody sooner or later gets acne.

THE COMBINED USE OF ARSPHENAMIN WITH OTHER ANTISYPHILITIC REMEDIES

At first it was thought that one dose of arsphenamin would cure any form of syphilis in any stage. It was then found that more than one injection was necessary. To-day we know that our hope for a single sterilizing dose of the drug must be abandoned. Nevertheless, it is the best single drug available in the management of syphilis. Five years ago we thought that a weekly administration

of from four to six decigrams approximated the toxic danger point. To-day evidence is accumulating that it is therapeutically more efficacious, and its toxic effects not more likely to develop if administered every other day, or even every day for three or four days, and there are authorities who give small doses twice a day for three or four days running. If the drug is employed in inadequate quantities, or not frequently enough injected, there is reason to believe that the spirochetes become resistant to it. Thus, the object of the treatment is to destroy the microörganisms before their own resistance to any specific remedies develops. In early cases it is safe to give an injection, at least every five days, for from four to six weeks, and to combine these injections with injections of mercury salicylate in the dosage mentioned above, or with inunctions of mercury. In late syphilis the iodides may be added, and in early syphilis there is no objection to the iodides. I am not yet prepared to state that a daily injection of arsphenamin is safe, and am here offering a scheme of treatment that may be employed by the average physician without increasing the risks of therapy to an undue point. No one knows precisely how much treatment the syphilitic needs. Thus, it is necessary for every physician to select for himself a standard, which is to be varied, according to the peculiar needs and indications of the individual case. In my own practice, I consider a single series one comprising six arsphenamine injections of four decigrams each, and ten mercury salicylate injections, but I have given as many as twenty arsphenamine injections and twenty mercury injections in a series, in late or obstinate cases. It is better to divide the syphilitic's year into four periods — two of active treatment and two of rest, alternating with each other, and controlled by clinical observation and the Wassermann test.

THE TREATMENT OF THE SPECIAL PHASES OF SYPHILIS

The Treatment of Primary Syphilis with a Negative Wassermann Test. This is the optimum time to inaugurate treatment, for the disease has not yet become disseminated. In a case of this sort, although I should personally not hesitate to give the patient a daily injection of four decigrams of salvarsan for five days running, I should not recommend such tenets to a novice. But it would be safe to give four decigrams every four or five days until from six to ten injections had been given. Combined with this, a mercury injection should be given every five to seven days. This course should be re-

peated after a rest period of two or three months, the physician watching the clinical symptoms, the Wassermann test, and the kidneys of the patient. If the Wassermann reaction does not develop during the first year, the patient should receive two series of mercury injections a year, for two years longer, before being discharged as cured. The group of patients mentioned in this paragraph are amenable to abortion of the attack.

Primary Syphilis with a Positive Wassermann Test. The chances of a patient in this stage of the disease are but slightly worse than in the phase mentioned above, and the treatment is almost the same, save that arsphenamine should be given in conjunction with the mercury for two years, instead of one.

Early Secondary Syphilis. The infection is here disseminated and theoretically more difficult to eradicate than in the above groups. Nevertheless, the majority of patients presenting this form of the disease are readily amenable to treatment, which should be carried out precisely as in the immediately preceding paragraph.

Late Secondary Syphilis. Here, although the infection is disseminated, it approximates the qualities of tertianism, and although the individual series may be modelled after the foregoing, my own experience leads me to counsel eight to twelve injections of arsphenamin, and as many mercury injections to the series, carried over a period of two to two and a half years, or longer, according to the behavior of the Wassermann test.

Tertiary Cutaneous Syphilis. Here the same principles are applied that were elaborated in the last paragraph.

Visceral Syphilis. In principle, visceral syphilis is tertiary syphilis, and the therapeutic indications for the various types of visceral syphilis conform to those included in the foregoing passages. Special points in the management of visceral syphilis require special mention.

Syphilis of the Cardiovascular System. In treating syphilitic aortitis, it must be remembered that the too sudden resolution of a syphilitic lesion in the wall of an important vessel may conceivably lead to a rupture of the latter. This also holds in aneurism of the aorta, and other major vessels. Therefore, it may never be advisable to use arsphenamin, partly because of the danger mentioned, and partly because the introduction of a large volume of fluid into an overtaxed circulatory system may harm the patient. Therefore, treatment should be begun with small doses of mercury, extending over three or four months, before arsphenamin treatment is thought

of. In this way it may be possible to cause the development of a scar sufficiently strong to resist perforation. The same principles apply in syphilitic myocarditis and pericarditis, but after pericarditis has resolved, provided the myocardium is intact, arsphenamin may safely be used. Syphilitic valvular disease is subject to the same theoretical laws outlined for aortitis. Angina pectoris of syphilitic origin should also be treated according to these principles. If it is deemed inadvisable to employ arsphenamin on account of the bulk of the solution, doses of neoarsphenamin, one to three decigrams, dissolved in five to ten centimeters of sterile water, may safely be used once a week, intravenously, provided that there be reasonable certainty that there will be no mass resolution of active syphilitic lesions. The management of these cases requires the greatest tact and coöperation between a good internist and a good syphilologist.

Syphilis of the Kidneys. In the early stages of syphilis, there is sometimes a cloudy swelling of the renal parenchyma, which of course is accompanied by albuminuria. This is not syphilitic nephritis, and usually the symptoms clear up under arsphenamin which is an infinitely safer drug to use under these conditions than mercury. Substantially the same is true of acute syphilitic nephritis. An error commonly made is the interruption of all treatment. Treatment is then begun with mercury and the patient gets worse. This is due to an unsound belief, entertained by many, that arsphenamin is less well tolerated by the kidney than is mercury, for the drug that should not be used is the latter. In late kidney changes dependent upon syphilis a weekly injection of arsphenamin, in a dose of three decigrams, should be given for eight or twelve weeks, and mercury should be used in small doses, or not at all, until the renal symptoms are ameliorated. If there are symptoms of uremia, no antisyphilitic treatment should be begun until the systemic manifestations dependent upon disturbed renal function have disappeared, or until the signs of hypertension have lessened.

Treatment of Syphilis of the Lung. Sometimes syphilis of the lung can be recognized. In such cases, it is safe to treat the patient with small, weekly doses of arsphenamin and large weekly doses of mercury. When pulmonary syphilis and tuberculosis co-exist, the later usually constitutes no contra-indication to the treatment of the former.

Syphilis of the Liver. Early secondary syphilitic involvement of the liver usually yields very rapidly to treatment, as already outlined for this stage of the disease. Icterus may develop, but usually

does not. If the icterus is definitely due to syphilis, it constitutes no contra-indication to the use of arsphenamin. Late hepatic syphilis, or gummata of the liver should be treated drastically, as are the ordinary forms of tertiary syphilis. The same holds true of the spleen.

Miscellaneous Forms of Visceral Syphilis. At times the lymphatic glands are apparently the only organs affected. Drastic treatment is safe. Rarely the thyroid and parotid glands alone are affected. Here, too, intensive treatment may be followed with impunity.

Syphilis of the Central Nervous System. Aside from tabes, paresis, meningitis, and endarteritis, or a combination of these conditions, there may be gummata of the brain or of the cord, giving the picture of brain or cord tumor, or transverse or partial myelitis. The special methods of treatment vary slightly, according to the clinical condition, and it would be impossible in so short a chapter adequately to discuss the question involved. In general it is safe excepting in the presence of gummata or myelitis, to give the patient a weekly injection of arsphenamin and a weekly injection of mercury. In treating gummata or myelitis, it is safer to prepare the patient with mercury (unless the symptoms are very urgent) in the manner outlined in connection with cardiovascular syphilis. In the first four conditions mentioned, the subdural administration of arsphenamine has been practiced according to the methods advocated by *Homer Swift, Hansen Ogilvie, Ravaut, Fordyce* and others. Two schools have arisen: one condemning the intraspinous treatment as inefficacious; the other endorsing it.

Without wishing to participate in the debate, my own experience leads me to believe that extravagant claims for the value of intraspinous treatment are as vain as the equally unscientific negative attitude of those who condemn it. There is no doubt that it does not always work, but there is equally little doubt that it has arrested cases, particularly of tabes in which the general treatment had failed to produce any beneficial results. Although the treatment should not be attempted by anyone but an expert, it should never be denied a patient with central nervous syphilis whose symptoms are not yielding to ordinary antisyphilitic therapy. Paresis, on the whole, is little influenced by any treatment, because of the peculiar pathology of the condition, but tabes, meningitis, and cerebrospinal syphilitic endarteritis often yield better to a combined general and subdural treatment, than to the former alone. The debate has been

carried on with considerable venom by antagonists to the method, and although their theoretical arguments are all quite as sound as the theoretical arguments of those who favor the treatment, the practical fact remains that there is something active and constructive about subdural therapy, even though its actual value may appear to some as inconsequent as the efforts of a drowning man to catch at straws.

In treating syphilis of the auditory nerve and the optic nerve the results depend largely upon the extent of involvement. Often progressive advance and blindness may be halted by intravenous and intraspinous therapy, and in my own experience I have seen a contracting color field expand after intraspinous therapy was begun, although it had progressively contracted under general therapy alone.

Syphilis of the Bone and Periosteum. The periosteum of the tibia, the sternoclavicular junction, the small bones of the nose and the flat bones of the skull, are liable, in adults, to attacks by the spirochetes, forming osteo-periosteal gummata. In congenital syphilitics, and in children, these structures as well as the phalanges are susceptible, but no bones are immune. In the very young, too, the epiphyses of all long bones are highly susceptible. In secondary syphilis there is very commonly periostitis of the tibiae. All of these forms can safely be treated by the methods already outlined with reference to the stage and phase of the disease with which the bony involvement is associated. Very often, too, actual syphilis of various articulations, particularly that of the knee, is encountered. Here, too, the therapeutic indications correspond to what has been mentioned. There is more danger from under treatment than over treatment in these patients.

Congenital Syphilis. Newborn babies with syphilis are best treated with inunctions, and the substance best tolerated is the United States Pharmacopoeia ten percent. ammoniated mercury salve, rubbed in according to the instructions outlined in connection with inunctions. When the baby is six to ten weeks old small doses of neo-arsphenamin may be injected into the jugular vein, once a week, in doses of from one-half to one and one-half decigrams. As the baby grows older, the dosage may be increased, and in congenital syphilitics of from five to ten years of age, two to three decigrams of neo-arsphenamin may safely be given intravenously, once a week, combined with mercury inunctions of a gram of the United States Pharmacopoeia mercury ointment, as outlined above for adults. Congenital syphilis in older children, adolescents and adults is to be treated as acquired syphilis, but the outlook for cure is poor. Kera-

titis, iridocyclitis, retinitis, and optic neuritis in congenital syphilitics are to be treated as in adults, with mercury and arsphenamin, the doses varying according to the age and tolerance of the patient.

Malignant Syphilis. Malignant syphilis, better known as precocious syphilis, is rarely seen now-a-days. It is characterized by a tendency on the part of the disease to acquire tertiary characteristics, immediately after the primary stage. This may be due to peculiar virulence on the part of the spirochete, or a defect in the immunity mechanism of the host. Formerly, such cases could not be cured by mercury, but since the advent of arsphenamin they are readily amenable to treatment, and on the whole do not terminate fatally, as used to be the case before this drug had been discovered. The method of treatment corresponds to that outlined in connection with tertiary cutaneous syphilis, the mercury being used as an adjuvant to the arsphenamin.

Prognosis. It is infinitely easier to treat syphilis than to know when to stop treatment. There are rough standards, of course, but how reliable they are will depend upon the judgment of another generation of observers. To-day we believe that if a syphilitic has had no symptoms or signs of the disease for a year without treatment, he is cured, and we include among the signs a persistently negative Wassermann test, and a normal cerebrospinal fluid. This is all very well, provided we know when to start the test year, and no one ever knows precisely when. For this reason no syphilitic, however early in the disease he begins his treatment, should receive less than three years of treatment. If, after three such years, he has had no signs of activity, and the laboratory manifestations are negative, it is safe to inaugurate the test year, but during this period the patient should have at least three Wassermann tests performed, and an examination of the spinal fluid at the beginning and at the end of the test year. In spite of all these precautions, it is possible for so capricious a disease as syphilis to become active ten, or twenty, or thirty years after it seems to be cured. This is said without pessimism, for in the majority of instances the statement just made does not hold true, but it is quite impossible to state to a given patient that he does not belong in the smaller group in whom, after all due precautions, late activity may not arise.

It has become fashionable during the past two years to scoff at the reliability of the Wassermann test, and academically considered, the Wassermann test is by no means bomb proof. It unquestionably may be negative in patients who subsequently show active syphilis.

It may even be negative at times in the presence of active syphilis, and it is undoubtedly positive in some non-syphilitic diseases, notably yaws and nodular lepra. These two diseases can easily be excluded, and thus, for all practical purposes, a positive Wassermann test represents active syphilis, even though a negative Wassermann test need not exclude active syphilis. However, in the vast preponderance of cases, a negative test denotes either latency or inactivity or cure, and it is distinctly the duty of the physician to learn how to apply the negative test without condemning the entire procedure because it does not always corroborate his views. The Wassermann test in general, is the last manifestation of activity to disappear, and the first manifestation of activity to return, before there are frank clinical evidences of recrudescence. Therefore, it is a very subtle and, if properly used, useful symptom or sign of syphilis, and should be studied and understood rather than condemned by those who are disappointed in its apparent lack of constancy. The tendency to make the positive diagnosis of syphilis depend upon the Wassermann test is seriously to be condemned, for it is only a single manifestation of the disease. If this were understood, if the test were always conscientiously performed by experts instead of by a great many people whose only claim to a knowledge of serology is their ability to purchase a laboratory equipment, the procedure would have never fallen into any disrepute whatever. A safe rule as to prognosis may then be considered a year of absence of all organic and serological evidence of the disease without treatment, following three years of conscientious treatment. At times the organic signs of the disease disappear, but the Wassermann test seems never to get negative. In such instances it is safer to give the patient two series of ten injections of mercury annually for an indefinite period, although it is quite conceivable that this may be unduly cautious, and that there are cases in which the Wassermann test can never be made to vanish.

Marriage, and Syphilis in the Gravid. It is often trying to the patient and to the physician to try to decide when marriage is justifiable. Medical consent should always be withheld within three years of a fresh infection, and never given at all while there are clinical evidences of activity, regardless of the stage of the infection or type of the manifestations. The persistent Wassermann reaction in old, clinically inactive syphilis is perhaps not an adequate reason to insist upon celibacy, but the various problems are too numerous and too involved for detailed discussion in this volume.

Suffice it to say that conservatism is better than bravado. It is important, however, to avoid the peril of creating hypochondriacs.

In the early months of pregnancy, in fact up to the seventh month, it is safe to treat the infected woman energetically according to the stage and character of the disease. In the later months the problem demands nice discrimination. In any case the kidneys should control the indications. The earlier that treatment is begun, and the more energetically it is possible to pursue it, the greater the chances to save the baby from infection. At the confinement the placental blood should be collected, and a Wassermann test performed, in order to detect a possible congenital infection, and in the event of serological evidence thereof, to begin treatment before clinical evidence shows itself in the newborn. In any case the child should be carefully watched for symptoms or signs for ten years, both by physical and serological examination.

CHAPTER XLVI

CONGENITAL SYPHILIS[1]

A SURVEY OF ITS ESSENTIALS

The subject of congenital syphilis seems needlessly complex to those who have not thought about it much. It is only within recent years that our knowledge of this phase of the disease has undergone a certain degree of simplification. It is necessary merely to point to the historic laws of Colles and Profeta; the disputes as to the source of infection, whether maternal, paternal, or both; to the old name, hereditary syphilis, itself, in order to illustrate the ancient confusion.

On the other hand, to illustrate how this confusion is gradually becoming clarified, the following facts will suffice. The laws of Colle and Profeta have fallen. Abundant evidence exists proving that the apparent immunity of the mother without clinical syphilis to a syphilitic child, or that of a child without clinical syphilis to a syphilitic mother, is not immunity, but actually latent syphilis. The mothers and infants without clinical evidence of the disease nearly all give the positive Wassermann test, paralleling conditions in the latent stages of the acquired form. The source of congenital syphilis is invariably maternal, and the infection takes place through the placenta. This fact eliminates entirely discussion of congenital lues of paternal origin. The mother may have been infected by the father, or otherwise, but so far as the offspring is concerned, the source of the mother's infection is incidental. The term, hereditary syphilis, is unscientific and should be abandoned. It is not within the province of this paper to define the word hereditary, but a fundamental distinction exists between this expression and congenital, as the works of Darwin, Romanes, Hertwig, Haeckel, or any other good biologist or embryologist will show. If syphilis were actually an inherited disease, the theory of embryonic transmission of acquired characteristics would need no further support. Syphilis, passed on from mother to offspring in utero, however, has nothing

[1] This chapter is based upon "Congenital Syphilis," *N. Y. Med. Journal*, Sept. 23, 1916.

to do with chromosomes. It is bequeathed far more simply and directly by actual involvement, through contact, of the placenta, and the invasion of the latter by the mother's spirochetes. Thereupon the placenta, a small participant in the mother's systemic infection, becomes the starting point of the disease in the fetus.

No fundamental difference exists between syphilis acquired before birth and that acquired after birth. The disease is always the same, subject to variations in the virulence of the strains of spirochetes and the resistance of the host. Syphilis is syphilis. Any distinctive features the congenital form may possess, not found in the acquired form, are due to biological differences between the unborn and born human being.

Roughly speaking, the infected placenta is the primary lesion in fetal syphilis. Thence the spirochetes are conveyed by means of the placental circulation direct into the fetal blood stream. This serves to disseminate the microorganisms rapidly and effectively, sparing no organ or tissue. The liver is the first fetal organ to receive the placental blood, because the branches of the umbilical vein go direct to this organ, and because of the intimate association of placental and portal circulations. Thus the liver can never escape infection. In post-natal syphilis the entrance of the pathogenic agents into the blood stream is accomplished differently. From the primary lesion the spread of the spirochetes takes place first through the lymphatics. The entrance into the cardiovascular circulation is effected by transit of the spirochetes from the perivascular lymphatics through the vessel walls into the blood stream. (Chapter XL.) At the moment that this is consummated, such slight differences as there are between pre-natal and post-natal syphilis vanish.

The month in which infection of the fetus takes place varies; if very early the fetus will die, become macerated, and be discharged before or at term; if somewhat later, the fetus may survive and be born prematurely, or at the normal time, with the usual evidences of the disease, or as a defective, or as a monstrosity. If the infection occurs very late, for instance in the last month of intrauterine life, the infant is born at term, alive or dead, and with or without objective evidence of syphilis. Much depends upon the type and stage of the mother's syphilis. Should she be the victim of a virulent strain of spirochetes, and in the secondary period, or a secondary latent period, the degree and severity of the fetal disease will probably be greater. Should the mother have become infected in the fifth month of pregnancy, the fetus could not have become involved

in less than from two to three months, or in its seventh or eighth month. The later during parturition that the maternal infection occurs, the greater the chance of the fetus to escape the disease entirely, or to be born with earlier or milder congenital syphilis, as the case may be.

It is not uncommon to obtain the following account from a woman married about ten years. The first child is nine years old and well. Shortly after the birth of this child there came three miscarriages in the fifth, sixth and seventh months, respectively, followed by a full term baby with snuffles and an eruption. This baby lived ten days. A year later, a normal infant was born which seemed well and is now five years old. Since its arrival there have been two stillbirths, each child having appeared normal, however, and now there is another baby about nine months old. It appears well, but "catches cold" easily, and has had a rash about the anus and lips. The mother herself asserts that she has always been healthy, but remembers that after birth of the first child she had a sore on the vulva and a discharge, but paid no attention to them and consulted no physician. Such a history is significant. The child just escaped syphilis. Fortunately, he was not nursed. From her second to her fifth pregnancies, the mother was in the secondary and secondary latent stages. During her sixth pregnancy the disease was in abeyance. An exacerbation occurred contemporaneously with the seventh and eighth pregnancies, resulting in stillbirths, and during her ninth pregnancy she was again in a latent period. The Wassermann test in this family would be positive in the mother, negative in the first living child, either positive or negative in the second living child, and positive in the third.

Thus, to recapitulate, an interplay of two factors determines the form that congenital syphilis assumes; first, the month of fetal life in which infection occurs, and, secondly, the stage of maternal syphilis during which transmission occurs. In addition to this, a third influencing factor must now be included; namely, therapy. Satisfactory treatment of the mother may prevent fetal infection. Inadequate treatment may have no effect on the offspring, or may diminish the maternal infection and at least increase the chances of the fetus to escape the disease, by prolonging or intensifying the periods of the mother's latency. The older the mother's infection, the less the likelihood of transmission to the child. Thus, in the tertiary period fewer syphilitics are born than in the secondary.

Subject, too, to these factors, the symptoms and pathology of con-

genital syphilis will vary. Its morbid anatomy corresponds to that of the acquired form. In brief, no tissue or organ is sacred to the spirochete, and thus the symptoms of congenital syphilis include a wide range of possibilities and form numerous pictures, many of which are well known, others rare or uncommon. In this paper only such features of the disease will be dealt with as are found in congenital syphilitics born alive. The symptoms are in the main referable to the skin, eye, ear, liver, spleen, bones, and joints, but other organs are frequently involved, and commonly enough central nervous and mental disturbances, and diseases of the kidney, suprarenal and other ductless glands are observed, and the Wassermann test is positive in ninety-eight percent. of the cases.

For the sake of simplicity and convenience congenital syphilitics will be divided into the following groups:

1. Patients born with cutaneous and marked systemic evidences.
2. Patients born with cutaneous and slight systemic evidences.
3. Patients born with cutaneous lesions alone, subsequently manifesting systemic evidences.
4. Patients born apparently healthy, acquiring evidences of syphilis within a year.
5. Patients born apparently healthy, acquiring evidences of syphilis between the second and seventh years.
6. Patients born apparently healthy, acquiring evidences of syphilis after the age of seven years. This includes Fournier's group of delayed congenital syphilis.

1. *Patients born with cutaneous and marked systemic evidences.* This form denotes an infection of the fetus in about its first month of development. When infected so early, the fetus rarely reaches term alive, but if it should it presents a picture of general maldevelopment; the face is drawn and gives an impression of senility. A papular or bullous dermatosis, the latter the so-called pemphigus neonatorum, covers the palms and soles. Mucous patches are present on the lips, in the pharynx, and on the perineum. The liver, spleen, and lymph nodes are found enlarged. The child nurses and cries feebly, and lies quietly as if palsied. This is the so-called pseudoparalysis of syphilis due to specific epiphysitis and osteoperiostitis. The bones and joints hurt when the child moves, and are sensitive upon manipulation, the later eliciting a sharp cry. Usually death occurs shortly after birth, the cause, as a rule, being general weakness, but sometimes pneumonia. This form of pulmonary disease is an interstitial inflammation caused by the activity of the spirochete,

and known as white pneumonia, The Wassermann reaction is positive, and spirochetes are demonstrable in the lesions on the mucous membrane and in the eroded papules near the body orifices.

2. *Patients born with cutaneous and slight systemic evidences.* A macular or maculopapular eruption, with pemphigus neonatorum, slight or no snuffles, a certain degree of unwillingness to suckle, some swelling and tenderness of the bones, and even pseudoparalysis characterize this form. If the liver and spleen are not palpable at birth, they soon become so. The Wassermann test is positive. Unless treatment is begun promptly, severer symptoms set in, such as iritis and iridochoroiditis, and if therapy is still neglected all the general features of the disease appear, including permanent blindness or deafness, bony deformity, or even death from marasmus.

3. *Patients born with cutaneous lesions alone, subsequently manifesting systemic evidences.* In this form syphilis has been acquired in the seventh or eighth month of fetal development. Thus the symptoms at birth represent those of the early secondary stage. Only suspicious skin lesions may be present, or a frank maculopapular rash, mucous patches, flat condylomata at the anus, or mucous patches in the mouth or on the genitalia. When so fully developed these symptoms indicate the florescent secondary stage. At times, however, only mucous patches are seen, or groups of papules, or circinate papuloulcerous lesions in the perineum or elsewhere, or tuberoserpiginous gummatous lesions. These symptoms are associated with snuffles. The Wassermann test is positive, and spirochetes may be found in the secretion from the mucous lesions. This form indicates fairly good resistance on the part of the baby, and that the disease is in its late secondary, or perhaps even early tertiary stage. Should it progress because of neglect of treatment, all the manifestations already mentioned in the first two groups may arise.

4. *Patients born apparently healthy, acquiring evidences of syphilis within a year.* At birth these infants are sound, nurse well, cry lustily, and thrive. Sooner or later, in seventy percent. of the cases between the third and eighth week, and in ten percent. of the cases between the third and twelfth month, mild, followed by severer symptoms arise. At first the infant's desire to nurse lessens, it ceases to gain, begins to lose in weight, cries, gets hoarse, and has snuffles. Next an eruption appears similar to that noted in the previous group. Adenopathies develop, the digestion begins to suffer, and loss in weight goes on more rapidly. The crying increases and hoarseness grows more pronounced. Bony swelling and tenderness, keratitis,

neuritis of the auditory branch of the eighth nerve may develop. The liver and spleen enlarge. Subject to the age of the infant and the intensity of the digestive disturbance, these symptoms abate or lead to death, unless treatment is timely. The Wassermann reaction is present. According to Knoepfelmacher and Lehndorf, however, this is not the case before the appearance of the rash. This form of the disease is practically always tertiary, or at least very late secondary.

From this point on, cengenital syphilis is invariably tertiary in type. The systemic evidences are not generalized, but tend to be selective, picking out organs in widely separated regions. The disease now acts precisely as does late syphilis in the acquired form. It develops a peculiar affinity for the bones, skin, cerebrospinal axis, liver, spleen, and glands.

5. *Patients born apparently healthy, acquiring evidences of syphilis between the second and seventh years.* For the first years of its life the child develops normally, or constantly remains somewhat behind the average in physical and even mental progress, but is not actually ill. In the second year, it becomes indolent, makes a rhachitic impression, but lacks the rosary, or it may have actual rickets in conjunction with syphilis. The fontanelles close slowly, or the cranial bones are thinned to a papery consistence in some areas, or thickened into bosses through periosteal infiltration in others. The first of these conditions is called craniotabes; the second, Parrot's nodes. Instead of developing as in normal children, the bridge of the nose remains flat, the tip growing. Thus, this organ acquires the shape of a saddle, through failure to develop, or through the destruction of the small bones composing its structure, chiefly the nasal spine of the frontal bone, the vertical plate of the ethmoid, and the crest of the nasal bones. As a sign of congenital syphilis the saddle nose possesses no value before well on in the second year, as all infants are more or less wanting in the nasal bridge, a fact that Knoepfelmacher used to emphasize to his students. But from the second year on, the sign is of great corroborative value. Other changes in the bones and joints are also significant. The long bones are peculiarly vulnerable, both periosteum and epiphyses being affected. Involvement of the former leads to thickening and deformity of the shaft, of the latter to thickening and deformity of the ends, causing the outlines of the joints to appear abnormal. The tibia is often bent forward so that its convexity is increased and the anterior margin is sharp but roughened. This gives it the general appearance of a

sabre, and on palpation the irregularities of the anterior margin and surfaces may be detected. Roentgenographs reveal all these changes. In the early stages of involvement these alterations cause pain, again giving the symptoms of pseudoparalysis, named after Parrot. Tension of the muscles on the weakened scapula causes abnormalities in its shape, exaggerating its convexity and producing the well known scaphoid scapula. Gummas of the sternoclavicular junction, sternum, and osteoperiosteal cutaneous gummas elsewhere are frequently seen.

The skin and mucous membranes are the site of mucous patches, gummas, circinate, tuberocircinate, or tuberoulcerous lesions and fissures at the mucocutaneous junctions, besides other changes already described.

In the central nervous system and organs of special sense the commonest symptoms are produced by meningitis, pachymeningitis, and hydrocephaly, but tabes, spastic paraplegia (Little's disease) and hemiplegia, and juvenile general paralysis are known. Some cases of epilepsy, mental deficiency, imbecility, and early insanity are due to congenital syphilis. By far the most frequent changes in the organs of special sense are found in the eye and ear. Keratitis, both interstitial and parenchymatous; irodochoroiditis and auditory nerve neuritis are the commonest, but optic nerve neuritis, retinal pigmentation, and deafness due to gumma in the auditory centre, are met.

The liver practically invariably, and the spleen somewhat less frequently, are enlarged, usually because of chronic interstitial inflammation. At times, however, gummas may develop in the liver, and rarely parenchymatous hepatitis. The last mentioned condition, however, rare as it is, is almost unknown after the first weeks of the congenital syphilitic's existence, when it causes jaundice and other symptoms referable to the liver. The lymphatic glands act as they do in late acquired syphilis. Isolated enlargements are found, with a relative but not constant predilection for the epitrochlears.

6. *Patients born apparently healthy, acquiring evidences of syphilis after the age of seven years.* It is from this point on, or after the time of the second dentition, that Hutchinson's triad, two elements of which have already been considered, comes into prominence. The triad consists of keratitis, or scars resulting therefrom, deafness, or other definite evidence of eighth nerve disease, and the presence of Hutchinsonian teeth. The first two of these have been sufficiently discussed. By the term, Hutchinsonian teeth, is under-

stood an alteration in the shape and position of the upper incisors of the second set. They converge, their distal margin is shorter than their base, and their edges are notched from corner to corner by a semilunar concavity. The corners themselves are somewhat blunt, and the anterior surfaces of the teeth are often ridged vertically. Although highly suggestive of congenital syphilis, this sign is by no means pathognomonic. Other conditions, notably rickets, may produce similar alterations, while syphilis itself may produce other varieties of dental dystrophy. In addition to the triad, in the retarded variety of congenital lues, all the other conditions enumerated in the previous group may be present, and this period coincides with life. The very first symptoms of the disease have been known to occur as late as the end of the second, or early in the third decade, and all changes which are characteristic of tertiary acquired syphilis may be found in syphilis congenita tarda. The Wassermann test varies according to the laws governing this phenomenon in acquired lues, but all in all it is far more likely to be positive than negative.

Having described, as far as possible, congenital syphilis according to its picture at given ages of the patients, and in given periods of the disease, it will perhaps be not amiss to refer categorically to its common individual manifestations, and to some of the rarer ones.

The skin lesions are in the main identical with those seen in the acquired form. Two points, however, merit emphasis: first, bullous syphiloderms are not uncommon, and are usually seen at or soon after birth, chiefly on the palms and soles, in the form of the so-called pemphigus of the newborn. Secondly, eroded papules are frequently found on the nates, which are of peculiar significance, since a nonspecific dermatosis closly simulates them. The latter is the erythema gluteale of Leiner, first described by Parrot, in 1877. It is characterized by the presence of erythematous macules, vesicles, or papules, and it sometimes resembles pemphigus neonatorum. The lesions rapidly become eroded. Jacquet, Hallopean, and Sevestre pointed out their nonluetic nature, Jacquet calling the vesicular variety erythème vesiculeux érosif, and the eroded form syphiloïde post-érosif. No spirochetes are found in the lesions and the Wassermann reaction is absent. Leopold has recently reviewed this disease, calling it the napkin eruption, and he was able to prove conclusively that it was caused by alkalies remaining in the napkins after washing with cheap soap, inadequately rinsed out. Gummas,

wrinkling of the skin, onychia, paronchia, and alopecia are often present. In all these congenital syphiloderms the Wassermann reaction is present.

The lymphatic glands present changes not differing from those of acquired syphilis. Abnormalities in the testes, thymus, thyroid, and pancreas are rare, but in the suprarenal glands somewhat more frequent, causing symptoms referable to these particular organs. The spleen is enlarged in about sixty-two percent. of all cases at all ages, and the liver is increased in size almost without exception. Often, too, there is jaundice, and at least one form of icterus of the newborn is syphilitic. Usually the cause of this is interstitial hepatitis, but in five percent. of the cases gummata are found at autopsy.

Although the changes in the bones and joints have already been discussed, the following statistics may be of interest. Still found epiphysitis in twenty percent. of the cases during the first three months of life; dactylitis in two percent.; tibial periostitis and craniotabes in forty-seven percent.; and synovitis and arthritis in seven percent. Marfan includes congenital syphilis in the causes of rickets. Gaucher and Lévy Bing, in an analysis of 347 children with Pott's disease, cold abscess, and coccyditis, found that, of fifty-eight children in whom there was reason to suspect congenital syphilis, actual evidences thereof existed in sixty-five percent.

Among unusual central nervous manifestations precocious tabes must be mentioned, encephalitis, gummas of the brain and cord, juvenile general paralysis of the insane, and Little's disease, while Mott mentions two cases of endarteritis of the circle of Willis, associated with pachymeningitis. He also states that seven percent. of all cases of epilepsy may be ascribed to congenital syphilis, and that hyprocephalus is also frequently due to this cause. The cerebrospinal fluid in these cases obviously has the same characteristics found in central nervous diseases caused by acquired syphilis. All types of mental disturbances are possible, the commonest being backwardness.

The eye, as already mentioned, shows changes in the cornea, iris, and retina. The iritis is often bilateral, and somewhat more frequent in girls than in boys. Atrophy of the optic nerve, and cataract are rare. The most common affection is interstitial keratitis. With the exception of auditory neuritis and otitis media, there are very few diseases of the ear.

Cardiovascular and blood diseases are, on the whole, uncommon. Among these are arteriosclerosis, aortitis, myocarditis, and

endocarditis. Secondary anemia is not infrequent and one form of hemorrhagic disease, syphilis haemorrhagica neonatorum, characterized by icterus and bleeding from the mucous surfaces and umbilicus, is occasionally encountered. This appears to be due to parenchymatous hepatitis and is always fatal.

The respiratory phases of the disease are Virchow's white pneumonia, necrosis of the nasal septum and hard palate, frequently with perforation, laryngitis in fourteen percent., and snuffles in seventy percent. of the cases.

The Wassermann Reaction is almost invariably present. According to Noguchi it is present in ninety-six percent. of all cases, and this author's compilation of the work of other investigators raises the figures to ninety-eight percent. In a very careful study published by Mark Reuben ninety-nine percent. were found positive in the early stages of the disease, and fifty percent. in the later, about paralleling the figures respectively in secondary and tertiary acquired syphilis. During active congenital syphilis the reaction is never absent. It is only during periods of latency and at the onset or waning of activity, that negative results are found.

Summary. So concentrated an outline as this perforce had to be, could barely accomplish more than to sketch in the essential features of congenital syphilis. It is hoped, however, that several points have been sufficiently emphasized to eradicate its unnecessary traditional complexities. Congenital syphilis is syphilis acquired during intrauterine life through the placenta, which after having become infected, plays the part of the primary lesion. From this point the umbilical vein conveys the spirochetes to the fetus, the liver being the first organ involved. Thence the spirochetes are rapidly disseminated throughout the fetus. Here all differences between congenital and acquired syphilis disappear. Subject to the month of fetal infection, the infant presents cutaneous and systemic evidences of the disease in its secondary, latent or tertiary stages, and the tertiary stage may be protracted for years, or tertiary changes late in adolescence, or early in adult life may be the first sign of the congenital infection. Remarkable as such facts may appear, they need cause no astonishment, for in acquired syphilis we see, frequently enough, examples of freedom from secondary manifestations, the first reappearance of activity occurring twenty years or more after the primary lesion, in the form of tertiary changes. These facts serve only to prove the identity of congenital and acquired syphilis.

In conclusion, one more circumstance must be recorded, and this

depends not upon the disease, but upon the age at which the disease is acquired. Congenital syphilis is transmitted to its victim during the most important period of development, namely, before birth, and the struggle to overcome this burden takes place partly before, and partly soon after birth. Thus, aside from its actual pathological alterations, the disease may cause all sorts of anomalies of development, physical deformities and dystrophies; and mental backwardness, if not actual imbecility, or even insanity. Aside from these considerations, prenatal syphilis and postnatal syphilis are identical.

To recur for a moment to the mode of infection. It has already been stated that the mother must always be syphilitic in order to have the disease transmitted to the offspring. At times it is difficult to explain the infection, the father having had no active local manifestations, and perhaps no general ones, or only a Wassermann reaction. It is conceivable, in view of Warthin's work, that spirochetes may have been inactive but present in the testes and that the mother may have been infected by the seminal fluid containing micro-organisms. This does not imply that the spermatozoa conveyed the pathogenic agent.

Prognosis and Treatment. In general the outlook for a syphilitic baby is not good. In patients born with cutaneous and marked general evidences the prognosis is bad. The treatment consists of arsphenamin injections of one to ten milligrams a week, into the jugular vein, and daily rubbings with a gram of ammoniated mercury salve (U.S.P.). In patients with cutaneous and slight systemic evidences the prognosis is better, and the treatment as above. In patients with cutaneous lesions alone, and later systemic manifestations the outlook is still better, and the treatment again the same. When apparently healthy infants develop symptoms within a year, the prognosis is good; the treatment as outlined, save that gray ointment may be used instead of ammoniated mercury, the dose being the same. When the manifestations arise between the second and seventh years, the prognosis becomes that of acquired syphilis. The treatment consists of all the methods outlined in the chapter on treatment of acquired syphilis, but the dose of the arsenical solution should not exceed 0.1 a week, the site of the vein employed depending upon the child's build and tractability, and the choice of mercurial injections or inunctions depending upon these factors too. The dose of mercury injected should not exceed a half grain, nor the quantity rubbed in one and a half grams. In late congenital syphilis the prognosis and treatment absolutely parallel conditions in late acquired syphilis.

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