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SYPHILIS

A TREATISE FOR PRACTITIONERS

BY

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WITH SIXTY-NINE ILLUSTRATIONS IN THE TEXT AND NINE PLATES, SEVEN OF WHICH ARE COLORED

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FOREWORD

A WEARIED medical public, already surfeited with text-books, monographs, and essays upon syphilis and kindred matters, may well ask in resentful indignation: Why this new work upon syphilis?

Being the father of the writer and the godfather of many of the generalizations advanced, I think I may answer.

The facts upon which the volume rests are the classified cases from the private office books covering forty years of continuous work by myself along syphilological lines, ten years of work in the same field by my son, and some gleanings from the twenty extra years included in Dr. Van Buren's case books, which the office has inherited—making a retrospect of sixty years.

In this considerable mass of experience there is much matter of value that would have remained buried but for the resurrection afforded it by this volume.

The generalizations, based upon the statistics above referred to, corroborate quite uniformly similar generalizations of other authors often based upon statistics numerically superior; these latter statistics being sometimes largely from the hospital and dispensary point of view.

In certain of the views advanced, where this treatise differs from the majority of text-books, I am willing to share the responsibility with my son.

E. L. KEYES.

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PREFACE

In order to make this work essentially a practical one, it has been founded upon the 2,500 cases of syphilis observed and adequately recorded in our office case books during the past forty years. The number of these cases might have been many times multiplied by the compilation of dispensary records; but this would only have confused the picture. For the records taken of charity cases are so hasty and inaccurate, their histories so vague and incomplete, their disease so complicated by alcoholism and privation, their adhesion to any one clinic and course of treatment so brief and intermittent, that it is not fair to compare them with cases that have been, in the main, intelligently treated and watched for a number of years.

Yet it is perhaps too much to say that even these cases have been intelligently treated. All have received treatment at our hands, but a large minority have received most of their treatment, and especially of their important early treatment, at the hands of others. That the result leaves something to be desired may be inferred from the forty per cent incidence of tertiaries we have to record. Yet this result, which makes our cases more interesting, if less creditable, represents fairly well the status of syphilis in the middle and upper classes in New York, and in some measure throughout the United States, during this term of years.

During the last decade an increased appreciation of the horrors of syphilis, and a new method—the intramuscular injection of mercury—for its control, have certainly lessened the severity of the disease, and we may feel assured that, even without the

serum diagnosis, immunization, and therapy, which the future seems to promise, syphilis during the next forty years will be far less destructive, because far more intelligently treated, than ever before.

Be that as it may—and it is very difficult even to-day to define accurately in what good treatment consists and to distinguish which case has received it—the contemplation and tabulation of these cases has proved most valuable, not so much for the statistics derived from them as for the point of view they have enforced and elucidated.

This point of view, in large measure inherited from and constantly inculcated by my beloved father, is his contribution to this work; and if the development of syphilology has spread these chapters over many more pages than those of his earlier work upon the same topic, the spirit of the two is the same. I can but regret that the advance of science permits me to incorporate so little of the letter of that work, and hope that thereby the text has not lost all its interest.

Whether the new theories that have poured in upon us since the discovery by Schaudinn of the *Spirocheta pallida* will destroy this point of view; whether it will prove, for instance, that, after all, there are "periods" in syphilis, or that "paternal heredity" is possible, matters not at all. With this discovery we see the dawn of a new era of syphilology; we hope for complete mastery of the disease; we pray that we may live to see the day when syphilis shall be reduced from a volume to a chapter, from a scourge to a mere annoyance. Yet we fear that many a year shall pass ere some such volume as this one shall cease to be of practical interest to the student and the practitioner.

Such is the excuse for the publication of this book—to express practically a view of syphilis that has been two generations in the making, and to elucidate this by American statistics. Concerning the matter herein set down, it is impossible to be egotistical. The world's great authorities, Fournier, Neumann,

Lang, Hutchinson, Finger, and Taylor—to mention only a few to whom a special debt is due—have been drawn upon freely for information, and reference upon disputed points has been made for the convenience of the student to the latest, the most comprehensive, or the most accessible monographs. Thus we hope to spare the reader aimless divagations among the literature, and ourselves the laborious sciolism of copious bibliography.

The many chapters on special subjects have been a source of grave concern, lest the writing of all of them by the same hand should sacrifice accuracy of matter to uniformity of manner. But my friends have kindly come to my aid. With a courtesy for which I cannot be too grateful, Dr. L. M. Hurd has reviewed the chapters upon Syphilis of the Nose, the Throat, and the Upper Air Passages; Dr. E. G. Zabriskie, those upon Syphilis of the Nervous System; Dr. J. I. Middleton, that upon Syphilis of the Eye; and Dr. John Howland those upon Hereditary Syphilis.

Finally, while including in my general debt of gratitude all those whose writings or whose illustrations upon syphilis I have been able to lay my hands on, I must acknowledge a more precise indebtedness to the many others who have lent kindly personal aid to the matter in this volume: to my father, *facili principi*; to my associate, Dr. Chetwood, for privilege to tabulate cases observed by him, as well as for the keen interest with which he has shared the labor of testing various methods of treatment; to Drs. G. H. Fox and J. A. Fordyce, for many original photographs; to Drs. H. G. Piffard, J. Lincoln Davis, Prince A. Morrow, E. L. Shurley, and W. A. Pusey for illustrations; to Dr. W. R. Bross for his contribution on Syphilis and Insurance; to Dr. E. W. Caldwell and Dr. L. G. Cole for skiagraphs; and to Dr. C. H. Jaeger and Dr. L. E. La Fetra for the loan of cases from whom these skiagraphs were taken.

109 East Thirty-fourth Street,
New York City.

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SYPHILIS

CHAPTER I

SYPHILIS IN RELATION TO PUBLIC HEALTH

SOME fifteen years ago there appeared for the first time in America a book on syphilis,[1] the last chapter of which bore the above title. Fifteen years ago the science of preventive medicine was in its infancy and venereal sociology did not exist. Yet Dr. Armstrong then said: "The relation that any disease bears to the public health is in a direct ratio to its prevalence and its preventability."

In the intervening years evidence of the appalling prevalence of syphilis has been accumulating with ever-increasing force; and if evidence of its preventability is lacking, this is due to the prudishness of our morality, the inherent sluggishness of public opinion, and in no small degree to the unwillingness of medical men to take their part in the crusade.

But at last we begin to see light. The International Society of Sanitary and Moral Prophylaxis, with its national and local branches, is the first fruit of an awakened public appreciation that there is work to be done in the prevention of social diseases in general and of syphilis in particular. Thus this question of the prevalence and the preventability of syphilis, which formed an almost surreptitious last chapter a decade and a half ago, now very properly usurps the initial position so long held by fruitless disquisitions on the history of syphilis—disquisitions as intimate to the practical study

[1] Morrow's "System."

of the disease as a lecture on colonial architecture when the townhall is afire.

The three important points to be considered are the prevalence of syphilis, the effect of syphilis upon the community (syphilis and marriage), and the prevention of syphilis.

THE PREVALENCE OF SYPHILIS

The prevalence of gonorrhea is generally accepted. The current statement that some sixty to eighty per cent of young unmarried men suffer, at one time or another, from it has almost ceased to evoke comment. Gynecologists have come to agree that sixty to eighty per cent of pelvic suppurations requiring hysterectomy or oöphorectomy are due to gonorrhea, and to recognize that no class of society is spared by this terrible scourge. But the public mind is not yet enlightened concerning syphilis. This disease is considered so disfiguring that people will not realize how their fellows, whom they see daily bright-eyed and clear-skinned mingling with the rest of mankind, can yet be syphilitics. Tell the man in the street that some ten per cent of the inhabitants of Berlin or Paris are syphilitic, and he will see visions of poverty and filth; he will not believe that the upper classes form an appreciable proportion of the sufferers. Although we recognize that many men of the higher class have gonorrhea at least once in their lives, we must, nevertheless, concede that they acquire the disease much less frequently and suffer from it far less intensely than do the lower classes. The same is true of syphilis. It, undoubtedly, strikes less frequently and less sharply the man protected by wealth and position than the grimy slave of toil. Yet no one is immune. Among our office records, which go back half a century, I find the proportion of syphilitics to gonorrheics varies from 1–8 to 1–15, and this I take to be not far from the true proportion among

the middle and upper classes of men in New York City. Fournier is, if anything, more pessimistic in his oft-quoted statement that, among the syphilitic women who come to his office, one in five is a wife infected by her husband.

Perhaps as good evidence as can be obtained of the influence of social position in determining the incidence of syphilis is that afforded by the Report of the Surgeon General of the Army.[1] In 1904 the proportion of syphilitics among the enlisted men (white) was 27.83 per mille; among the officers, 2.78 per mille (just one tenth). The ratio of syphilis to gonorrhea among the men was 1–4; among the officers, 1–1; among the negroes, 1–6; the incidence of syphilis, per mille per annum, being among the white men, 35.11; officers, 3.51; colored men, 13.78.[2]

This same report shows the prevalence of venereal diseases very strikingly. "By far the most important diseases affecting the efficiency of the army during the year have been the venereal . . . causing sixteen per cent of all admissions, twenty-eight per cent of all non-effectiveness, and eighteen per cent of all discharges for diseases."

The number of syphilitics treated during the year 1904 was 1,996, and the disease stands fifth on the list, preceded by gonorrhea (7,106), malaria (3,953), bronchitis (3,091), influenza (2,485), and closely followed by chancroid (1,961) and alcoholism (1,498).

But if we measure the importance of a disease by the damage it does, syphilis immediately jumps into far greater prominence. Taking as a criterion the "number of days sick," we find syphilis with 70,398 days second only to gonorrhea with

[1] The venereal statistics of the navy are bound always to be singularly bad on account of abnormal social conditions.

[2] It may be noted that the two chief causes for rejection of recruits were, per mille, for whites, defect of development, 120.08; venereal diseases, 100.46; for colored, venereal diseases, 170.78; defect of development, 98.68.

146,609, and well ahead of dysentery (49,518) and tuberculosis (49,195). Although only responsible for 2 out of 233 deaths from disease that occurred during the year 1904, syphilis stands first of all as a destroyer of careers, causing 166 discharges (2.76 per mille), whereas tuberculosis caused only 101. This fact alone would be enough to mark syphilis as one of the great scourges of the human race. There are more soldiers rendered permanently unfit to follow their profession by syphilis than by any other disease.

SYPHILIS AND MARRIAGE

The very importance of syphilis in the army is enough to condemn it in certain minds to classification as a righteous punishment for defiance of the moral law. Yet this is precisely not the case. The syphilitic is a constant menace to his fellows, whom he may infect accidentally at any moment; and this danger is greatest to those nearest and dearest to him, to his mother or sister and, above all, to the wife he marries and the children he begets. Any syphilologist can cap the following incidents that have come under my observation:

Case I.—A young and upright man kisses a girl at an evening frolic. Shortly after he becomes engaged to marry another girl. A supposed fever sore upon his lip causes him no anxiety until a similar sore appears upon the eyelid of his betrothed and an eruption breaks out upon his own body. Then to the family physician, who finds both syphilitic.

Case II.—A man acquires a chancre upon the lip in some way to him unknown. His malady runs a very mild course, and he consults no physician, remains ignorant of the nature of his disease, and soon marries. A year later a child is born at term, but dies in ten weeks of syphilis. A second child dies at three years of age, also syphilitic. Meanwhile the husband, who has had nodes on his skull and cerebral symptoms, has undergone a mild course of treatment, just enough to cure his symptoms.

Then the mother, who has never shown any sign of syphilis, becomes pregnant again. She is put on mercury during gestation, and bears a healthy child. When this child is two and one half years old, five years after infection, the mother shows the first sign of the disease, a naso-pharyngeal ulceration that carries away some of her septum and continues for seven years, during which time she is pretty constantly under treatment and bears another healthy child. Meanwhile the husband has necrosis of his septum, submits to treatment for three years, and remains well when last seen, eleven years after the cessation of treatment and twenty-eight years after the beginning of the disease. Not so the wife; she continues to bear healthy children, and remains well from the twelfth to the twentieth year of her disease. Then it attacks the tibiæ, the heart, the frontal bone, the thumb. Five years later what there is left of her no longer shows active symptoms of syphilis.

Nor is this all. Even before the first child died, its mother's milk gave out, and her sister, who was nursing her own child and had milk to spare, offered to take the infant until a wet-nurse could be obtained. She nursed it ten days. In a few weeks her nipple got quite sore, and later she had a little sore throat. She took no treatment, and infected her child, but not her husband. Her next child was born syphilitic, but, happily, was cured by treatment. She then took treatment, bore a healthy child three years after infection, and had no further symptoms herself.

Here, then, is syphilis innocently acquired by a citizen of the highest class. Before he discovers its nature he bestows upon his wife a disease which pursues her for a quarter of a century and leaves her disfigured and shattered, which kills two of her infants and one of her sister's. In sum, three deaths, at least one life ruined, and sickness and misery to four who did not die.

Such is *syphilis insontium*—syphilis of the innocent. These cases have been selected rather because they represent an infection acquired innocently than for any other reason. Similar ones are common enough, and among the ignorant

and impoverished widespread epidemics thus arise. Of the infections spread by midwives, by physicians, by dentists, there is no end. I have known an innocent wife to put her syphilitic infant to the breast of an equally innocent wetnurse, and when this woman became infected to drive her from the house and supplant her by another victim, who was infected in turn ere the mother learned the horror of the true situation.

And this is the unspeakable disease which the public will not hear named, much less publicly attacked. Truly the wonder is that *syphilis insontium* is so rare.

But it is rare, relatively speaking. It does not call for hysterical syphilophobia. It can be cured. Its transmission to the next generation can be prevented. The individual treatment of syphilis is well understood in our day. But its prophylaxis we are only beginning to study.

THE PROPHYLAXIS OF SYPHILIS

Individual prophylaxis is futile. As long as men indulge in illicit sexual intercourse, so long will syphilis exist. There is no preventing it. The condom is to-day, as it was in the time of Ricord, "a cuirass against pleasure and a cobweb against danger." Antiseptic washes have yet to prove their protective powers. Even cauterization of a torn frenum within six hours of the time of infection has twice (Hill, Ruggles) failed to prevent the subsequent appearance of syphilis (cf. page 32).

Yet many of our most aristocratic brethren insist that they do not haunt the dens where such diseases fester. They fancy that high price purchases immunity. No fancy could be more fallacious. A contemplation of the cases just recited may serve to impress the truth that it is possible to transmit the disease before you know you have it. And she who is kind to one may perhaps be generous to twenty in his absence.

In short, there is no individual prevention of syphilis. To control this disease it must be attacked as a plague.

Naturally, the first attempts at control have been directed against the prostitute. "Here is the *causa teterrima*; let us wipe it out." And since prostitution refuses to be eradicated from any large community, the effort has been made to control it, and war has waged for many years between the advocates of licensed prostitution and the purists who have been described in delightful mixed metaphor as "closing one eye to the existence of prostitution while trying to stamp it out with the other."

Up to the present our Puritan blood has kept us Americans from considering the subject sanely. We still cling blindly to the theory that prostitution may be suppressed, though the history of every century and every country proclaims that it never has been.

On the other hand, the adherents of reglementation are little better off. They find that, at the expense of much effort, time, and money, they can show no appreciable improvement in morals, no marked decrease in the incidence of venereal disease.[1] The causes of their failure are chiefly three.

First, they attack venereal disease, yet make no effort to improve the morals of the community, and so leave untouched the foremost of its causes.

Secondly, they examine the prostitute at most once a week, present her with a certificate of cleanliness; and, before she returns, she will (according to Parisian statistics) have cohabited with from three to five men a day. So that (accepting the infallibility of the hasty and routine examination) she has, toward the end of the week, risked contamination from twenty to thirty times. Manifestly such an assurance of cleanliness is not overeffective.

[1] Cf. the Report of the New York Committee of Fifteen, published under the title, "The Social Evil," by G. P. Putnam's Sons.

Thirdly, venereal disease can never be stamped out by the inspection of licensed prostitutes so long as unlicensed prostitution remains unsuppressed (and who shall suppress it?) and so long as the men are not examined as well as the women. Would the suppression of tuberculosis be taken seriously if only the tuberculous women were reported while the men were allowed to go scot-free?

The problem is a difficult one, and the solution is not yet; but, recognizing the prevalence of syphilis and of gonorrhea as well as the destruction of life and happiness they cause, it is surely essential that we, as medical men, should gravely consider not only the methods of their cure, but also the means of prophylaxis. These means are now being intelligently discussed throughout the country, especially in the various branches of the Society of Sanitary and Moral Prophylaxis.

The few fundamental principles that have been generally accepted may be summed up as follows:

1. Absolute continence does not, in man or in woman, impair sexual instinct or appetite, nor does it diminish the procreative power. Moreover, the effect of continence upon the general health is highly beneficial.

2. Apparent exceptions to this rule, cases of general or of sexual neurasthenia, impotence, etc., occurring in persons who do not run to sexual excess, may always, on intimate investigation, be attributed to one of three causes: commonly to some concealed secret vice, less frequently to mental incontinence (the most degrading of all forms of incontinence in that it prostitutes the mind to constant contemplation of vicious subjects while covering this inward rottenness with an outward veneer of physical cleanliness), and rarely to systemic abnormality or disease.

3. Conversely, sexual immorality of any sort, besides tending to disseminate venereal disease, lowers the morals and assails the general health; for

4. Sexual intercourse, though undeniably productive of a sense of physical well-being which renders it desired by all men and essential to many (once the habit is formed), is not comparable to such physiological needs as breathing air and eating food, but rather to smoking and drinking alcohol. Note, for example, that it is a pleasure which becomes a necessity only from the same intemperate indulgence that makes alcohol or tobacco a necessity. Note, also, that the hold it gets on most men more closely resembles the morphin or cocain habit than the milder intoxications of alcohol and tobacco; hence *the only hope of physical purity for most men lies in avoiding the first misstep.*

5. Consequently the most important prophylactic measure is the protection of childhood; not the futile protection of assumed ignorance, but the protection of intelligent instruction from a respected source, individual (parental, if possible) at first, perhaps collective later (not by books or tracts), and advancing in accord with the awakening instincts of the individual.

6. Moreover, it is the paramount duty of the physician to instruct every patient treated for venereal disease concerning the grave dangers of infection, especially in matrimony.

Such are some of the fundamental moral principles upon which the prophylaxis of syphilis must be founded. The application of these principles to the needs of children, of factory and shop girls, of the army and navy; the passage of laws requiring that venereal diseases be classified as infectious and reported to the board of health; the provision of adequate hospital facilities for venereal cases in place of the present refusal of hospitals to accept them; the renewal of the army canteen, the withdrawal of which has been followed by a progressive increase in venereal disease;[1] such practical matters we may

[1] The incidence of syphilis is highest in the English army (75 per mille, per annum). Then follow the United States (33.98), Austria (19.2), Russia

hope to see thoroughly thrashed out in the future. In the meanwhile it is our duty to present the facts to the laity and to animate discussion among them, while protecting to the best of our ability the innocent wives and children of our venereal patients.

(12.8), France (6.7), Holland (4.9), Bavaria (4.3), Prussia (4.0). The prudish Anglo-Saxon refusal to countenance protective measures for the health of the army here shows its results.

CHAPTER II

THE GENERAL CHARACTERISTICS OF SYPHILIS

Syphilis (the pox) is an infectious disease, probably caused by the *Spirocheta pallida.* It is acquired by contagion or by heredity; it is chronic in course, indefinite in duration, essentially intermittent in character, manifesting itself by a succession of lesions which may involve any part of the body, and which are arbitrarily classified as primary, secondary, tertiary, and parasyphilitic.[1]

Syphilis is as varied in character as it is widespread in distribution. No country in the world, no organ in the body is exempt from its taint. In the detailed description of the disease there is some danger of losing amid a maze of minutiæ the main thread of the story; hence the need of a preliminary *résumé.* The ability to distinguish forty different syphilides, though it may permit an occasional brilliant diagnosis, is scarcely essential to a true knowledge of syphilis. For syphilis, whatever else it may be, is not a skin disease. The skin lesions of syphilis are as common and obvious as the foaming crest of an ocean wave, and, like it, they are but the outward and relatively insignificant manifestations of forces which human science has not yet measured. Syphilis is an infection of the whole man which, for intensity and pertinacity, has no rival but tuberculosis and leprosy. It shortens more lives than we can estimate, and its ultimate ravages are not yet known.

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[1] The classification of late sclerotic lesions (e. g., tabes, paresis, aortitis), as quaternary has been suggested, but is not generally accepted.

HISTORY OF SYPHILIS

Until the year 1494 syphilis, as we know it to-day, did not exist. Doubtless it was known to the Chinese, and perhaps it was imported into Europe by Columbus's returning squadron; the evidence for and against is conflicting and inconclusive. But this we know: modern syphilis was first recognized in the last decade of the fifteenth century, beginning among the soldiers of Charles VIII, King of France, in his campaign against Naples. Whether the disease was, as an ancient chronicler shrewdly guesses, "*non nova simpliciter, sed nobis tantum*," matters little. It spread rapidly over Europe, infecting every grade of society from king to peasant, and execrated as the *morbus Gallicus*, because of its supposed origin in the French army before Naples. The French themselves entitled it more gracefully *souvenir de Naples*, and it flourished impartially in many lands under a variety of names.

Whether the modern name of the disease means "with a girl" (*σὺν φιλία*), or "friend of a pig" (*σῦς φίλος*) depends upon one's point of view. At all events it was first bestowed by Frascatori (1483–1553) upon the mythical *pater syphiliticorum*, hero of a metrical venereal romance.

But no sooner did syphilis force itself upon the notice of physicians than they promptly confused it with gonorrhea and chancroid, regarding the three as different manifestations of the same disease. All doubts as to the unity of this venereal Cerberus were quieted for nearly a century by John Hunter, who, in 1767, inoculated gonorrheal pus on the glans and foreskin (whether his own or another's he does not state, though tradition makes him the martyr) and produced syphilis. The inference is obvious: this "gonorrheal" pus was syphilitic; perhaps both syphilitic and gonorrheal. But the inference drawn by Hunter and accepted by the world at large was that gonorrhea, syphilis, and chancroid were one and the same,

which error, matched curiously with the accurate description of what has since been known as the Hunterian chancre, has spread John Hunter's name farther than the conscientious labors that made him the father of English surgery.

The next—perhaps one might say the first—great name in the history of syphilis is Philippe Ricord. Born in Baltimore, December 10, 1800, Ricord carried out his lifework at Paris. He proved that gonorrhea and syphilis were distinct diseases, enunciated the theory of primary, secondary, and tertiary syphilis upon which the modern study of the malady is based, and cleared the way for Bassereau's distinction between true chancre and chancroid.

For fifty years more "unalists" and "dualists" fought it out, with the believers in "syphilization" (i. e., prophylactic inoculation against syphilis with chancroidal pus) inoculating themselves daily in the vain hope of producing immunity. But gradually the dualists gained ground until the advent of bacteriology and the successive discoveries of the gonococcus and the bacillus of chancroid have swept away the last vestige of hope that gonorrhea, syphilis, and chancroid can ever again pose as one and the same disease.

Such, briefly, is the history of our knowledge of syphilis. The more recent developments will be dwelt upon each in its appropriate place in our study of the disease.

ACQUIRED SYPHILIS

Syphilis is a contagious disease whose general characteristics are best studied in its "acquired" form. The peculiarities of hereditary syphilis may be subsequently discussed.

The disease may be acquired only by contact with a syphilitic sore or with some substance upon which the secretion of a syphilitic lesion has been recently deposited. The infection does not travel in the air, nor do rooms occupied by syphilitics

become infected. Moreover, the virus is probably incapable of piercing the intact integument.

The Primary Lesion.—Acquired syphilis always begins, after a few weeks' incubation, with an eroded papule at the point of inoculation. This lesion is called chancre, and is accompanied by a characteristic inflammation in the adjacent lymph glands. This chancre and adenitis constitute the *primary lesion.*

The Secondary Lesions.—A few weeks later the secondary lesions appear. These consist of typical exanthemata upon the skin and mucous membranes, acute inflammations of certain organs (iritis, periostitis, etc.), and evidences of general infection. The toxemia is often very light and generally lasts but a few weeks, while the localized secondary lesions have a tendency to relapse, after intervals of apparent health, for at least two years. These localized secondary lesions are infectious, superficial, and benign (i. e., tending to spontaneous cure and, generally speaking, not destructive of tissue nor productive of a permanent scar).

The Tertiary Lesions.—The tertiary lesions of syphilis may occur at any time after the appearance of the chancre. They may relapse after an interval of years. They rarely appear until after the first outbreak of secondary lesions has spent itself. In contrast to the secondary lesions they are clinically not infectious (page 36), are deep rather than superficial in location and malignant in that they destroy tissue and show little or no tendency to spontaneous cure. Moreover, they bear no special affinity to the skin, but impartially attack every organ. Histologically the tertiary lesion is either a specific syphilitic granuloma (gumma) or a diffuse interstitial sclerosis.

The Parasyphilides.—Finally, there is a class of lesions, partially syphilitic in character, termed by Fournier the parasyphilides. These lesions occur usually in persons who have

had syphilis, but after the other syphilitic lesions have ceased to appear. Yet they also occur in persons who are not apparently syphilitic; they are often entirely unmanageable by mercury and iodides (the antisyphilitic specifics) and, even when occurring in syphilitics, they are habitually in part due to other causes.

Thus the exact nature of the parasyphilides is not clear. Some of them, such as tabes, are almost exclusively due to syphilis; others, such as arterial sclerosis, are far from being exclusively syphilitic; while in all, the absence of lesions pathologically syphilitic as well as the resistance to antisyphilitic treatment render a decision still more doubtful.

Are there Periods of Syphilis?—The importance of these various syphilitic lesions to the patient varies inversely with their influence upon the community. The primary and secondary lesions are rarely sufficiently grave to be a serious inconvenience to the patient; yet they are actively infectious and a constant menace to the public health. The destructive, malignant tertiary lesions, on the other hand, are clinically not infectious, but may gravely disable their host; while the parasyphilides are even more evil in prognosis as far as the individual is concerned.

Happily, however, the tertiary syphilides may be prevented, or at least much ameliorated, by proper early treatment and the parasyphilides avoided by intelligent hygiene.

Finally, note that the above classification is an arbitrary one. The primary lesion is definite and immutable (though it may be overlooked), but beyond this all is variable. Secondary lesions may be so mild as to pass unnoticed. Tertiary lesions may never occur, or they may appear before the secondary, or the two may exist side by side; or, more confusing still, a given lesion may be on the border line, perhaps secondary, perhaps tertiary; and a superficial lesion, apparently secondary at first, may later develop the characteristics of ter-

tiarism. Moreover, parasyphilitic lesions may coexist with tertiary manifestations.

Hence there is no such thing as a purely secondary or tertiary period of the disease. Secondary symptoms; yes. Tertiary symptoms; yes. Periods; no. The terms secondary and tertiary are conventional symbols to express the quality of certain symptoms. It is utterly misleading to apply them to periods of time when these periods so overlap as to produce confusion from the use of terms whose only purpose is to prevent confusion.

HEREDITARY SYPHILIS

The worst horror of syphilis is the fear of passing it on to generations as yet unborn. For syphilis may be transmitted by inheritance from a syphilitic parent.

The laws that govern the inheritance of syphilis are not well understood, lacking as they do any scientific control and depending entirely upon clinical facts.

A syphilitic father may beget a syphilitic child without apparently infecting the mother; yet this mother cannot be infected with syphilis by nursing the child (Colles's law), although the child will promptly infect any other wetnurse. Moreover, the mother of such a syphilitic child, though herself remaining healthy many years, almost invariably ultimately breaks out with tertiary syphilis (*choc en retour*). Manifestly, therefore, the mother of a syphilitic child, even though she remain apparently sound, is in some way or in some degree syphilitic.

The danger of transmission from father to child ceases in from two to five years if the father is properly treated. Under inefficient treatment paternal virulence may last indefinitely, though it is likely to terminate within five years in any case.

The danger of transmission from mother to child does not, however, terminate at any definite time. Some mothers con-

tinue to bear syphilitic children for years after the disappearance of their own symptoms. That such cases are exceptional does not lessen their importance.

And the child itself. It may die *in utero* and be expelled as a fetid disorganized mass. It may be born to live but a few days. It may reach maturity, bearing in mind and body the scars of its parent's misfortune. It may remain well many years only to fall victim to an unsuspected " delayed hereditary syphilis." It may not be infected. Freaks of fortune and effects of treatment ring every possible change. Of twins even, one may be born healthy, the other syphilitic. But, generally speaking, the infection lessens with each succeeding conception. Thus, when a man in the infectious stage of syphilis marries, the first product of conception usually dies *in utero*, and miscarriage of a deformed, macerated fetus ensues. After one or more such mishaps a child is born, cachectic, perhaps actively syphilitic at birth or soon showing evidences of the disease. Such children usually die promptly. Later children are born which show no signs of the disease at first, and may either remain well or show certain stigmata of syphilitic heredity, or become actively syphilitic.

Hereditary syphilis is the same disease as acquired syphilis. But its lesions are modified by the undeveloped condition of the organism attacked as well as by the mode of infection. Whether the fetus is infected by father or by mother (the theories upon this subject will be discussed later), there cannot be said to be any port of entry for the virus (unless it be the placenta); hence there is no chancre, no primary lesion.

The secondary infectious lesions are sometimes skipped (or overlooked) in hereditary syphilis, and the so-called delayed hereditary syphilis is always tertiary in type.

But the overshadowing features of hereditary syphilis are the virulence with which it overwhelms the infant and the

characteristic developmental deformities it imparts, deformities especially of the teeth, the cranium, and the remainder of the skeleton.

CLINICAL TYPES OF THE DISEASE

So varied is the course of syphilis, so distinctive each individual case, that it is a thankless task to attempt any classification which will absolutely include all cases. But, if for no other reason than to show the futility of cut-and-dried prognoses, the following classification may be of service:

1. Mild early syphilis.
2. Malignant early syphilis.
3. Mild but persistent syphilis.
4. Relapsing syphilis.
5. Malignant late syphilis.
6. Mild late syphilis.

1. Mild Early Syphilis.—Young men in good health and with good habits usually suffer very little from syphilis if they attend closely to hygiene and treatment. A type case has chancre followed by one or two early skin eruptions, but without any marked general toxemia, and then nothing more, unless it be some tendency to relapsing, superficial ulcers of the mouth and throat for a year or two. The disease may end there; yet every experienced syphilologist has learned to dread such cases, for the good health of the patient during the active period of his disease and the absence of grave or disfiguring symptoms leads him to the conclusion that his syphilis does not amount to much and, in spite of all the physician can do to keep him to his duty, he is very likely to lapse from treatment, with the result that, whereas he might have cured his syphilis in the early stages had he been more attentive to its management, he neglects himself at this time and permits the disease to acquire that hold upon him which will later show itself as relapsing syphilis, or as malignant late syphilis.

Manifestly, therefore, the mildness of early syphilis is not an unmixed blessing.

2. Malignant Early Syphilis.—In contrast to these cases that go through the first two or three years of their disease without any real inconvenience, are those tragic ones in which the patient seems to be overwhelmed from the first by the poison of the disease. Within the past year I have seen six such cases, which are sufficiently typical.

Case III.—The first was a woman. Her outward symptoms were mild, nothing more than a light, papular syphilid; but she developed fever and lost weight progressively. These symptoms could not be controlled by her physician, and it was only after six months of fever and two months in bed that vigorous treatment finally succeeded in controlling the disease, which has caused no symptoms since. But in the meanwhile she had developed intestinal tuberculosis, and when I saw her, six years later, she had also a tubercular elbow and was, physically speaking, a shadow of her former self.[1] This type of malignant toxemia with few local symptoms is more common among women than among men.

Case IV.—Another case, a young man in robust general health, but a confirmed drinker, developed, within three months of the beginning of his disease, multiple ulcers of the skin, especially of his face, which made of him a horrible spectacle, required three months of the most vigorous treatment to cure, and left him scarred forever. Doubtless his addiction to alcohol occasioned the violence of the attack.

Case V.—In a third case the patient was fifty-five years old and developed, within three months, and while under regular treatment, first a double iritis, and immediately afterwards a "gumma" of the iris with iridochoroiditis, which threatened at one time to cost him his eye, and which was only controlled by treatment so vigorous as to salivate him for three or four weeks. Immediately thereafter he developed diarrhea, and is now dying of rectal carcinoma.

[1] During all this time she was under vigorous antisyphilitic treatment; she came to me vainly trying to cure her elbow by passive motion and calomel!

Case VI.—The fourth case is the most striking in that it occurred in a young and healthy woman (infected by her husband), in whom no predisposing cause could be alleged and whose general health before the attack seemed perfect; but who was rather lightly treated at first because her symptoms were insignificant, until, at the end of about one year, she developed an ulcer on her right tonsil, then an ulcer on the left one, then rapid gangrene of the whole soft palate and of much of the lateral pharyngeal walls, then gumma of the base of the tongue and necrosis of the lower jaw. To control this outbreak mercury had to be administered to the point of salivation, iodides to the limit of toleration. She was delirious for days (from iodism), unable to eat solid food for weeks. The attack lasted six months.

Case VII.—In the fifth case the termination was not happy. The patient had mild syphilis for six months; then his wife noticed that he dragged his left leg. Two days later he was suddenly paralyzed on the left side. Taken to a hospital and vigorously dosed for two months, he regained partial control of his muscles, but had sunk to imbecility. A month later I saw him, feeble, inarticulate, drooling, and paralyzed, with a rapid, feeble pulse, and a slight temperature. Ten days of mixed treatment left him worse than before, and a few days later he died with hyperpyrexia, apparently due to encephalitis.

The sixth case is cited elsewhere (Case XXVII, page 231).

3. Mild but Persistent Syphilis.—This class includes the greatest number of cases. The patients do fairly but not absolutely well under treatment. They recover from one set of lesions to remain well only a few months, and then plunge into another. Often the cause of the persistence of the disease is quite manifest. It may be alcohol, malaria, tuberculosis, bad hygiene, or inability to take a sufficient amount of mercury or iodides. Such cases may potter along year after year, more or less sick, to their own despair and to the disgust of the physician who treats them. But patient care and the application of every device of treatment—although it may not be followed by brilliant results at the time—will slowly wear down

the patient's disease, and in the end he is likely to be permanently cured, perhaps more likely than the brilliant case of mild early syphilis.

4. Relapsing Syphilis.—All syphilis is relapsing syphilis. It breaks out when or where it will, and no man shall say when it is finished. One may cruelly but truthfully say to the patient who asks for an absolutely accurate prognosis of his disease, "I will tell you whether your syphilis is grave or not after you are dead." Prognosis, in a certain sense, we can assuredly give; but swear and sign and seal, we never can.

5. Malignant Late Syphilis.—The patient who, after years of perfect health, develops locomotor ataxia or is stricken down by an apoplexy or develops a bone gumma is a type of malignant late syphilis. The caption is employed here only to drive home the truth that such accidents may happen at any stage of the disease (cf. Cases XVIII, XIX, and XXI, pages 98, 101).

6. Mild Late Syphilis.—As a rule, however, if syphilis relapses after many years' interval, it is in some relatively mild form: a tuberculo-squamous patch, a chancre redux, a tibial node, or a tongue ulcer. Prompt treatment dissipates the lesion and, if treatment is kept up thereafter for a year or so, another relapse need not be expected. Yet, if treatment is neglected, any relapse, however mild at first, tends to progress and to invade other organs of the body until it becomes grave indeed.

CHAPTER III

ETIOLOGY OF SYPHILIS

Syphilis has long been classed among infectious diseases, and its manifest contagious characteristics have made it seem certainly parasitic in nature. Some twenty or thirty alleged bacterial causes of the disease have been described, among which, up to the year 1905, the most important claimant was the bacillus of Lustgarten, a microbe closely resembling, if not identical with, the smegma bacillus. But in May, 1905, Schaudinn and Hoffmann, while endeavoring to identify the *Cytorrhyctes luis* (Siegel [1]), one of the alleged causes of syphilis, observed a hitherto undescribed organism in the secretions from syphilitic sores. The publication of their findings was immediately followed by confirmatory evidence from all parts of the world. Metchnikoff and Roux not only confirmed the observation, but called attention to the fact that Bordet and Gengon had previously observed this microörganism in certain syphilitic secretions, but not finding it constantly had dropped further investigation. Levaditi found it in lesions of congenital syphilis. Bertarelli, Volpino, and Bovero were able to identify it in stained sections of syphilitic organs. Larrier and Bergeron have recently identified it in the blood of syphilitic patients, and innumerable observers in every country have noted clinically that the organism may be found in almost every lesion of early untreated syphilis, though most common in the most infectious lesions.

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[1] Münch. med. Wochenschr., 1906, vol. liii., p. 2.

So overwhelming indeed is the mass of evidence that, in spite of the fact that the organism has not been cultivated, it seems certain that the cause of syphilis has at last been found, and we may safely say that the spirocheta of Schaudinn is either the cause of syphilis or one phase in the life cycle of some microörganism which is the cause of syphilis.

THE SPIROCHETA PALLIDA

The *Spirocheta pallida* (*spironema, treponema*[1] *pallida*) best shows its characteristics when examined alive in the secretion of an infectious syphilitic lesion. It may be examined in a hanging drop or, better, mounted under a cover glass and sealed with wax or paraffin.

It is a spiral organism (Pl. I), varying in length from 4 to 14 μ, in diameter from an immeasurable thinness up to about $\frac{1}{4}\,\mu$; it is cylindrical, not flattened; its spirals usually number from 6 to 14, though as high as 20 and 25 have been counted in exceptionally long ones. The length and depth of the spirals varies from 1 to 1.5 μ.

It is a motile organism, having three modes of motion; viz., a corkscrew rotation on its own axis, a gliding back and

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[1] The name "treponema" was suggested by Schaudinn as more accurately descriptive of the exact nature of the parasite. But this name has met with no general favor and the special features upon which Schaudinn based his preference are still contested. Indeed, so little is yet known about the spirochetæ, that it is impossible to place them as a class, still less to determine accurately the relative positions of individual members of that class.

Whether *S. pallida* is a bacterium or a protozoön, whether it divides by longitudinal or by transverse fissure, whether its flagella are real, whether it has an undulating membrane and nuclei, the future must decide.

The most recent contributions summing up our present knowledge of *S. pallida* are: Sobernheim, *Hanb. d. Path. Microorg.*, 1907, No. 2, p. 527 (with eleven pages of bibliography) and in American literature, Rosenberger, *Am. J. Med. Sci.*, 1906, vol. cxxxi, p. 143; Pfender, *Am. Med.*, 1906, vol. xi, p. 350; and Ewing, *N. Y. State J. of Med.*, 1907, vol. vii, p. 177.

forth, and a bending upon its axis. In fresh specimens these motions are very active, but do not appear to entail any material progress from one spot to another, for a specimen remains for hours in the microscopic field.

It is a peculiarity of *S. pallida* that, whether in motion or at rest, it retains its spiral shape.

Although fixing and staining, of course, destroy its activity and somewhat distort its spirals, so tenuous an organism cannot be accurately studied unless stained. Under these conditions it shows a tapering at each extremity, which is sometimes adorned with one or two flagella and a tendency to occur in irregular, overlapping, or star-shaped groups. The spirochetæ often lie adjacent to or overlap red blood corpuscles. They are practically always extracellular.

Methods of Staining.—The *Spirocheta pallida* approximates the protozoa in its resistance to the usual bacterial stains. The classic method of preparing and staining is Schaudinn and Hoffmann's modification of Giemsa, as follows:

1. Obtain the specimen by scratching the surface of the lesion (chancre, mucous patch), after having washed it thoroughly clean of all contamination [1]—the admixture of a trace of blood does no harm. Spread the secretion thus obtained as thin as possible; *dry without heating*; harden for fifteen minutes in absolute alcohol.

2. Employing the Giemsa stain (made by Grübler, of Leipzig), dilute this by adding about one drop to 1 c.c. of water (to which one to ten drops of 1 : 1,000 calcium carbonate has been previously added—this is advantageous but not essential).

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[1] Rille and Vockerodt found that antiseptic wet dressings eliminate mixed infection without affecting the spirochetæ. Inasmuch as the spirochetæ lie among the epithelia rather than upon them, the best results are obtained by scraping the lesion quite deeply. The fewer leucocytes in the exudate the more spirochetæ it is likely to contain (Ewing).

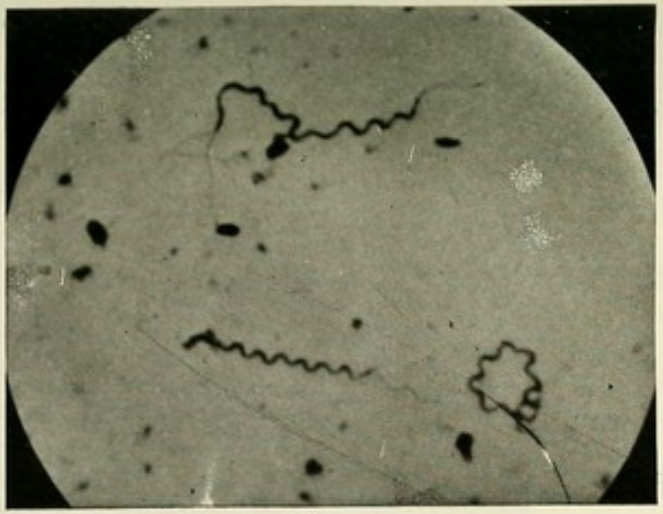

Fig. 1.

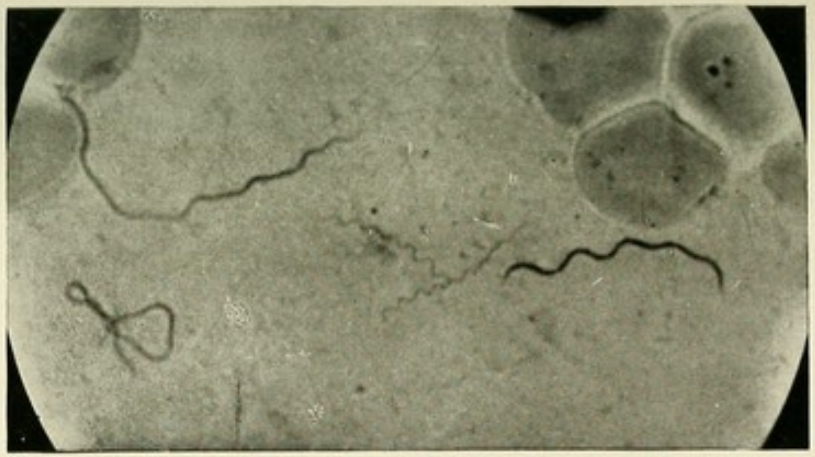

Fig. 2.

Fig. 3.

Fig. 4.

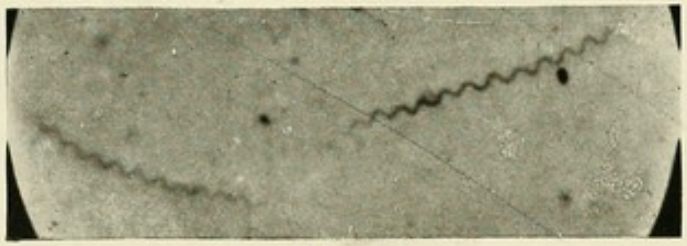

Fig. 5.

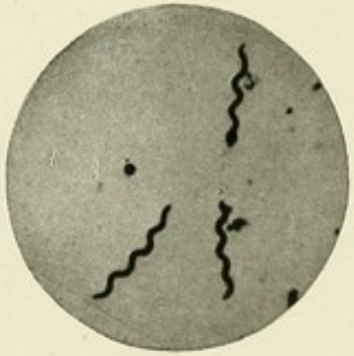

Fig. 6.

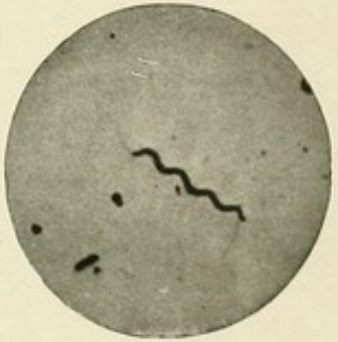

Fig. 7.

PLATE I.—SPIROCHETÆ.

FIG. 1.—*Spirochetæ pallidæ.* Remarkably intense stain: shows flagella, one of which is bifurcated. The distortion of the spirochetæ is unusually great. (Schaudinn.)

FIG. 2.—Contrast between *Spirocheta pallida* and *Spirocheta refringens;* the former in the centre of the field (two specimens) very faintly stained, but with numerous sharp curves, the latter more deeply stained and only slightly undulating. (Schaudinn.)

FIGS. 3 and 4.—*Spirocheta dentium* or *buccalis*, a short, thick, long curved, deeply staining organism found in the mouth. (Hoffmann and Prowazek.)

FIG. 5.—Two typical *Spirochetæ pallidæ.* (Schaudinn.)

FIGS. 6 and 7.—*Spirocheta balanitictis*, similar to *S. buccalis.* These specimens show flagella, in one instance apparently joining two spirochetæ. (Schaudinn.)

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3. Immediately spread the diluted stain on the specimen and let it stand one hour.

4. Wash freely in water, dry without heating, and mount.

This is the standard stain. A quicker method is that of Simonelli and Bandi [1] or of Goldhorn,[2] sold in this country under the name of Goldhorn's stain. A few drops of this will stain a specimen (dried without heating) in two or three seconds. The smear is then rinsed in water and dried (cold).

In France a favorite stain is the Marino blue; innumerable other stains have been tried, but the Giemsa and the Goldhorn fill all requirements. With Giemsa the spirochetæ appear of a faint red color. The Goldhorn stains them purplish, which may be changed to brownish black by treating the specimen with gram or lugol.

Tissue Stain.—The best is Levaditi's modification of the Ramon y Cajal silver stain.[3]

1. Sections are cut 1 mm. thick and hardened in ten per cent formalin for twenty-four hours.

2. Wash and harden in ninety-six per cent alcohol twenty-four hours.

3. Wash a few minutes in water until they sink.

4. Impregnate with silver by soaking for three (to five) days in a 1.5 per cent (to three per cent) solution of silver nitrate at a temperature of 38° C.

5. Wash rapidly in water and place for twenty-four (to forty-eight) hours at the room temperature in

Acid pyrogallic	2 gm.;
Formalin	5 c.c.;
Aq. destill.	100 c.c.

[1] *Centralbl. f. Bact., Parasit. v. Infect.*, 1905, vol. xl, p. 159.

[2] *J. of Exper. Med.*, 1906, vol. viii, p. 451.

[3] The so-called old Levaditi, in contradistinction to the new or pyridin Levaditi, which is quicker but not so accurate.

6. Wash, dehydrate in absolute alcohol, and mount in paraffin.

7. Cut sections no thicker than 5 μ.

8. Stain either with (*a*) Giemsa, for a few minutes; wash in water, differentiate in alcohol containing a few drops of oil of cloves, clarify in xylol, and mount in balsam; or (*b*) concentrated toluidin-blue solution, differentiate in alcohol containing a few drops of Unna's ether-glycerin mixture—xylol, balsam.

Diagnosis of S. Pallida.[1]—The time and skill required to perform the Levaditi stain successfully suffice to keep tissue staining apart, to be employed only by the most competent specialists. Current examinations for *Spirocheta pallida* may be attempted only on smears stained with Goldhorn or Giemsa.

Examination of such a smear promptly reveals how appropriate is the name, *S. pallida*; for so pale and thin are these microörganisms, that at first one finds great difficulty in perceiving them. It has been generally noted that the observer at first makes very few positive finds. But, having at last identified the spirocheta and got it photographed, as it were, on his retina, he can return to specimens previously found negative and discover the organism in them—perhaps in great numbers.

Hence the discovery of *S. pallida* requires not only familiarity with ordinary laboratory and microscopic technic, but also a relatively long and tedious special training. Unhappily, it is to be foreseen that, as spirocheta diagnosis assumes greater and greater prominence in the diagnosis of syphilis, the tribe of near pathologists will feel fully competent to pass upon this—the most delicate point in the diagnosis of the most important infectious disease that afflicts mankind; to the cocksure diagnosis of the hasty practitioner will be added the scientific diagnosis of incompetence. For spiral organisms

[1] The diagnosis by inoculation is referred to on page 31.

abound both upon the skin and upon the mucous membranes. In the mouth are found *S. denticula, S. buccalis, S. Vincenti*; in the bowel, *S. dysenteriæ*; on the skin, *S. refringens* and various saprophytic varieties.

The accompanying photographs (Pl. I) show, however, the characteristic features distinguishing *S. pallida* from every other variety of spirocheta. These are:

1. Extreme tenuity and faint staining.
2. Multiple small, abrupt spirals.

All of the other familiar spirochetæ are much thicker (most of them are longer), stain readily with the common dyes, and exhibit long, gentle undulations in brilliant contrast to the sharp, short, almost angular spirals of *S. pallida.*

This is not to say that every *S. pallida* seen can be identified as such, or that every specimen containing *S. pallida* can be diagnosed even by the most skilled observer. Indeed, quite the converse is true. Even Neisser confesses to a doubt about certain of his cases. But a characteristic *S. pallida* is as typical to a skilled eye as is a characteristic gonococcus, for example, and affords quite the same diagnostic certainty.

But before a negative report can be given, repeated, prolonged, systematic, and skilled examinations must be made.

Where may S. Pallida be Found?— The earlier observers were able to discover *S. pallida* only in the earliest (and most infectious) lesions of the disease. The following condensed list shows the results obtained by those reporting the greatest number of cases.

Oppenheim and Sachs examined 118 cases, with 39 positive results.

Mulzer, 22 cases, 2 positive; 56 controls, all negative.

Nicolas, Favre, and André, 42 cases; 13 positive.

Kraus and Prautschoff, 37 chancres; 32 positive, 25 secondaries, 18 positive.

Siebert, 18 chancres; 13 positive, 46 secondaries, 39 posi-

tive; 46 control examinations negative; 6 lymph node serum negative; 7 gummata negative; cerebro-spinal fluid, blood and semen negative.

Sobernheim and Tomasczewski, 50 cases, all positive; 28 controls, all negative.

Scholtz, 37 cases, all positive.

Schaudinn (second report), 70 cases, all positive.

Roscher, 32 chancres, 31 positive; 58 moist papules, 55 positive; 40 dry papules and pustules, 34 positive; 29 mouth lesions, 28 positive; 38 lymph node serum, 30 positive; 24 controls negative.

Sufficiently expert and conscientious investigation, therefore, reveals *S. pallida* in fully three fourths of the smears taken from chancres, moist papules, and mouth lesions.

It was to be hoped that aspiration of the lymph nodes adjacent to the chancre might prove a simple means of obtaining uncontaminated smears of *S. pallida*; but, unfortunately, the microörganism is rare in the center of nodes, being chiefly confined to the region of the periphery, so that there is a distinctly less probability of finding them there than in the chancre itself.

Though the later secondary lesions contain fewer spirochetæ, these have been found as late as nine years after chancre by Sobernheim and Tomasczewski.

They have been found in the pus from a non-syphilitic abscess occurring during the acute stage of the disease (Flügel), in the serum of blisters raised by cantharides (Levaditi and Petresco), in albuminous urine (Dreyer and Toepel), in the blood—after many failures, and only during the first few months and before the beginning of mercurial treatment (Nöggerath and Stähelin, Schaudinn, Richards and Hunt *et al.*).

Most interesting of all has been the search for spirochetæ in tertiary lesions, which, for a long time, was fruitless, but

Fig. 1.

Fig. 2.

PLATE II.—Spirochetæ in Tissues.

Fig. 1.—Macular syphilid. Section of venous capillary, showing distribution of spirochetæ and phagocytosis by leukocytes and fibroblasts (at F, L, P). (Ehrmann.)

Fig. 2.—Spirochetæ in bone marrow in hereditary syphilitic osteo-chondritis. (Bertarelli.)

was finally crowned with success (page 37). Tomasczewski, who has found them in five out of ten gummata examined, states that eight to ten hours must sometimes be spent in examining smears before finding a typical spirocheta.

The moist lesions of early hereditary syphilis swarm with spirochetæ, and they have been found (either in smears or in sections) in practically all the organs of stillborn syphilitic infants; viz., liver, lung, spleen, kidney, suprarenal, muscle, heart, stomach, intestine, mesenteric glands, gall-bladder and ducts, ovary, uterus, prostate, testis, urinary bladder, thymus, tonsil, bone, joint, etc. They are usually most numerous in the liver, lungs, and skin. They have been found in both fetal and maternal placenta, and once in the inguinal glands of the apparently healthy mother of a syphilitic child (Buschke and Fischer)! Curiously enough masses of spirochetæ are sometimes found in and about the capillaries where no tissue change has taken place.

They have not been found in the cerebro-spinal fluid, though they doubtless will be, for positive inoculations have been obtained upon monkeys with this fluid (Hoffmann).

The examination of normal secretions—except the semen—is always negative, except in severe congenital syphilis (page 53). Whether the exception in the case of semen is due to syphilitic lesions in the seminal canals it is impossible as yet to say.

Distribution and Fate.—Though spirochetæ have been kept alive for a few days on artificial media, none of the attempts at artificial cultivation have thus far been successful.[1] Hence we are not in a position to affirm with absolute certainty that the spirocheta is self-sufficient, self-multiplying, like the known bacteria, and not a mere developmental form of some unknown organism (possibly the cytorrhictes). More-

[1] Quite recently *S. refringens* has been cultivated by Levaditi; *S. obermeieri* by Novy; perhaps *S. pallida* will be the next.

over, the multiplication, distribution, and fate of the spirocheta in the body of a syphilitic patient have not been worked out in detail. But the following facts we know:

Spirochetæ are found most frequently in the earliest and most infectious lesions of syphilis. Whether in the chancre and early secondary lesions of acquired syphilis or in the organs in inherited syphilis, they abound in the walls of the blood-vessels and in the perivascular tissues. They are relatively rare in the lymph vessels, surprisingly few in the nodes, and when found in the nodes are usually in or about the blood capillaries at their circumference (Hoffmann and Beer). They have been found in great numbers among the epithelia of the chancre or the moist papule. A few observers believe they have seen evidence that the spirochetæ are destroyed by phagocytosis. In tertiary lesions (gummata) they have been found only in the active, advancing edge of the lesion, never in its necrotic center or in its secretion.

It seems probable, therefore, that the spirochetæ are distributed by the lymph rather than by the blood current, directly excite the perivascular inflammation characteristic of all syphilitic lesions, and are destroyed by phagocytosis.

Although a few observers have denied that the administration of mercury has any effect upon the spirocheta, it is the consensus of opinion that, as soon as mercury is administered, the spirochetæ rapidly disappear and soon are undiscoverable, and this disappearance seems to be especially rapid under inunction and hypodermic medication.

EXPERIMENTAL SYPHILIS

The countless attempts at animal inoculation made with the virulent secretions of syphilitic lesions before Schaudinn's discovery of the spirocheta may all be accounted failures, with the exception of the chimpanzee successfully and convincingly in-

Fig. 1.

Fig. 2.

PLATE III.—Spirochetæ in Tissues.

Fig. 1.—Hereditary syphilis of liver. Spirochetæ (*s*, *s'*) are shown within the liver cells (*h*, *h'*): *p*—polynuclear leukocyte; *f*—fibroblast. (Levaditi.)
Fig. 2.—Skin affected by hereditary syphilitic pemphigus.
The epidermis (*e*) shows pemphigus vesicles (*v*), containing mononuclear (*l*) and polynuclear (*p*) leukocytes, and spirochetæ (*s*): *pa*—infiltrated papilla. (Levaditi).

oculated with syphilis by Metschnikoff and Roux in 1903. But even this experiment derives much of its force from the confirmation of post-spirocheta investigations. Of all the rest, whether upon monkeys, dogs, cats, or other animals, the best that can be said is that they were doubtful and unconvincing.

But with the advent of the spirocheta it became possible to follow the infecting agent as well as the infective lesion; to find spirochetæ in the infectious secretion, again in the resulting sore, and again in other chancres produced by inoculations made from this sore upon other animals.

Much work has been done in this line by the distinguished chiefs of the Institut Pasteur at Paris, by Neisser, who made a special trip to the tropics for this purpose, and by others. These investigators have proven beyond possible doubt that monkeys may be infected with syphilis, and have even worked out the type of simian syphilis.

The anthropoids develop chancre followed by secondary skin lesions closely resembling those found in man. Moreover, they sometimes become markedly cachectic, and may even be paralyzed; but this acute stage is of very brief duration, and is not followed, as far as known, by any evidence of tertiarism. In the lower orders of monkeys inoculation can be performed only upon certain parts of the body (eyebrows, eyelids, genitals) and is followed, after the chancre, by but few and irregular evidences of general infection (skin lesions, toxemia).

In other animals successful inoculations have been made; but the resultant syphilis is a travesty on the dire disease we know. Thus Bertarelli, Hoffmann, and Scherber have inoculated the corneas of bitches and produced a localized typical syphilitic inflammation from which spirochetæ could be obtained. But such investigations are of no practical import.

But from the results of monkey inoculation many and most important inferences have been drawn. It were impracticable

to relate them here in full detail. The student will find practically all of them in the writings of Metschnikoff and Roux,[1] and in the report of Neisser, Boermann, and Halberstädter.[2] We must summarize as follows:

Inoculation.—It is impossible to inoculate by subcutaneous injection. The secretions clinically the most infectious are experimentally the most noxious; yet it is possible to perform successful inoculations and to obtain characteristic spirochetæ from the resultant lesions, although the most careful examination of the inoculating fluid fails to reveal in it any spirochetæ. Successful inoculations have been made with human blood (Hoffmann, Finger) and with fragments of gummata (Finger, Nitze), but only after repeated failures. Finger obtained a positive inoculation with apparently normal seminal fluid.

Reinoculation (producing multiple chancres) is possible for from ten to fourteen days; after this—as in man (page 225)—it is impossible.

But the most important practical conclusion yet reached is that *syphilitic secretions cease to be infectious after twelve to twenty-four hours, and much sooner (at most six hours) when dry.* This explains why we are not all infected by cigars and why the syphilitic may live and dine with his family in absolute safety so long as the cups, forks, and spoons that enter his mouth are washed and dried before being used by anyone else at the following meal.

Prophylaxis.—As soon as Metschnikoff and Roux had proved to their satisfaction the transmissibility of syphilis to monkeys, they turned their attention to its prophylaxis. The results of their experiments were startling. They found that *excision of chancre, even of commencing chancre, is entirely futile as a preventive or minimizer of the subsequent develop-*

[1] *Bull. de l'Institut Pasteur*, vol. i, p. 620; vol. ii, pp. 113, 178; vol. iii, pp. 73, 451; vol. iv, p. 116; also *Annales de l'Institut Pasteur.*

[2] *Deutsche med. Wochenschr.*, 1906, vol. xxxii, pp. 1, 49 and 97.

ment of the disease. Cauterization of the chancre they found equally futile. Indeed, *wide excision of the inoculated area at any time later than eight hours after inoculation failed to prevent the development of chancre*[1] (confirmed by Neisser). Moreover, the application of heat to the site of inoculation (to destroy the virus) and of various antiseptics (including strong solutions of sublimate) availed nothing.

The only way in which they were able to destroy the virus after inoculation was by applying, within six hours, a calomel salve (calomel 20, lanolin 40). So universally preventive did this prove that they performed a confirmatory inoculation (with monkey controls) upon a medical student, inoculating him with the virulent secretion of a chancre and of a mucous papule, rubbing in the calomel salve within one hour, and watching him long enough to be sure that no sign of syphilis developed.

Hence we may infer that *the only personal prophylaxis against syphilitic infection lies*, not in cauterization or excision, but *in inunction with a strong mercurial ointment within one hour*[2] *of inoculation.*

Inoculation Immunity.—All efforts to develop artificially either an active or a passive immunity to syphilis have failed in man as well as in monkey. Taking as a starting point the immunity to reinfection, which begins so early in the disease (tenth to fourteenth day), and usually lasts a lifetime, inoculation experiments have been made with the blood serum of syphilitics, with the juices from syphilitic lesions (filtered through porcelain), as well as by "passing" the infection through several individuals. The result has been absolute zero: as yet "the only way to be immune to syphilis is to have it."

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[1] But in one case amputation of an inoculated ear after twenty-four hours was preventive, and the monkey was subsequently successfully inoculated.

[2] Perhaps as late as six hours the remedy might still be efficacious.

Serum Diagnosis and Treatment.—In this line lie our brightest hopes for practical results from animal inoculation. Imagine what would become of syphilis on the discovery of a diagnostic chemico-physiologic test and of a therapeutic serum! The promise that some such sera may soon be obtained is almost too good to believe. Thus far no practical results have been obtained,[1] though Wassermann, Neisser, and Bruck claim to have shown the development in infected monkeys of a syphilitic immune substance.

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[1] The Wassermann diagnostic serum has given accurate findings in little over half the cases tested (Cf. Ewing).

CHAPTER IV

THE NATURE OF SYPHILIS

At this date when the protozoal (or microbic) cause of syphilis has just been discovered and a study of its nature has scarcely begun, it seems futile to waste many words over ancient theories concerning the nature of the disease; for scientific investigations of the coming decade will surely modify the more or less accurate guesswork upon which we have hitherto depended.

At present we may be sure that syphilis is an infectious disease. As such its primary and early secondary lesions are readily explicable. These local lesions are inflammatory in type, a reaction to aggregations of the specific protozoa. Accordingly their secretion is infectious and contains the spirocheta.

But tertiary lesions are not clinically infectious, contain relatively few spirochetæ, and often appear after an interval of years, during which the patient has apparently enjoyed perfect health. How shall we interpret these characteristics, how explain the malignancy of these lesions as contrasted with the mildness of those early ones that abound in spirochetæ?

Many theories have been conceived to explain the facts. We lack space to consider them in detail.[1] They may be classed under three heads:

[1] Neumann (*Medical Record*, 1906, vol. lxix, p. 649) has recently published a masterly summary and critique on this subject. Thalmann (*Münch. med. Wochenschr.*, 1907, vol. liv, p. 601) reviews the subject in the light of the recently discovered spirocheta and its relation to the body cells according to Ehrlich's theory.

1. Unna believes that the late lesions of syphilis are local recrudescences in the site of old lesions. The distribution of the poison occurs in the first few years of the disease. Thereafter local areas of inflammation may remain for years dormant and unsuspected until, stimulated by local trauma or general debility, they awake to a purely local renewed activity.

2. Finger maintained that, although the primary and secondary lesions are due to the microbic (or protozoal) cause of syphilis, the tertiary lesions are toxic in character and due to the poisons evolved by these microbes. (The discovery of spirochetæ in gumma and its proven infectiousness have led Finger to give up this theory.)

3. Most authorities, notably Neumann and Lang, accept in a modified way the theory of Virchow. The microörganisms which cause the primary and early secondary symptoms are supposed to be destroyed only in part. They lie dormant in the lymph nodes (Virchow) or in other regions (Lang), or in the scars of old syphilitic lesions (Neumann) until, awakened by trauma or debility of the host or by their own gradual multiplication, they suddenly set up, not a local reaction, not a general toxemia, but a general septicemia, as it were, a renewed spreading of infection throughout the system. Hence result the various late secondary or tertiary symptoms, differing in distribution and in quality from the first outbreak of the disease because the host is partially immunized.

Experimental syphilis (in animals) is rapidly solving many of the problems suggested by these conflicting theories.

The Contagiousness of Gumma.—The most important questions thus far solved by experimental inoculation relate to the theories of infection. But one point may be emphasized here: the contagiousness of gumma has been definitely proven. We have already had occasion to state that *clinically gumma is not contagious.* A very few exceptions to this clinical rule

have been recorded (cf. Williams [1]), but their very rarity makes them suspect. Indeed, every attempt at inoculation of gummatous secretions, even upon non-immune human beings, failed until 1905. In that year both Finger and Neisser successfully inoculated monkeys with the scrapings from an active but not ulcerated gumma. Previous experimenters had employed the secretions of ulcerated lesions, and herein lay the secret of their failure. They worked with a virus whose feeble contagiousness was overcome (doubtless by destruction of the spirocheta) by mixed infection with the vulgar pyogenic cocci. The success of Finger and Neisser was due to the employment of an uncontaminated virus obtained by curettage or incision. Herein, too, lies the distinction between the clinical innocuousness of gumma and its actual contagiousness. Its ordinary secretions are not contagious, though itself contains a *virus vivum* and is contagious. The distinction would seem a perilously tenuous one to put faith in were it not borne out by an infinity of clinical experience. That the duration of the disease has no effect upon the contagiousness of the lesion is proven by the fact that one of Neisser's successful inoculations was made from a gumma occurring in the tenth year of the disease.

Persistence of Spirocheta.—The proven infectiousness of gumma at so late a date, the evidence (page 29) that all gummata probably (and from thirty per cent to fifty per cent of them certainly) contain spirochetæ, the possibility (page 61) that syphilis *may* remain infectious long after the appointed five years, the assurance (page 65) that an apparently sound syphilitic woman may continue to bear syphilitic children for ten, fifteen, and twenty years, and the belief of certain excellent authorities (page 75) in syphilis of the third generation, combine in cumulative probability to show that

[1] *Med. Record*, 1906, vol. lxix.

the persistence of spirochetæ [1] is coincident with the duration of the disease. So long as the patient remains syphilitic (page 132) he bears within the active infectious agent of the disease. Yet this is far from implying a continuance of infectiousness (page 61).

It may still be true that the essential difference between a tertiary lesion and a secondary one is that the former is the product of some tissue-destructive chemical agent (toxin) plus the spirocheta, while the latter is due to the protozoön only; but this is pure speculation.

But if primary, secondary, and tertiary lesions are all caused by the same living parasite, present and active in the host even during long years of apparent health, we may well conclude that some serum reaction indicative of its presence might be devised (and pray for its advent), and we may ask what causes the relapse of symptoms.

Cause of Relapses: Trauma and Syphilis. — Every theory of syphilis invokes local trauma or general debility as the cause of relapse, and—broadly speaking—the rule holds true enough. The effect of trauma in evoking syphilitic lesions is daily exemplified by the smokers' mouth. Some patients, however thoroughly treated, cannot smoke even one cigar a day without suffering repeated relapses of mucous patches and ultimately having tertiary glossitis. In a similar way, skin lesions often relapse *in situ*, and trauma to bone frequently starts a node or a gumma. Thus I have known a horse kick to be the cause of an enormous gumma of the femur. Twice I have seen the application of a plate of false teeth to the upper jaw promptly followed by perforation of the hard palate. And every now and then one hears of a syphilitic bone lesion following fracture.

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[1] Or of some other microörganism of which *S. pallida* is one of the manifestations.

Yet this is by no means the whole story. I can call to mind two persons (one of them a woman) who never had smoked, and yet suffered severe and prolonged mouth lesions. Indeed, the vagueness of our knowledge on this point is demonstrated by our inability to prophesy relapses with any certainty. The washerwoman and the baseball player are not peculiarly subject to palmar eruptions, nor the microscopist to iritis, nor the athlete to periostitis. Surgical wounds rarely become syphilitic; bruises and fractures often do.

There are probably two other elements in the equation: the inherent, congenital susceptibility of the patient's various tissues (which makes one patient prone to skin lesions, another to brain lesions, etc.) and the rate of growth of the spirocheta. This latter variant is perhaps influenced by the patient's general condition; for *close questioning often reveals the fact that a relapse due to no apparent cause has followed a period of mental depression*; *stress, or grief* obviously calculated to depress the vital resistance of the tissues, and thus to permit more rapid multiplication of any parasites in the body.

But when a relapse of symptoms does occur, if no treatment is administered, further relapses in various parts of the body are almost inevitable. The renewed virulence of the disease is not localized, but general. It may even spread in spite of vigorous treatment. Indeed, a vigorous, rebellious relapse of syphilis after years of quiescence is one of the most mysterious phenomena a physician ever witnesses.

Examples are common enough; let the following suffice:

Case VIII.—A patient who, after a few early symptoms and adequate treatment, has remained well twelve years, develops a severe neuralgia of the right arm. This lasts several months, and is almost well, though no antisyphilitic medication has been given, when a typical gummatous periostitis brings him to me. This yields rapidly to mixed treatment, but six months later

relapses. Again it is promptly cured, and remains well two years.

Case IX.—Another, a chronic, steady drinker, whose disease is four years old, well treated, and who has had no symptoms whatever since the first eruption, develops gumma of the nasal septum. This he neglects for a year, when a large gumma suddenly appears on the right parietal bone. He promptly takes treatment, and within six weeks is entirely well. Yet, while continuing his treatment, a few weeks later he develops simultaneously an interarytenoid edema (causing aphonia) and a chancre redux. The former yields only after six weeks of the most vigorous treatment. The latter continues as a little, indurated lump for six months. Then he has two attacks of vertigo, due probably to cerebral syphilis. (Neurologists differ as to the exact condition.)

Such a train of circumstances (and it is much commoner than the occurrence of an isolated lesion due to trauma) shows that a tertiary outbreak is rather a general reaction than a local one, and attacks a certain spot rather because it offers less resistance (whether on account of previously existing syphilitic inflammation, trauma, or some cause unknown) than because there is an actual local deposit of syphilitic virus in that spot already.

The Cause of Parasyphilids.—The parasyphilids are apparently due to some form of toxemia. The fact that other toxemias, such as alcohol and lead-poisoning, produce similar effects argues for this conclusion, while the rule that parasyphilids are rarely associated with active tertiary lesions and occur quite frequently in cases that have shown no tertiary lesions suggests the thought that, doubtless, the syphilitic toxemia which produces parasyphilids is related to the *virus vivum* of the primary and secondary lesions rather than to that of the tertiary symptoms. Moreover, the toxemia is usually a mixed one; partly syphilitic, partly alcoholic, or contributed to by some other poison.

IMMUNITY

Is there a personal immunity from syphilis? Though the question is a vital one, it has never been answered. Clinically, a certain proportion of those exposed to syphilis do not contract it; but this is due to the protection of an unbroken integument. Inoculation experiments made upon man have occasionally failed, and it has been inferred that these failures were due to a natural immunity. Perhaps it was because the subjects were already syphilitic.

Various general phases of immunity merit consideration. Such are racial immunity, immunity conferred by syphilis, and the relative Immunity of Women.

Racial Immunity.—Negroes, though utterly careless about treatment, are relatively immune to syphilis, and rarely exhibit parasyphilitic lesions. This fact, accepted in our Southern States, has also been noted by Livingstone in Africa.[1] The inhabitants of Iceland, Greenland, and Newfoundland are said to be equally blessed. On the other hand, other races not only readily fall victims to this plague, but also show a peculiar vulnerability when first attacked. Thus syphilis, when introduced among the South Sea Islanders by Captain Cook's sailors, is alleged to have attacked the poor natives with fiendish virulence, although nowadays their descendants suffer no more from the disease than the rest of the world.

Whether there is any foundation for the belief that syphilis acquired by a European from an Indian or a Chinese is the more virulent because of its origin would be difficult to prove. Certainly the few cases I have seen imported from Cuba, Porto Rico, China, and Japan happen to have been rather mild. Yet some authorities allege that the ravages of syphilis at the end of the fifteenth century were due to the fact that the new species

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[1] Hirsch's *Handb. d. Histor-geogr. Pathologie*, 1883, vol. ii, p. 25.

of syphilis imported by Columbus's sailors from the American aborigines was peculiarly noxious to Europeans, although they were already pretty well immunized by heredity against some other species of the disease handed down to them by their own forefathers.

Is Our Race Immunized?—But this amusing speculation suggests a point of intense practical interest. If our forefathers became immune to one species of syphilis, shall not we become immune to another? And is not syphilis perhaps milder in this generation than in the preceding ones? Assuredly, one might answer, if we were ever to attain a racial immunity against syphilis we should have acquired it ere this. For twelve generations all Europe has been steeped in syphilis, and it is to-day the greatest plague, except tuberculosis, of civilized nations. No, syphilis is not dying out; but it is, thank God, becoming milder. And the reason it is becoming milder is that it is being better treated.

For example, among the cases of tertiary syphilis treated in this office previous to 1880, 94 are known to have received at least eighteen months' early treatment, while 109 had been treated less than a year. In the next decade the proportion was 107 : 88, and since 1890, 138 : 53.[1] Or, to express the same figures more concretely: Before 1880, forty-five per cent of these cases were treated at least eighteen months; in the next decade, sixty per cent; and since then, seventy-two per cent. And, although prolonged treatment is, unhappily, not altogether synonymous with intelligent treatment, still the diminution in the intensity and duration of the tertiary lesions of syphilis in this generation is, I believe, due to a progressively increasing application of intelligent hygiene and therapeutics to the disease rather than to any progressive immunity.

[1] In most cases of ancient syphilis it is impossible to learn accurately the nature and duration of early treatment; hence the relative smallness of all these figures.

THE SECOND ATTACK OF SYPHILIS

In a recorded office experience of over two thousand cases of syphilis and an unrecorded hospital experience of far more than as many again, neither my father nor I have encountered an authentic case [1] of a second attack of syphilis. Our experience, therefore, convinces us that a single attack of syphilis confers immunity that lasts an ordinary lifetime. It were, however, quite as absurd to insist that this immunity must last a lifetime as to insist that syphilis is incurable.[2] All that one can safely allege is that there is only an infinitesimal danger of error in assuring one's patients that syphilis boasts a solitary virtue, viz., having acquired it once they will never acquire it again.

How is it, then, that almost every syphilographer can recall numerous instances of reinfection with syphilis? How is it that such a collection of cases as Koebner's [3] can be gathered from reports made within a few years of one another? The chief reason is that there is a very common tertiary lesion of the penis, the so-called chancre redux, which mimics in every detail the original chancre. This is best exemplified by a recital of cases. For example, Lang,[4] who believes reinfection common enough, cites the following instance of reinfection in a case of hereditary syphilis:

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[1] And only two probable instances.

[2] The duration of immunity is to some minds an evidence of the duration of the disease. "Since the syphilitic remains forever immune to syphilis, he is forever liable to new outbreaks of the disease." As well might we say that, since an attack of small-pox confers an immunity that usually lasts a lifetime, the immune is still a sufferer from small-pox. The converse proposition, however, that in the present state of our art a second attack of syphilis is the only possible proof that the patient has recovered from his first attack is unimpeachable.

[3] *Berlin. klin. Wochenschr.*, 1872, vol. xlvi, p. 549.

[4] *Pathol. u. Therap. d. Syph.*, 1896, p. 103. The reinfection of a hereditary syphilitic is, of course, no more and no less probable than when the first infection is acquired by extra-uterine contagion.

Case X.—A waiter, whose father was syphilitic and whose mother had died in childbirth, stated that he was the youngest of a number of children, none of whom had lived more than a few months. He remained well till sixteen years of age, and then developed an ulcer on the right leg. This was cured by inunctions and iodid of potash. Two years later he lost his uvula by ulceration, and was again treated by iodid. He then remained well until his twenty-fifth year, when, a few weeks after coitus, he developed a sore on the penis. Lang diagnosed this as chancre, noted typical syphilitic scars on shin and soft palate, and observed that the glands in the right groin were enlarged. In three weeks the ulcer healed under mercurial ointment, and the patient was never seen again.

Compare with this Koebner's Case VIII, upon which he lays the greatest stress:

Case XI (Koebner's VIII).—A man of forty-five has syphilis in 1866, and his wife an ulcerated tubercular syphilid in 1867. In 1871 this man, having had intercourse with a prostitute ten weeks previously, with his wife nine and nineteen days previously, exhibits an indurated sore on the penis. No enlargement of the inguinal glands. The wife remains healthy. The sore heals under iodid of potash, and no further symptoms develop.

These are typical instances of alleged second attacks of syphilis—alleged by men whose experience and authority are unquestioned. Yet they found their diagnosis of reinfection upon evidence which it is hoped they themselves would not consider adequate for a diagnosis of the first attack, viz., chancre alone, whether accompanied by adenitis or not. My records include over fifty cases of chancre redux (page 482), and though many of them simulated the initial sore in every respect, the diagnosis of reinfection was, happily, not made in any instance.

To make a diagnosis of reinfection, the necessary elements are (1) sufficient evidence of a previous attack whether in the history of primary and secondary lesions sufficiently clear to

exclude the possibility of error, or, better still, fortified by characteristic scars of tertiary lesions. Besides this there must be (2) convincing evidence of a second infection. This consists of a typical chancre with *characteristic* syphilitic adenitis followed by *unmistakable* secondary lesions. As our knowledge of the spirocheta progresses we may doubtless add as a further essential the identification of this microörganism in the primary or secondary lesions.

Subject to such close scrutiny the cases of reinfection with syphilis are surely very, very few.[1]

SYPHILIS IN WOMEN

Syphilis is a far milder disease among women than among men. Thus my records show that among 207 women 18 never had primary or secondary lesions sufficiently marked to demand treatment, and could not when their tertiary lesions occurred recollect any early symptoms at all. This does not necessarily imply that these women had no primary or secondary lesions; for in only a small proportion of them were these lesions searched for and not found at the time of supposed infection. But it is in marked contrast to the 22 among 2,170 men who did not recollect their early symptoms. Hence among women, nine per cent, more or less, have primary and secondary lesions so mild as to be overlooked, and among men, only one per cent.

The same general rule holds good throughout the disease. Women are far less liable to parasyphilids. Thus I find in my records only three cases of tabes among women and 99 among men (respectively fifteen per cent and 4.7 per cent). However, there is one notable exception to the general rule.

[1] Mlle. Contamine has recently collected thirty-five cases (*Thèse de Gènes*, 1904) and Finger relates a few reinfections in hereditary syphilis. (*Wien. klin. Wochenschr.*, 1900, vol. xiii, p. 429.)

Women in the early stages of syphilis are peculiarly liable to be overwhelmed by the general toxemia; so that, whereas male patients rarely show grave anemia or alarming loss of weight, or nervous debility, it is not uncommon for women to be completely prostrated by the systemic infection while showing relatively slight local signs of the disease (page 79).

This relative immunity from syphilis enjoyed by women, while it is a thing to be thankful for, inasmuch as they are so frequently innocent victims of this disease, is a very confusing factor in the study of hereditary syphilis. For, when a woman married to a syphilitic man bears a syphilitic child, and yet fails, under the closest scrutiny, to disclose any symptoms of the disease, one naturally concludes that she is not syphilitic. Indeed, so commonly is this diagnosis made and so commonly do these women in later years break out with tertiary lesions of syphilis, that the ingenious theory has been evolved of infection of the mother through the child (*choc en retour*); the chain of events thus being infection of the fetus by syphilitic semen, infection of the mother by the syphilitic fetus. But if we remember that a very large proportion of women have extremely light early syphilis, and yet have tertiary lesions in later years—women who have never been married or borne children—the chief reason for this roundabout explanation of paternal syphilitic heredity is removed. (Contemplate the case of an apparently healthy mother of a syphilitic child, whose inguinal nodes nevertheless contained spirochetæ!)

PATHOLOGY

Syphilis is apparently caused by the *Spirocheta pallida*, and is characterized by lesions of the vessels and of the tissues surrounding them, excited by this parasite. Whether the lesions are excited by the parasite directly or through the medium of a toxin we do not know, though the latter is the more

probable hypothesis. Nor is it yet clear whether the damage is done chiefly through the lymph vessels and spaces or through the blood capillaries. The facts in favor of the former theory are:

1. The ingress of spirochetæ at the point of inoculation into the lymph spaces or lymph vessels of the skin (Hoffmann).[1]

2. The primary endo- and perilymphangitis observed in chancre (Hoffmann—though Ehrmann[2] believes the lymphatic involvement secondary).

3. The marked lymphatic involvement about the chancre and the early satellite and general adenitis.

4. In the later lesions the possible lymphatic origin of syphilitic arterial sclerosis and of tabes (Marie).

On the other hand, the facts in favor of hematogenous origin of the lesions are:

1. The manifest and marked involvement of the blood capillaries throughout the disease from the chancre to the latest visceral lesion.

2. The circulation of *S. pallida* in the blood at an early stage in the disease.

3. The lack of direct evidence that the late vascular lesions are of lymphatic provenance.

It is probable, therefore, that the primary lesion is of lymphatic origin, the later lesions of vascular origin.

With a few exceptions (of which the most notable are paresis, tabes, iritis, and the macular syphilid) the lesions of syphilis may be classed as perivascular, granulomata, i. e., infiltrations of the vessel walls and of the surrounding tissue with small, round cells and plasma cells. The vessels most commonly affected are the capillaries.

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[1] "Die Aetiologie der Syphilis," 1906.

[2] *Archiv f. Dermat. u. Syph.*, 1904, vol. lxviii, p. 1.

Syphilis is, therefore, classed (with tuberculosis and leprosy) among the infectious granulomata.

Lesions of the Capillaries.—The congestion and proliferation shows itself by dilatation of the vessel lumina, swelling

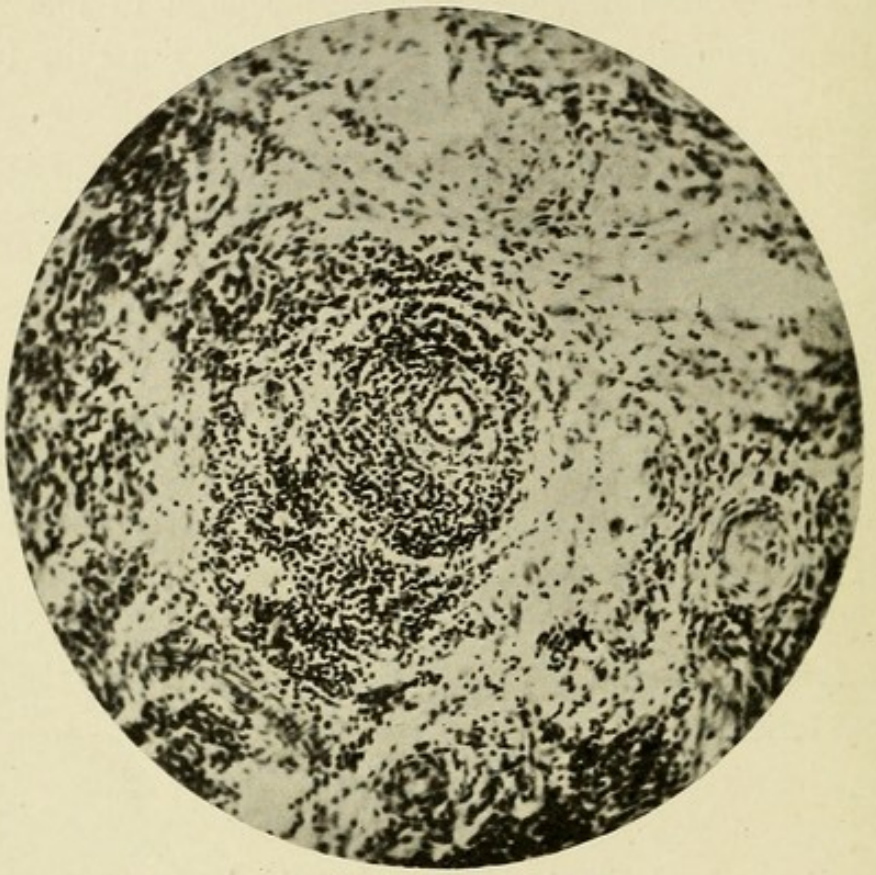

FIG. 1.—CHANCRE OF THE PENIS. (Zeiss 8 mm., comp., oc. 4.) An early lesion with mantle of round and plasma cells about vessels. The latter are increased in number and show an endothelial proliferation. The intervening connective tissue contains hyperplastic fibroblasts and a few plasma cells and lymphocytes. (Fordyce.)

and proliferation of the endothelium, and formation of new capillaries. Congestion and proliferation also occurs in the connective tissue surrounding the vessels, as a reaction to the exudation (Fig. 1).

The exudation occurs in a "coat-sleeve" fashion, surrounding the inflamed vessels with a zone of infiltrate many times the diameter of the vessel itself. This zone gradually merges at its edge into the surrounding normal tissue. Groups

of capillaries are usually affected and, when the inflammation is at all marked, the zones of infiltrate merge into one another to form an irregular area more or less widespread.

The exudate consists of small round cells and plasma cells, mingled with other cells (more or less proliferated) of the infiltrated tissue. Here and there a giant cell [1] is sometimes seen.

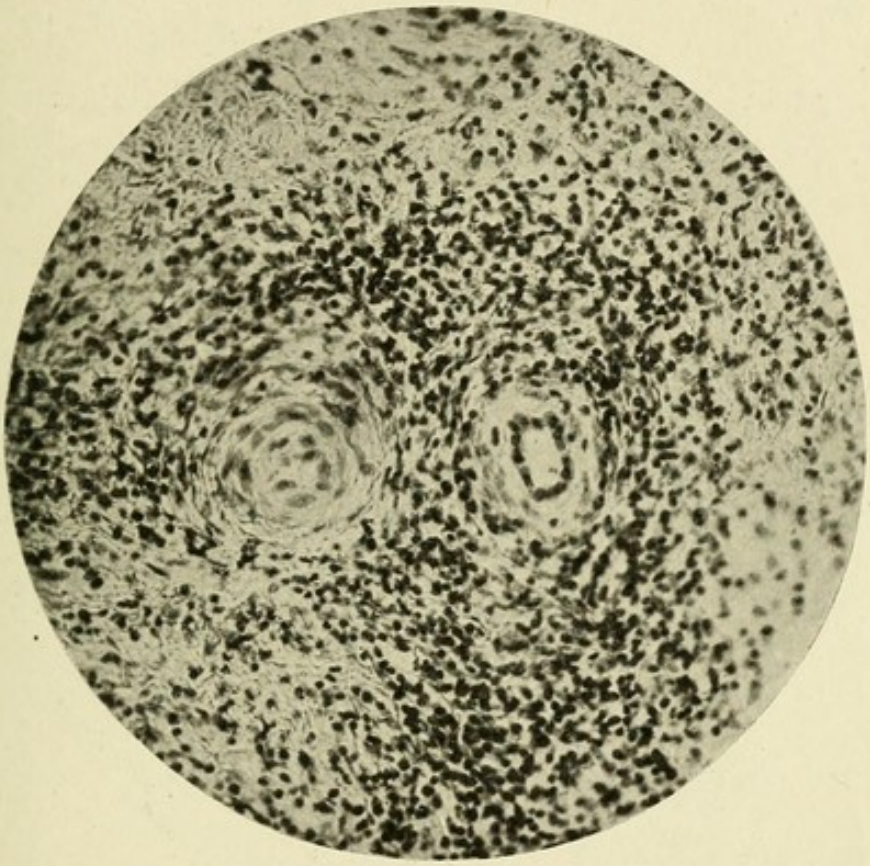

FIG. 2.—SYMMETRICAL ATROPHY OF THE SKIN WITH SYPHILIS. (Spencer ¼ in. Zeiss projection oc. 2.) Cross-sections of an artery and a vein. The former shows a proliferation of its intima and adventitia with narrowing of the lumen; the latter is completely occluded by intimal proliferation. Surrounding them is an infiltration of round and plasma cells. (Fordyce.)

These giant cells are neither constant nor characteristic of syphilis (they are much more common in tuberculosis).

The reaction of the exudation upon the capillaries themselves is most important. The vessels are primarily

[1] *J. Am. Med. Ass'n.*, 1907, vol. xlix, p. 462.

1. Congested and proliferated (see above) and secondarily

2. Thrombosed, "their former site being marked by solid cords, groups of irregularly disposed cells with pale staining nuclei and giant cells with peripheral or central nuclei or both" (Fordyce [1]). Without discussing further the disputed origin of the giant cells, one may recognize that many of them are due to this capillary degeneration (Fig. 2).

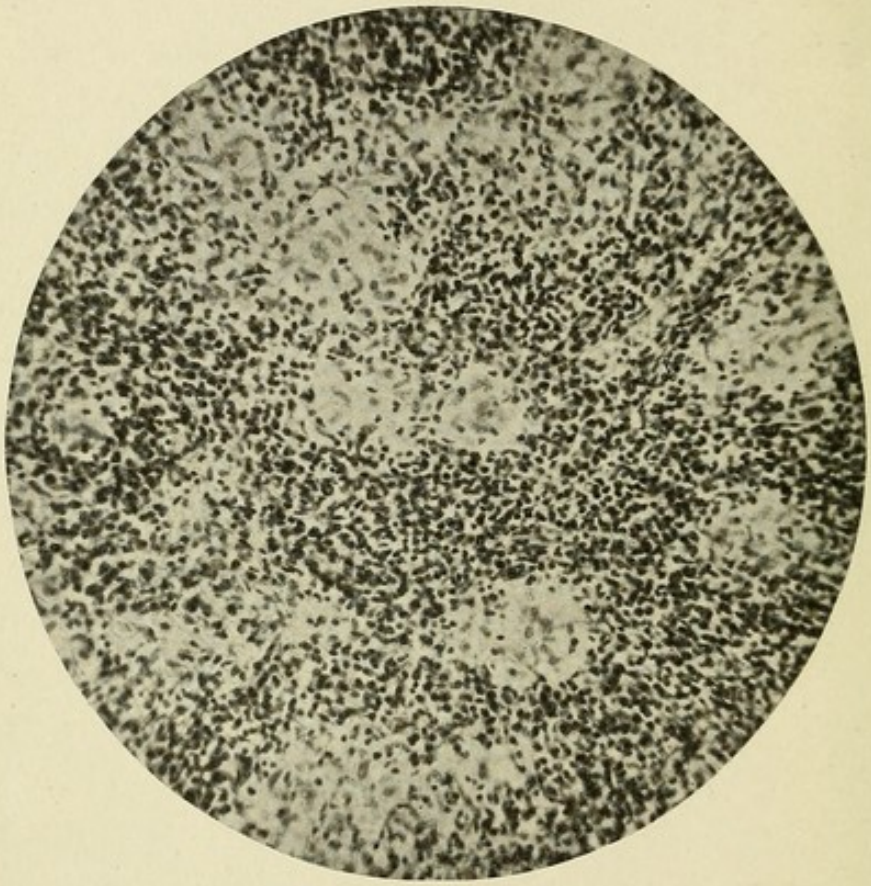

FIG. 3.—TUBERCULAR SYPHILID OF THE FACE. (Spencer $\frac{1}{4}$ in., Zeiss comp. oc. 4.) Showing obliteration due to endothelial proliferation and mantling of transformed vessels by round and plasma cells. (Fordyce.)

As a result of the thrombosis and destruction of the vessels, the infiltrated zones may be mottled, showing amid the cellular exudate lighter areas representing the degenerated capillaries (Fig. 3). Moreover, when this vascular degeneration is ex-

[1] *J. Am. Med. Ass'n*, 1907, vol. xlix, p. 462.

tensive, there is macroscopic necrosis of tissue on the exposed surface or in the center of the lesion. Hence the erosion of chancre, the desquamation of the syphilitic papule, the ulceration of the tubercular syphilid, and the caseation of gumma.

Three types of cellular exudate may be distinguished, viz.:

1. The diffuse exudate caused by an acute, intense syphilitic infection in a soil of little resisting power. This is seen characteristically in the visceral lesions of hereditary syphilis. A whole lobe of liver (page 506) or lung (page 462) is infiltrated; there is practically no tendency to localization of the exudate or to central degeneration (gumma); all the vessels are involved.

2. The circumscribed, mild, multilocular exudate with no tendency to central degeneration, but with a marked tendency to spontaneous resolution. Such are the ephemeral lesions of secondary acquired syphilis.

3. The circumscribed, grave, malignant lesions of tertiary syphilis (whose characteristics are shared in a less degree by the chancre), of which the gumma is the type. These tertiary or gummatous lesions thus consist in dense perivascular exudates with central degeneration (caseation) due to vascular obstruction and obliteration in the periphery of the lesion.

Disposition of the Spirochetæ.—In hereditary syphilis the spirochetæ are found in the capillaries both at the site of lesions and where no pathologic change is discernible.

In the pathologic exudate they are present in great numbers (Plates II, III).

In acquired syphilis they are numerous in the tissues infiltrated by secondary lesions, extremely rare in the tertiary lesions (page 29).

Lesions of the Larger Vessels.—Of the larger vessels the arteries are much the most commonly affected by syphilis, the veins less often, the lymphatics least of all.

The lesions are comparable to those that occur in the capil-

laries, i. e., exudation into and about the vessel with obliteration and degeneration as its terminal stage. Indeed, some larger vessels are often involved in the capillary lesions described above. But the typical syphilitic lesions of the large vessels, especially the arteries, derive their importance rather from the lesions of the vessels themselves than from those of the surrounding tissues.

The "coat-sleeve" infiltrate is seen about small vessels; but this systematic disposition of the exudate diminishes progressively in proportion to the size of the vessel attacked until, in the aorta, the lesions are usually patches distributed over the circumference of the artery, but showing no definite tendency to surround it (page 487).

The changes in the walls of the larger arteries are most common and have been best studied in the arteries of the brain (page 373). Whether these changes usually begin in the intima, as Heubner, their discoverer, supposed, or in the adventitia, as is now generally believed, they spread around and along the vessel in the form of a round cell exudate, usually involving the adventitia rather more than the intima, but in large measure sparing the muscular tissue of the media. Actual gummata may occur in the adventitia. Thus the wall of the vessel is thickened, its elasticity lessened, its intima congested and roughened. Hence in the smaller arteries the clinical result of syphilitic inflammation is usually thrombosis or obliteration, less often rupture or aneurysm, while in the aorta aneurysm is the clinical manifestation of the disease.

Let this brief *résumé* of the pathology of syphilis suffice not to exhaust the subject, but to impress upon the reader the unity of the phenomena of syphilis, be it hereditary or acquired, primary, secondary, or tertiary. The gross pathology of gumma, etc., is best considered in relation to the various regions involved.

CHAPTER V

TRANSMISSION OF SYPHILIS

RECENT experimental investigations upon monkeys have confirmed in large measure the accepted theories of syphilitic infection.

We have learned that the spirocheta is the infectious agent, and that infection is most common from the chancre and the secondary lesions. But, inasmuch as rather intimate contact with the secretions of a syphilitic lesion is essential to infection, it is probable that of the secondary lesions only the moist papules and ulcers are clinically infectious. Dry secondary lesions, as well as all tertiary lesions, do not transmit the spirocheta even though they contain it (page 36).

There has always been question, however, of the virulence of the secretions of the syphilitic, notably his semen, and also of the virulence of his blood, notably in reference to surgical operations. That the secretions may be virulent is proven by the discovery of spirocheta in the nares (Levaditi), on the conjunctiva (Bab), in the epithelium of the bowel and of the kidney in hereditary syphilis, while a monkey has been infected by the apparently normal semen of a syphilitic man.

Whether in each of these instances some minute syphilitic lesion was present and accounted for the exudation of spirochetæ is a purely academic question. The clinical fact remains that the overwhelming infection of severe hereditary syphilis may produce an infectiousness of all the apparently normal secretions; while in acquired syphilis the apparently normal semen may be infectious.

That infection from such sources must be rare, however, is shown by the numerous failures of inoculations made with mothers' milk, with urine, with semen, etc.; and the danger of such infection is probably extremely small unless from the prolonged contact possible in seminal transmission.

The danger of infection from syphilitic blood is even slighter. The spirochetæ are few in the blood and are present only in the most fluid stage of the disease. The real danger of infection with syphilis lies in the mucous ulcer or papule, which may exist unsuspected and even undiscoverable about the mouth or genitals, though its infectiousness is undiminished by its insignificant size.

Whether, as has been alleged, a clean woman may act as an intermediary host and convey the virus from a syphilitic partner to a clean one, and yet escape infection herself would be hard to prove, though such an accident is eminently possible. Sexual contact with a person supposed to be clean is surely often the occasion of a syphilitic infection; less often the infection is extragenital and non-sexual.

EXTRAGENITAL AND NON-SEXUAL INFECTION

Though syphilis occurs in all lands and at all ages, it is relatively much more common in some countries than in others. Thus in some of the Balkan states and in certain parts of Russia and Asia Minor, in many tropical countries, and in certain isolated communities syphilis is practically endemic. Everyone has the disease, and it is transmitted fully as commonly by extragenital as by genital contact. In the United States, however, the disease, though universally distributed, probably affects a smaller proportion of the community than in most countries; and, inasmuch as we do not indulge in kissing and other forms of personal endearment so much as our European neighbors, extragenital infections are relatively uncommon

among us. Fournier estimates that in France syphilis is acquired through sexual intercourse in from ninety to ninety-three per cent of all cases. In this country the proportion is probably higher. Inasmuch as the initial symptom of syphilis —the chancre—occurs at the point of inoculation, the proportion between genital chancre and extragenital chancre may be taken as fairly indicative of the frequency of sexual infection.[1] Montgomery[2] finds 5.5 per cent of extragenital infections among 1,217 cases; I find a little less than four per cent in 2,400 cases. Montgomery adds the following data: Bulkley (New York), 5.5 per cent; Finger (Vienna), 1.3 per cent; Wracek (Vienna), 7.5 per cent; Van Walsen (Amsterdam), 8.5 per cent; Fournier (Paris), and Broich (Germany), 9 per cent; Krefting (Christiania), 15.6 per cent; Russia, 75 to 80 per cent.

Among some 2,200 syphilitic men I find but 70 instances of extragenital chancre; while among 207 syphilitic women, 21 showed or gave history of extragenital chancre. This proportion—about 3.5 per cent for men and ten per cent for women[3]—confirms the accepted belief that women are more often innocently contaminated than men, while the method of contamination is sufficiently indicated by the following table, in which practically all chancres of lip, tongue, and tonsil were due to kissing, while almost all the finger chancres in men occurred in doctors and were due to vaginal examination, only one of them being attributed to infection during a surgical operation. The breast chancres were acquired by nursing syphilitic infants. A chancre of tongue or tonsil may surely

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[1] Buchler, "Report of New York Presbyterian Hospital," 1906, vol. vii, p. 105.

[2] *J. Cutan. Diseases*, August, 1905.

[3] Fournier has tabulated 10,000 chancres (96 per cent of them in men), of which 94 per cent were genital in man, and only 63 per cent in women.—They kiss everyone in France.

be acquired by simple lip contact with an infected surface, just as urethral chancre may result from normal coitus.

EXTRAGENITAL CHANCRE

TABLE I

Male	70 cases	*Female*	21 cases
Finger	34 "	Lip	13 "
Lip	24 "	Finger	2 "
Tongue	4 "	Vaccination	2 "
Tonsil	2 "	Breast	2 "
Abdomen	2 "	Tonsil	1 case
Cheek	1 case	Eyelid	1 "
Chin	1 "		
Eyelid	1 "		
Arm	1 "		

Dr. Bulkley has collected from various sources a larger table of extragenital chancres, showing their distribution as they are likely to occur in general practice, with the exception that vaccination and other surgical operations are no longer common causes.

TABLE II

Lip	1,810 cases	Perigenital region	77 cases
Breast and nipple	1,148 "	Legs and thigh	73 "
Buccal cavity	734 "	Forearm	59 "
Fingers and hand	462 "	Neck	47 "
Eyelids and conjunctivæ	372 "	Gums	42 "
Tonsils	307 "	Forehead and temple	37 "
Throat (deep oral and nasal)	264 "	Ears	27 "
Tongue	157 "	By vaccination	1,863 "
Chin	146 "	By cupping and phlebotomy	745 "
Cheek	145 "	By circumcision	179 "
Trunk	100 "	By tattooing	82 "
Nose	95 "		
Anus	87 "	Total	9,058 "

Among the lower classes extragenital chancres are much more frequent than among the wealthy, and occur in curious

locations because of strange methods of infection. Thus I find recorded the case of a young woman who developed a chancre on the forearm from carrying an infant with mucous patches and condylomata about the anus.

It is quite futile to attempt a classification of the various ways by which syphilis has been or might be transmitted outside of sexual relations. We still occasionally hear of little epidemics caused by physicians, or by midwives whose infected fingers spread the disease, while mediate contagion caused by infection from some object by which the secretion of a purulent syphilitic sore has been recently deposited is the cause of sporadic outbreaks. Thus Fournier narrates cases of infection transmitted to a child by its uncle who blew his syphilitic child; and this exception is fully compensated for by 3 other case in which a kindly lady bound up the scratched knee of a little child with her handkerchief saturated with saliva. One of my cases of mouth infection was attributed to sucking a pencil known to have been previously used by a man in florid syphilis. Epidemics have been caused by dental instruments, obstetrical instruments, Eustachian catheters, blowpipes, and anything which may pass from one mouth to another, such as pipes, cigars, cups, spoons, etc. In a few instances washerwomen have been infected by clothes befouled with virus. Vaccination syphilis and circumcision syphilis have been abolished by the elimination of human vaccine and of the orthodox sucking of the circumcision wound.

The practical conclusion to be drawn from what we know of extragenital syphilis is an appreciation of the danger of taking into one's mouth anything which has recently been in the mouth of another, or of kissing not wisely, but too promiscuously (Cases I and II, page 4). For, unhappily, syphilis may be transmitted not only to an innocent person, but also from an innocent person—i. e., from one who does not know

that he has the disease or who thinks that its active manifestations are past.

Although the disease may only be transmitted by inoculation of the secretions from the primary or secondary lesions, and although, theoretically at least, the possession of an unbroken integument protects from danger of infection with syphilis, one can never be absolutely sure that a patient in the first two or three years of syphilis has not some insignificant lesion of mouth or genitals whose presence he does not realize, nor can one be sure of an absolutely unbroken integument. It cannot be too often repeated—syphilis is frequently transmitted by a person who believes he has no lesions of syphilis upon him to a person who believes his integument intact.

AGE OF INCIDENCE

Syphilis is primarily a disease of young adults. I have tabulated 1,775 cases in men and find that only 1.3 per cent (excluding hereditary syphilis) began under eighteen; 5.2 per cent began between eighteen and nineteen; 55.2 per cent between the ages of twenty and twenty-nine; 17.9 per cent between thirty and thirty-four; 10.4 per cent between thirty-five and thirty-nine; 5.8 per cent between forty and forty-four; and 4.2 per cent above forty-five. The actual percentages between the ages of eighteen and thirty-six are the following:

18 years old	1.1 per cent.	28 years old	4.6 per cent.
19 " "	3.3 " "	29 " "	4.3 " "
20 " "	5.4 " "	30 " "	5.1 " "
21 " "	5.4 " "	31 " "	3.5 " "
22 " "	5.6 " "	32 " "	3.1 " "
23 " "	7.0 " "	33 " "	3.1 " "
24 " "	6.8 " "	34 " "	3.1 " "
25 " "	5.9 " "	35 " "	2.8 " "
26 " "	5.6 " "	36 " "	1.9 " "
27 " "	4.6 " "		

Eighty per cent, therefore, begin between the ages of nineteen and thirty-five.

Among prostitutes the average age of incidence is about seventeen years (in France), while reputable women usually acquire syphilis at the time of matrimony.

THE PROBABILITY AND POSSIBILITY OF INFECTION

Nothing is certain in syphilis except its uncertainty. And if at the outset of the disease we are unable to prophesy what its symptoms will be or whether it will be mild or severe, still less are we competent to state when a given case will cease to be infectious. A host of practical queries on this subject are constantly clamoring for answer. For example: "I have been exposed to syphilis; am I infected?" or, "I have exposed another; is he infected?" or, "I am pregnant; will the child be syphilitic?"

Does Exposure Necessarily Imply Infection?—No; emphatically not. The physician who has the confidence of his patients will occasionally come upon cases where exposure, even repeated exposure, has not resulted in infection. I have recently seen three striking instances of this.

1. A married man who cohabited with his wife constantly throughout the active period of his syphilis; yet she was not infected.

2. A woman who cohabited indiscriminately with three men, one who had infected her and two others; but, though she had various lesions, the two escaped.

3. A woman who cohabited almost daily with a man during the two months from the appearance of his penile chancre to the eruption of his secondary syphilis. I have watched her closely for a year, and as yet she shows no sign of syphilis.

But such cases by no means tell the whole story. To acquire syphilis but one exposure is requisite. A syphilitic lesion

in contact with an abrasion, however minute, and the deed is done. The abrasion need be only microscopic, the lesion unsuspected, the contact momentary. Indeed, clinically, any one of these three conditions may be apparently lacking.

1. It is exceptional for the victim to remember any abrasion at the time of inoculation.

2. As a rule, examination of a person who has infected another with syphilis reveals infectious lesions in considerable number; but occasionally no trace of syphilis can be discerned. The transmission of syphilis by patients who *think* themselves clean is a common occurrence.

3. Exceptionally it is impossible to identify the culpable contact. No history of exposure is obtainable; yet the patient has syphilis. If, under such circumstances, genital chancre exists, the patient's veracity may fairly be doubted; but if the chancre is extragenital the mystery remains unexplained.

What is the Probability of Infection from Contact by Kissing or Sexual Intercourse with a Person Known to be Syphilitic?—This question cannot be answered by statistics. The cases quoted above prove the possibility of escape under almost any clinical circumstances, while the converse proposition that a single brief contact, even if followed by immediate and thorough washing, may prove infectious, is daily exemplified. The only attitude permissible for a medical adviser is one of complete impartiality. If your patient has been exposed, calm his fears without too much fortifying his hopes, and bid him wait the dreary weeks of probation. Examine the suspected partner, if possible, but do not put absolute reliance upon the positive or negative results of such an examination.

The following set of cases related to me by Dr. John F. Connors may serve as an example: Eleven men were repeatedly and in rotation exposed to infection from a single woman. They thought themselves well and she was not obviously diseased. Yet six of them contracted gonorrhea and chancroid,

four syphilis and chancroid, and one chancroid alone. Total, chancroid, one hundred per cent; gonorrhea, fifty-five per cent; syphilis, thirty-six per cent.

When Does Syphilis Cease to be Infectious?—Since the probabilities are so vague, what are the possibilities? The question is by no means easy to answer. Certainly eight out of ten syphilitics cease to be infectious within three years. Certainly 99 men out of 100 cease to be infectious within four years. Certainly the proportion of infections from syphilitics of more than five years' standing is infinitesimal. Yet one cannot expect the victim to care whether he is one of two or one of a million. Mathematics do not ameliorate the disease, and a five-year old syphilis, if it infects at all, infects just as virulently as a fresh case. We are in crying need of some criterion to prove that the last spirocheta in a given patient is dead, and that need is not yet answered.

SYPHILIS AND MARRIAGE

My records of marital infections have some bearing upon this point. (I include only those who took no precautions to prevent contamination.) Among 27 patients who acquired syphilis when married, or married within a year after acquiring it, 25 infected their wives (or husbands) and only 2 did not. Of those marrying during the second year of the disease, 10 proved infectious, 4 not so. In the third year 4 were infectious, 16 not so. In the fourth year 1 was infectious, 4 not so. Beyond this it has been impossible to estimate the proportion of those who escaped; but 2 infections are recorded in the fifth year and (an alleged) 1 in the seventh.

In 41 cases it is certain that the wife was infected during the first year of exposure (almost all during the first few months). In one case the husband's syphilis had been treated but a few weeks, and was three years old when he married;

yet the wife was not infected until one year later, after the birth of a healthy child (last seen well at the age of seven). The second child was born syphilitic four years after matrimony. No other instance of delayed marital infection has come under my notice (page 68).

Thus the danger of marital infection is instant, and the chances for it are at least 12 to 1 during the first year of the disease, 5 to 2 in the second year, 1 to 4 in the third year, and all but nothing after the fourth year—whether the patient has been well treated or not.

Most practitioners are willing to assure their patients that after three years of treatment all danger of transmitting the disease is past. Yet this is by no means an absolute rule. Though none of my cases of late infection was derived from patients who had been well treated, there are many such recorded. Fournier even goes so far as to state that though the mercurial treatment of early syphilis diminishes the danger of tertiary symptoms in proportion to its thoroughness, "the frequency of late secondary (infectious) manifestations [1] increases in proportion to the duration of treatment." While I cannot accept this conclusion, it is impossible to deny the thesis he has so forcefully maintained in "Syphilis et mariage" and "Syphilis secondaire tardive" that infection may occur not only after a thorough course of treatment and after the third year of the disease, but even after the fifth, the tenth, perhaps the twentieth year. Tarnowsky has reported [2] the transmission of syphilis in the fifth, sixth, ninth, tenth, and fifteenth years. Neumann [3] accepts the possibility of contagion between the fifth and the tenth year, and perhaps later. Fournier relates three infections during the sixth year, one in the seventh, six in the eighth, three in the ninth, two in the tenth; but at this

[1] See page 87.

[2] Third Internat. Congress of Dermatology, 1896.

[3] *Wien med. Presse*, 1899.

point even Fournier falters. He relates cases of supposed infection at the end of twelve, thirteen, and seventeen years; but confesses that "in view of the complexity of the subject and the great possibility of error in so delicate a matter, these cases are as yet too few to warrant any conclusion being drawn from them."

But Fournier is an extremist. He stands almost alone in his horror of late infections. Twenty years ago he acquiesced in matrimony at the end of five years of syphilis; to-day he says: "If my son contracted syphilis, I should not permit him to marry before the sixth or seventh year of his disease." [1] We can afford to be a little milder and assert that matrimony is often safe and sometimes justifiable at the end of three years, but unless the social elements at stake are very great it is more prudent to follow the rule that *marriage of a syphilitic is permissible only after five years, during the last two of which he has been without symptoms and without treatment.* The occurrence of symptoms, especially if they be secondary symptoms, after the third year of the disease should postpone matrimony until two years have elapsed since the termination of the treatment required to cure these symptoms.

The reason for requiring two years of health is this. Late infections, like early ones, are due practically always to contamination by secondary lesions in the mouth and upon the genitals. Of the eighteen cases related by Fournier in which infection occurred between the sixth and seventeenth years of the disease, ten were buccal and eight genital infections. Now these lesions are likely to relapse persistently and without long intervals of health.[2] Consequently the lapse of two years without such lesions is a sufficient guarantee that they will not recur. If the patient is an inveterate smoker, he should be

[1] *Bull. de la soc. franc. de prophylaxie san. et mor.*, 1906, vol. v, p. 125.

[2] Whereas tertiary non-contagious lesions often relapse after many years of health.

warned of the danger of relapsing lesions of the tongue to which his habit subjects him (page 130). Curiously enough several patients had been repeatedly warned of this danger before they succeeded in infecting their wives (see Case XVII, page 94).

Finally, be it remembered, the moral aspect of this question must carry fully as much weight as the physical. To prohibit matrimony in a given case may wreck a man's life even more completely than syphilis could blast his wife's, and though this consideration can have no force in the first two years of the disease when infection is all but certain, in the fourth and fifth years one may make exceptions for adequate social cause and with due precautions, deeming the possibility of infection light in comparison to the certain despair implied by delay. After the fifth year it is wiser even to urge matrimony, for nothing so completely disarms syphilis of its terrors as the possession of a calm fireside, a happy wife, and a ruddy child. Many a man has been driven to this happiness like a whipped cur, and has found in it a fullness of content which the medicines and maxims of no physician could provide.

CHAPTER VI

SYPHILITIC INHERITANCE

SYPHILIS AND MATERNITY

Syphilis in woman differs from syphilis in man in many respects, but nowise more than in its infectiousness. Syphilis in the male ceases to be transmissible to wife or to child [1] within five years in practically every instance and under any circumstances of neglect and inefficient treatment. But syphilis in woman, though it ceases to be transmissible by contact quite as early as in man, may continue transmissible by heredity for an indefinite number of years. Thus syphilitic children —aborted, stillborn, dying in infancy, or living in spite of the infection—may follow one another for ten, fifteen, even twenty years. In so long a series syphilis plays, as might be expected, the same pranks as in its course in the individual. Thus:

1. Most exceptionally, the woman recently infected with syphilis and untreated may bear only healthy children.

2. As a rule, the recently infected woman bears at least one syphilitic child, but, if well treated (probably) bears healthy ones thereafter.

3. If not treated, she usually has several miscarriages between the fourth and seventh months, two or three children who die at birth or within the first three months of their lives, and others who survive—syphilitic.

In general, the children are less and less infected as time

[1] Unless we accept the theory of paternal heredity, the reasons for doubting which are stated in another place (page 69).

elapses. The abortions precede the stillbirths, the stillbirths the syphilitic children, the syphilitic children the healthy ones. But this sequence is *never* absolute. It may be brief (consisting of but one or two miscarriages or one or two syphilitic children and nothing more, though the mother remains untreated): it may be indefinitely prolonged. It usually lasts from four to six years; but during this time abortions, syphilitic children, and healthy children alternate in a most confusing succession. It is even possible for one of twins to be healthy, the other infected.

This confusion in heredity resembles and doubtless reflects the alternation of storm and calm in the mother's disease. Just as the first eruptions are likely to be the most infectious, so are the first children likely to be the most infected. But periods of calm may intervene between infected children, just as between infectious lesions. It is to be noted, however, that periods of calm in the mother's disease may coincide with the production of syphilitic children; though the mother's disease may appear absolutely latent, the child may be—and often is—none the less virulently infected.

4. If well treated, the mother ceases to bear syphilitic children during the administration of treatment; but she may resume her tendency to bear infected children after the cessation of treatment, just as she may resume the tendency to show evidences of the disease.

5. But if well treated in the first three years, the chances of her bearing syphilitic children thereafter are slight.

Let me illustrate these general rules with a tabulation of cases observed by my father and myself.

Among 43 women innocently infected with syphilis in matrimony, and who bore children after said infection, only 2—think of it! only 2—escaped bearing at least one syphilitic child; and this exception is fully compensated for by 3 who, before realizing they had the disease themselves, infected

a child whom they had previously brought into the world healthy.

Of the infecting 41

23 bore 1 syphilitic child (or fetus), of whom 5 bore healthy children later.
8 bore 2 syphilitic children, of whom 6 bore healthy children.
4 bore 3 syphilitic children, of whom 2 bore healthy children.
2 bore 4 syphilitic children, of whom 2 bore healthy children.
3 bore 5 syphilitic children, and no others.
1 bore 8 syphilitic children, and 6 healthy ones.
Total, 41 bore 86.

The last was the poorest and most ignorant of the lot: she should really be classed as a hospital case.

Doubtless, many more of those who bore but one syphilitic child bore healthy children after they had passed from our treatment and observation, just as many of their sisters who acquired syphilis without a husband have gone through their treatment at our hands, and then married and borne perfectly healthy children.

Why this difference? Why does the unmarried syphilitic woman so commonly become competent to bear healthy children while syphilis of the wedded victim almost invariably impairs the health or destroys the life of at least one child?

The difference lies in knowledge and the lapse of time. The innocent wife does not realize she is diseased until she brings into the world her first syphilitic child: does not perhaps realize it even then—witness the frequency of multiple syphilitic heredity—but the woman forewarned and forearmed is in little danger. Our statistics enforce this point: In 37 cases the interval between infection of the mother and the birth of the first syphilitic child (or fetus) is known; in 32 it was a year or less, thrice between one and two years, and twice at two and a half years. On the other hand, the interval before the birth of a healthy child was twice two years, ten times three years, twice five years, once seven years, and once ten years.

Moreover, the duration of the mother's disease at the birth of the last syphilitic child (as far as known) is shown to fall rapidly by the figures in Column IV. of the table (below).

Let us compare the infectiousness of syphilis between husband and wife and between mother and (unborn) child in the following table, of which Column I (duration of matrimony before infection of wife) compared with Column II (duration of mother's disease at time of birth of first syphilitic fetus) shows the instant infectiousness of syphilis; Column III (duration of disease in husband at the time he married and infected his wife) compared with Column IV (duration of disease in mother at time of birth of last syphilitic fetus) shows the great decrease of infectiousness in the third year and its more gradual disappearance thereafter; Column V (duration of syphilis in man before marriage to wife whom he did *not* infect) compared with Column VI (duration of disease in mother before birth of first *uninfected* child) shows the opposite side of the picture, the sharp rise of non-infection in the third year.

Year	I	II	III	IV	V	VI
1, or less.....	41	31	25	16	2	0
2.....	1	3	10	9	4	2
3.....	0	2	4	6	16	10
4–5.....	0	0	3	6	(1)	(1)
6–10.....	0	0	1	1	(1)	(1)
11–13.....	0	0	0	2	(1)	(1)

It seems a fair inference, therefore, that the probability of syphilitic heredity, like the probability of contact infection (in matrimony) is overwhelming in the first year of the disease, great in the second and third years, slight in the fourth and fifth years, and negligible thereafter. But, as might be expected, from the more intimate contact between mother and unborn

[1] Returns in these later years too few and scattering to be conclusive.

child, the danger of syphilitic inheritance persists somewhat longer than the danger of contact infection.

THE THEORY OF SYPHILITIC HEREDITY

It would require many pages to elaborate the arguments employed and the instances adduced by every author who has discussed the theory of syphilitic heredity to prove that this disease may be inherited from the father alone, the mother remaining uninfected.[1] The opposite view, i. e., that every syphilitic child has a syphilitic mother, has practically no defenders except Sturgis [2] and Matzenauer.[3]

The facts of the case are these: the child (fetus) may conceivably be infected with syphilis in one of five ways:

1. At the moment of conception by the father's semen (paternal heredity).

2. At the moment of conception by the mother's ovum (maternal conception heredity).

3. By means of the placental circulation to the healthy fetus (maternal post-conceptional heredity).

4. To the child at birth from lesions of the mother's genitals (infection in parturition).

5. To the infant after birth either from its parents (e. g., by a kiss or in suckling), or from an indifferent person (e. g., a wet-nurse).

In these two latter instances (4 and 5) the disease in the infant is acquired syphilis, differing from hereditary syphilis in the presence of a chancre at the point of inoculation, and the absence of the grave visceral lesions of fetal syphilis.

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[1] Rosinski ("Die Syphilis in der Schwangerschaft") offers the latest contribution to this side of the argument.

[2] Morrow's "System."

[3] *Archiv f. Dermat. u. Syph.*, 1903; *Wien. med. Presse*, 1903, vol. xliv, pp. 284, 657, 805, contains a debate on this subject between Finger and Matzenauer.

Paternal Heredity.—Given a man with infected semen, it is probable that he will infect both wife and child, but conceivable that he may infect either without the other. Actual examples of such exception occasionally appear. Thus an untreated, actively syphilitic mother has been known to bear an apparently healthy child, though such an occurrence is to the last degree exceptional (page 69). *But that a syphilitic child should issue from an apparently healthy mother, though unusual, is by no means rare.* (It occurs in about fifteen per cent of cases.)

This purely paternal inheritance has been and is almost universally attributed by syphilographers to infection of the fetus from the father's semen, the mother escaping infection from fetus as well as from semen. But pathologists have always maintained at least an attitude of doubt concerning this unique seminal transmission which is not even alleged for any disease except syphilis. Let us, therefore, array the evidence upon each side.

In favor of paternal heredity is alleged the obvious fact that father and child are certainly syphilitic, the mother apparently sound.

Against it is urged that the mother is not absolutely sound; and this is exhibited in various ways. In the first place, she is subject to *Colles's law*, i. e., *the apparently healthy mother of a syphilitic child cannot be infected by that child*, whether in nursing it or by any accidental contact; the child may infect any non-syphilitic person except its own mother.

This immunity to syphilis exhibited by the mother of a syphilitic child means that she is in some sense syphilitic. But in what sense? Either she has absorbed from the fetus some immunizing substance (or developed this herself in the excretion of the syphilitic products of the fetus), or, on the other hand, she is actually syphilitic. In favor of the former theory are:

1. Certain reported exceptions to Colles's law, the mother developing chancre of the nipple and a general syphilis *after* nursing her infant,[1] and

2. The repeated insistence of almost innumerable observers that in such instances further syphilitic pregnancies may be prevented by *antisyphilitic treatment of the father alone.* The cases cited in illustration are as numerous and straightforward as the instances of exception to Colles's law are few and obscure.

Against it are:

1. Our inability to evoke a similar experimental immunity in man or monkey.

2. The fact that some nine per cent of syphilitic women do not have, or at least do not remember having, any early symptoms of the disease (page 78).

3. The frequency of *choc en retour*,[2] i. e., the appearance in later years of tertiary syphilitic lesions upon these apparently clean mothers. In such instances at least we may fairly say that the mother was syphilitic, but skipped her early symptoms; and

4. The decreasing nocivity of hereditary syphilis. It is well known that whether the syphilis in the parent be paternal or maternal, there is a distinct though irregular tendency toward lessened gravity of heredity syphilis in successive pregnancies. Thus in a typical untreated case two or three miscarriages of macerated syphilitic fetuses are followed by or intermingled with children stillborn at term or born so gravely infected as to die within the first few weeks of extra-uterine life.

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[1] So obscure are these alleged exceptions that Matzenauer rejects them all; Finger clings to a few; and Hochsinger believes them so frequent as to prohibit the apparently healthy mother from nursing her syphilitic infant!

[2] This term represents the theory that in these cases the fetus is infected by the father, the mother by the fetus; a distinction that seems to me both futile and unnecessary.

Then follow children less gravely infected, and finally the taint is eliminated and healthy children are born. When the syphilis is purely paternal, the sequence, though often briefer, is otherwise similar to that of maternal heredity.

Now this decreasing intensity of infection is commonly attributed to the decreasing virulence of the father's syphilis, although syphilis, like any other contagious disease, is entirely uninfluenced in its severity by the source from which it is acquired, and is grave or mild solely according to the susceptibility of the individual in whom it exists.

But the one thing most likely to affect the vitality of the fetus and its susceptibility to disease is the condition of the placenta; and Matzenauer has attempted to show that the decreasing virulence of syphilis in successive children goes hand in hand with decreasing syphilitic inflammation of the placenta. Accordingly, if one accepts the infection only from the mother, one can readily understand how her first children succumb more fully to the disease because of the more intense lesions in the placenta; while, as time goes on, the placenta, with the other maternal organs, becomes less and less likely to be diseased.[1]

The array of probabilities against a purely paternal heredity —fortified by the improbability of the mother escaping so virulent an infection in the fetus, the uniqueness of this alleged syphilitic seminal heredity and the impossibility of artificially producing maternal immunity—impresses me more cogently, I confess, than the purely clinical observations alleged in its favor. That paternal heredity is less frequent than would seem is surely proven by the frequency of *choc en retour*;[2] that it ever occurs I do not know.

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[1] The more prolonged and severe infectiousness of an obviously maternal syphilis is even more readily explicable by this theory than by the other.

[2] The discovery (page 29) of spirochetæ in the inguinal glands of an apparently healthy mother simply enforces this lessened frequency, as does the case

Maternal Conceptional Heredity.—Although from three fourths to four fifths of syphilitic heredity is directly traceable to syphilis of the mother, and although in most instances the mother is known to be syphilitic at the time of impregnation, it is difficult if not impossible to assure oneself whether the infection reaches the fetus through the ovum (conceptional), or through the placental circulation (post-conceptional). The objection of decreasing hereditary virulence urged in the preceding paragraphs against paternal (conceptional) heredity applies with almost equal force against maternal conceptional heredity. For if the fetus is infected from the ovum, it is difficult to understand why the second or third or fourth ovum should, if infected at all, be any less virulently infected than the first. Whereas a decreasing intensity of placental inflammation permits better and better nourishment of the fetus and by so much retards its disease. For all syphilitic fetuses, therefore, except those aborted in the first few months, one may doubt a conceptional heredity.

Maternal Post-Conceptional Heredity.—A fetus may be infected with a syphilis acquired by the mother during the first seven months of gestation. It is extremely doubtful whether a mother infected after the seventh month can transmit the disease to the fetus *in utero*, inasmuch as the disease would scarcely have time to be generalized in her (at any rate there are no conclusive cases in proof one way or the other). Such infection is the rarest form of hereditary syphilis as far as clinical records go; for the instances of fetal infection from which conceptional transmission (antecedent syphilis) both paternal and maternal can be excluded, are rare indeed. Yet following out the line of argument suggested above, it seems highly probable (though by no means absolutely proven) that

(related to me) of an apparently healthy woman continuing to bear syphilitic children to a second (healthy) husband after the death of the first (syphilitic) one.

maternal post-conceptional heredity is almost (if not altogether) the sole type of syphilitic heredity. The theories of conceptional heredity are founded upon evidence so exclusively clinical,[1] and are opposed so absolutely by our experience with every other disease that they merit at best a guarded and conditional acceptance.

Such is the etiology of hereditary syphilis as far as we know it.

Acquired Syphilis in Infancy.—No age is immune to syphilis. There does not seem to be even a relative immunity to the disease during infancy as there is to so many of the acute exanthemata. But it has been alleged, and was for a time generally believed, that the non-syphilitic child of a syphilitic mother could not (during infancy, at least) be infected with syphilis by that mother (or by anyone else). This rule, known as *Profeta's law*, has helped, by its mystery, to illustrate and to obscure the interpretation of Colles's law. But Profeta's law is so full of exceptions that the syphilographer of to-day adheres to it in little more than this sense—that if the child has escaped intra-uterine infection it may well hope to escape the lesser dangers of extra-uterine infection.

The alleged cases of infection during parturition are rare and doubtful. But during infancy the healthy child of syphilitic parents may, doubtless, be infected quite as readily as another. Apparent exceptions (examples of Profeta's law) are due either to the relatively slight infectiousness of the disease (page 59) or to the fact that the infant is already infected with (occult) hereditary syphilis.

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[1] And, one might add, so prejudiced—for since Kassowitz opened the study of this subject in 1876, with his masterly thesis in favor of paternal heredity, it has never been thoroughly discussed from the opposite point of view until Matzenauer's recent publication.

HEREDITARY SYPHILIS OF THE THIRD GENERATION

Inasmuch as a man's capacity to transmit syphilis to his wife almost always ceases within five years and a woman's capacity to bear syphilitic children within ten, it would seem to the last degree improbable that a child born syphilitic should retain the power of transmitting the disease until it reaches maturity. Yet such is the postulate of those who maintain that the hereditary syphilitic may in turn transmit the disease to his descendants. Improbable as such inheritance may seem, hereditary syphilis in the third generation (syphilis twice inherited) is accepted and instanced by a formidable array of authorities. Hutchinson denies it,[1] Taylor [2] and E. Fournier [3] accept it without reserve. Tarnowski,[4] Barthélemy,[5] Jullien,[4] and Finger [6] are inclined to believe in it, or believe at least that some of the stigmata are transmissible to the third generation.

E. Fournier has formulated the following scheme of requisites to the proof of a case:

I. The grandchild must—

1. Show an unmistakably syphilitic lesion or a dystrophy that can be fairly (*légitimement*) attributed to a syphilitic taint.

2. Be free from suspicion of acquired syphilis.

II. The parents of the grandchild—

3. One of the two must be indubitably affected with hereditary syphilis.

4. This parent must be free from suspicion of a superadded acquired syphilis.

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[1] *Med. Press and Circular*, 1906, August 1.

[2] *Med. Record*, 1906, vol. lxix.

[3] "Hérédo-syphilis de seconde génération," Paris, 1905.

[4] "Thirteenth International Congress," Paris, 1900.

[5] "Twelfth International Congress," Moscow, 1897.

[6] *Wien. klin. Wochenschr.*, 1900.

5. The other parent must be free from suspicion of acquired syphilis.

III. Finally, of the grandparents—

6. At least one of them must be proven to have had acquired syphilis.

It is questionable whether a chain of evidence so long, so purely clinical, can be accepted. So much depends upon the point of view of the reporter; so impossible is it for anyone to *prove* the capital conditions, 4 and 5.

Indeed, the conclusions of those who accept syphilis of the third generation illustrate by their diversity the frailty of the evidence. Thus Tarnowski says (referring to endemic syphilis):

"Parents showing symptoms of hereditary syphilis . . . do not transmit syphilis to their children as a hereditary disease (*sous la forme héréditaire*).

"The dystrophies[1] of the second syphilitic generation are not always transmitted to the third generation as identically the same dystrophies.

"Children of syphilitic parents inherit a certain immunity to syphilis (Profeta's law[2]); but this hereditary immunity is but temporary; generally (with rare exceptions) it ceases at infancy or at the age of puberty.

"At this age the individual, though born of syphilitic parents, becomes vulnerable to syphilis. This new infection I call *syphilis binaria*. I have frequently observed it, especially in rural (endemic) syphilis.

"Hereditary syphilis in the third generation is generally due to *syphilis binaria*.

"The third generation may also show symptoms of hereditary syphilis, in case (though this is relatively unusual) a person with hereditary syphilis marries one with acquired syphilis."

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[1] Cf. page 532.

[2] These children are presumed to be syphilitic. Hence Tarnowski's interpretation of Profeta's law is simply the assertion that, so long as an individual is syphilitic, he cannot catch the disease again.

In contrast with the implied frequency of this inheritance in Tarnowski's eyes we may place the experience of Barthélemy and Taylor who, though firm believers in the theory and favored with an immense clinical experience (and not leaning upon the ingenious theory of *syphilis binaria*) have reported respectively only five and two cases. Finally, E. Fournier denies the *theory* of *syphilis binaria*, while admitting the *fact*, reports in detail 116 cases (of which 6 are his own and 6 his father's—from among the tens of thousands of syphilitics seen by Professor Fournier), and concludes that:

"1. Hereditary syphilis of the third generation (*syph. héréd. de 2de génération*) exists.

"2. It is rarely noted clinically because of the impediments to its recognition.

"3. The mortality in this generation is such as to kill two fifths of its members.

"4. It usually shows itself—in four fifths of the cases according to my observations—by dystrophic stigmata similar in every way to those of hereditary syphilis (*hérédite prime*).

"5. It sometimes exhibits symptoms of syphilis (*symptômes virulents*), but far less frequently—fourteen per cent, according to my observations."

In face of such a general agreement as to principles, and in spite of the divergent interpretation of details, one must at least confess that hereditary syphilis in the third generation is possible, though it must be eminently rare. And one may add by way of precaution that the other factors in heredity —especially in the production of congenital dystrophies—such as alcohol, privation, etc., whose influence is so marked upon syphilis in the first and second generation, must be important elements, and are perhaps even *the* important elements in determining syphilitic heredity—especially dystrophic heredity—in the third generation.

CHAPTER VII

THE COURSE OF SYPHILIS

THE ONSET

ALTHOUGH utterly irregular in its course, syphilis is practically always quite characteristic in its beginnings (at least in the male), and thus the diagnosis of syphilis is happily most readily made at the time when it is most necessary to make it.

Onset in Man.—In order to get a picture of the way in which syphilis begins let us take, for example, a typical case in a man. He has intercourse on a given date (and may notice an abrasion upon the penis, or there may come out within the next few days a crop of herpetic vesicles. Yet, in the great majority of instances neither of these things is noted). He thinks himself entirely normal and notices nothing wrong for a period of three to five weeks. Then he accidentally becomes aware that there is upon the glans penis or upon the foreskin a single pimple. This grows and becomes quite hard, and is eroded or ulcerated upon its surface. In a week or ten days the glands in one or both groins begin to swell, yet the sore itself and the glands are practically painless, and, as neither of them attains any great size, the patient may neglect to consult a physician. But, though relatively insignificant, the sore does not get well; the glands do not disappear. Three or four weeks go by and the ulceration upon the surface of the sore gradually heals, but a hard, typical lump remains under the epidermis. Then, in from two to three

months from the time of infection, the first general symptoms appear.

First, he begins to feel a little miserable; his bones ache; he feels perhaps a little feverish, but probably not definitely sick, and may take no very great account of his malady until he notices a rash upon his body or some sore spots in the mouth or pharynx. These are pretty sure to bring him to the physician, who finds him with a characteristic macular, papular, or polymorphous eruption (to be described in detail later), with little sores in the mouth or pharynx, with disseminated, minute, scabbed ulcerations in the scalp, perhaps with a slight evening rise of temperature, and tender sternum and shins. He is in full secondary syphilis. The diagnosis is unmistakable.

Onset in Woman.—Such is the course followed by almost all men. In women the onset of the disease is by no means so characteristic. The chancre is so slight a lesion that it may very well be overlooked by the patient, and she may fancy herself entirely well until, two or three months after infection, she begins to feel run down. She may have enough fever to fancy she has malaria, or she may be treated for an imaginary typhoid; or she may suffer from frightful pains in her bones, in her head, these pains coming on in the evening and much worse at night than during the day. A short while after the beginning of this outbreak of general toxemia she may show lesions of skin and mucous membrane quite as characteristic as those found in the male. But in a certain proportion of cases (page 45) these objective evidences are so faint as to be overlooked and, consequently, she does not present a typical and unmistakable picture of the disease. In such cases repeated miscarriages, or the birth of children that promptly die, may constitute the only early symptoms.

If the diagnosis is not made, her symptoms may gradually abate, and it may be years before a tertiary outbreak proves

that she has syphilis. Or she may go on to have some characteristic lesion within a few weeks or months.

DURATION OF PRIMARY AND SECONDARY INCUBATION

Such is the beginning of syphilis as we see it clinically. It may be divided into two periods: the primary incubation, ending with the appearance of the primary lesion—the chancre—and the secondary incubation, ending with the appearance of the secondary lesions.

But this beginning is subject to certain variations, the clinical extent of which may be exemplified by the following statistics.

Lang has collected 45 cases of experimental inoculation with syphilis, in 36 of which the duration of the primary incubation was noted, in 37 that of the secondary incubation, and in 29 that of the total incubation—the time between inoculation and the advent of secondary symptoms. But such cases, though scientifically accurate, do not present the facts as we meet them in practice.

Clinically, we depend for the discovery of the chancre and even of the secondary symptoms, not upon the alert and trained specialist, but upon the casual and unskilled patient who may, and often does, overlook the most characteristic evidence of his disease.

In order, therefore, to contrast the scientific fact and its clinical semblance. I have studied a number of cases in which the various incubations are recorded clinically and place the two tables side by side.

PRIMARY INCUBATION			SECONDARY INCUBATION [1]		
	Experimentally	Clinically		Experimentally	Clinically
5 days	.....	1	1 week	.....	1
7 "	.....	1	2 weeks	2	7
8 "	.....	3	3 "	.....	9
10 "	1	7	4 to 5 weeks	5	105
11 "	.....	1	6 to 7 "	8	123
2 weeks	8	34	8 to 9 "	5	68
3 "	12	52	10 to 11 "	8	10
4 to 5 weeks	14	67	12 to 13 "	4	43
6 weeks	1	21	4 months	4	13
7 "	.....	1	5 "	1	3
8 to 9 weeks	.....	5	6 "	.....	3
10 weeks	.....	2	7 "	.....	1
82 days	.....	1	16 "	.....	1
Total	36	196	Total	37	387

TOTAL INCUBATION

	Experimentally	Clinically
1 month	1	2
1½ months	3	21
2 "	7	49
2½ "	8	30
3 "	5	16
3½ "	2	8
4 "	2	8
4½ "	..	3
5 "	..	1
6 "	1	..
17 "	..	1
Total	29	139

Comparison of the two tables shows the primary incubation to vary between ten days and six weeks actually [2] in every case, clinically in ninety-three per cent; the secondary incuba-

[1] Cases receiving no mercury before the appearance of the secondary symptoms; for if mercury is pushed the secondaries may be postponed or altogether inhibited.

[2] At least as far as the collected statistics show.

tion within five months actually in all, clinically in 98.7 per cent; and the total incubation between one and six months actually in all, clinically in ninety-nine per cent.

These we may, therefore, accept as the extreme limits of the various incubation periods, viz.:

First incubation (inoculation to chancre) = ten days to six weeks.

Second incubation (appearance of chancre to secondaries) = within five months.

Total incubation (inoculation to secondaries) = one month to six months.

How important the delimitation of these periods is can be appreciated only by the prudent practitioner who, convinced of his inability to make an infallible diagnosis from the chancre alone must wait the full limit of time before declaring his patient free from syphilis (page 243).

But if the patient actually has syphilis, it is rarely necessary to wait so long, for clinically the secondary incubation falls within three months in ninety-four per cent, and the total incubation within four months in ninety-six per cent of all cases. What might be termed "the normal case" runs a primary incubation from two to five weeks, a secondary incubation from one to three months, a total incubation from six weeks to four months.

The chancre itself, though usually single, may be multiple in ten per cent to twenty-five per cent of cases (page 224), or extragenital (page 54), or urethral (page 235). In about five per cent of men [1] the chancre is never found and in a far larger proportion of women. The chancre lasts at least two weeks, usually six weeks, sometimes longer.

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[1] Twenty-four out of 549 cases. In many instances the examining physician would undoubtedly have found the chancre had he possessed the skill of an expert.

THE SECONDARY SYMPTOMS

The secondary symptoms, if anticipated by treatment, may never appear. Indeed, the early symptoms, both primary and secondary, are entirely overlooked [1] in one per cent of men (22 out of 2,170) and in nine per cent of women (18 out of 207).

But when they do occur, what is their usual type? their order of occurrence? their intensity? their duration?

Any student of syphilis can answer such questions vaguely, though it is all but impossible, so various are the influences concerned, to answer them accurately. The patient's age and social condition, his constitution (tuberculosis, malaria) and habits (alcohol, tobacco), his willingness and ability to take adequate treatment, and above all else the physician's intelligence in administering treatment—how estimate the results of such diverse influences? To be accurate is surely impossible. Yet it seems not unprofitable to tabulate for what they may be worth the clinical facts recorded upon the office cases of which I have a record.

Definition.—The secondary symptoms of syphilis are twofold, an acute toxemia and certain local lesions varying in character from simple congestion to exudation. These local lesions are not destructive in character, do not invade the adjoining tissues, do not undergo caseation, and on healing leave little or no scar. They have usually a spontaneous tendency to heal.

Differentiation between Secondary and Tertiary Lesions.—Although the local lesions of syphilis are often typically secondary or typically tertiary in type, there is a large intermedi-

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[1] I do not say skipped. In only one man do I know that careful inspection failed to reveal any secondary symptoms following the chancre. Such cases are occasionally recorded but almost invariably develop tertiary lesions in later years.

ate class that taxes the keenest diagnosis. Many a skin syphilid, for example, may begin with a frankly secondary aspect only to degenerate later into an equally typical tubercle or ulcer.

But the difficulty in classifying syphilis of the skin which can be watched and studied day by day is as nothing compared to the errors constantly committed in classifying syphilis of bone and syphilis of the nervous system, for these we know less directly and judge chiefly by their effects. The distinguishing features of these various lesions need not be discussed here; but, be it borne in mind, the differentiation between secondary and tertiary lesions may be difficult and is sometimes impossible.

Secondary Toxemia.—Examination of the blood during the first few months of syphilis, or at least until the disease is controlled by treatment, reveals a secondary anemia, the characteristics of which are described elsewhere (page 254). In the majority of cases in one's private practice this condition reveals itself but by one symptom: viz., loss of weight. Whether the patient's distress and worry over his malady is not often largely responsible for this is an open question. But whatever the exact cause, one very frequently finds the syphilitic ten, twenty, or even thirty pounds lighter at the end of six months than he was the day of his infection. This loss is gradually regained under treatment.

On the other hand, typical, severe syphilitic toxemia with fever, prostration, and various pains, preceding the outbreak of the localized secondary symptoms, is so rare as to be almost always mistaken for malaria or typhoid fever until the eruptive lesions appear and establish the diagnosis, unless, as happened to a youth who came to me in the full glory of a general papular syphilid, it suggests measles.

The long drawn-out debility, with loss of weight as its most striking symptom, is much more severe among women

than among men. The acute, preëruptive febrile toxemia I find noted only 31 times among 826 men and thrice among 86 women; about 3.5 per cent.

Local Symptoms.—The first local secondary symptom appears on the skin in almost every case. Thus, among 826 cases, one showed no secondary symptoms, 772 (ninety-three per cent) began with skin lesions, the remainder with lesions of the mucous membranes.

This first eruption is macular or maculopapular, and generally distributed over the trunk, less marked on the extremities (see page 280). Exceptionally, it is preceded by a few scattered papules.[1]

THE EARLY SECONDARY LESIONS.—But soon a whole group of symptoms appears to form the characteristic picture of early syphilis. The body is covered with macules or papules; the scalp is full of moist crustaceous papules ("scabs," the patient calls them); there are pains in the joints, the bones, the muscles, the head; the mouth and throat are filled with mucous papules and erosions; the lymph nodes, especially the epitrochlear and posterior cervical, become enlarged, while the scar of the chancre with its satellite adenitis still marks the port of entry.

Such is the pathognomonic picture of secondary syphilis at its outset. The picture lasts, in any or all of its constituents, from a week or two to a month or two.

The following list shows the lesions noted at this time. The occurrence of "scabs" in the scalp and of enlarged lymph nodes is not tabulated. The former is all but constant. The latter develop within the first six months, but are by no means always found:

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[1] In five cases, four men and one woman, the first skin lesion noticed was psoriatic in type, in three men it was bulbous, in two others pustular, in one tubercular.

LESIONS AT ONSET OF SECONDARY SYMPTOMS

	Male	Female
Lesions of skin and mouth or throat (pains slight or absent)	520	16
The same with severe pains	131	16
Skin lesions alone	78	10
Skin lesions and severe pains	38	11
Skin lesions and mucous papules about the genitals or anus	5	0 [1]
Skin [2] lesions absent — Lesions of the mouth or throat	40	3
Skin [2] lesions absent — Lesions of the mouth or throat and pains	6	2
Skin [2] lesions absent — Lesions of the mouth or throat and genitals	7	5 [1]
Total	825	63

THE SUBSEQUENT SECONDARY LESIONS.—After the subsidence of this first outbreak the occurrence of secondary symptoms follows no rule. Lesions of the skin and mucous membranes are likely to relapse, but at what intervals one cannot prophesy. A surprisingly large proportion of well-treated cases—perhaps one in five—have no further secondary symptoms. Relapses are most frequent in the first year. Thereafter in a well-treated case they are likely to be few and far between. That there is any period to the relapse, any definite interval of three months or six months, after which a recrudescence of symptoms may be looked for (as some Continental authorities allege), I do not believe.

The farther we go from the beginning of the disease the longer are likely to be the intervals between outbreaks. With the lapse of time the symptoms themselves become less general, less symmetrically distributed, less evanescent, less superficial, less infectious; in fact, they approach more and more the tertiary type.

The following list shows the *rarer* secondary lesions observed during the course of 911 cases. It conveys a general

[1] Modesty has surely diminished these figures as much as cleanliness.

[2] In some of these cases an antecedent skin eruption had doubtless escaped the patient's observation and vanished leaving no trace.

notion of the frequency of these lesions and symptoms, though by no means exhausting the lesions of secondary syphilis:

OCCURRENCE OF THE RARER SECONDARY LESIONS

	Men	Women
Onychia and Paronychia	45	2
Headache (severe)	40	3
Periostitis (marked)	35	1
Iritis	34	5
Deafness	5	0
Pleurodynia	5	1
Laryngitis	3	0
Jaundice	2	1
Tenosynovitis	2	0
Epididymitis	2	0
Nephritis	1	0
Hydrarthrosis	1	0
Choroiditis	1	0
Opacity of the vitreous	1	0
Pigmentary syphilid	1	0
Hemorrhagic syphilid	1	0

Duration of the Secondary Symptoms.—Even to the present day physicians continue to speak of secondary and tertiary periods of syphilis. Despite all that has been written to discourage such a classification they cling to the theory that the secondary lesions of syphilis cease to recur after a year or two, and that every symptom of syphilis thereafter must be tertiary. No belief could be more false or more misleading. How false I shall show in a moment, how misleading we may realize when we consider that the secondary lesions are essentially curable by mercury rather than by iodides, while rarely a late secondary lesion *may be* infectious.

Yet we daily encounter such secondary lesions as the squamous palmar syphilid or the erosive glazing glossitis late in the disease. We meet them constantly as late as five, rarely as late as ten, years after the chancre, and other secondary lesions may rarely, to be sure, yet most indubitably appear

years after the chancre, years after the last tertiary symptom even.

It would take a volume to develop this subject in all its detail. Indeed, Fournier has recently consecrated a volume to it,[1] from which I borrow confirmation to my own observations. First, let me detail a few striking case histories; then a statistical *résumé*:

Case XII.—Relapses of squamous syphilid at six (+) *and twenty-five* (+) *years.* This patient, when seen by Dr. Van Buren on January 15, 1869, at the age of forty, had had "true syphilis—chancre and spots on palms." He was suffering at the time from "pains in the chest, which he calls rheumatism, weakness and slight wasting of the lower limbs, constipation, and difficulty of bladder." All these symptoms had begun in the spring of 1868 with two attacks of acute retention of urine. He was found to be suffering from cystitis, with eight ounces of residual urine, atony of the bowel, and weak and atrophied leg muscles from "inflammation of the spine." "Syphilis of the cord" was diagnosed. Under sounds, regular catheterism with lavage, and mixed treatment, the bladder improved for a time, but soon settled into chronic, complete retention, in which it has remained ever since. With advancing years, legs and bowel have also grown weaker. But there has never been any ataxic gait, girdle pains, or eye symptoms. The patellar reflexes are gone. In short, the condition appears to be one of irregular syphilitic spinal sclerosis.

The only other symptoms of syphilis observed are the following: In May, 1869, "secondary eruption on palms, scrotum, and tongue." In November, 1875, "small nummular patches of dry, livid scales (scaly patches)," cured in a month by mixed treatment and arsenic, then relapsing and followed by "large general excoriations on inside of mouth," which cleared up in January, 1876, under increase of iodid and continuance of arsenic.

In December, 1894, "squamous, circinate syphilid on the sole of the right foot," promptly cured.

He took mixed treatment for about one year in 1869, over a

[1] "Syphilis secondaire tardive," 1906.

year in 1875–76, and for a short period in 1894. He has been constantly under observation and treatment for the bladder paralysis and urinary infection therefrom resulting from 1869 to 1907, and the history is therefore singularly accurate, except in that we cannot learn how long before 1869 the disease began—surely before the acute retention in 1868, perhaps several years before.

Case XIII.—Interval of over fourteen years; frequent short courses of treatment; secondary syphilid of tongue in twenty-third year. Female. Contracted syphilis in 1883, at the age of twenty-three. No chancre noted. Early skin and mouth lesions and treatment. When first seen, in October, 1884, about a year after the commencement of the disease, she had vulvar condylomata. Treatment was continued, and in October, 1885, she had a squamous palmar syphilid. In May, 1886, a mucous papule in the mouth. In July, 1888, palmar papules. In October, 1889, "vulvar lumps." In January, 1894, "rheumatism." In March, 1899, "anemia." In February, 1900, "nervous dyspepsia." In April, 1903, "neuralgia and anemia." In April, 1905, "neuralgia," and there are some indefinite erosions on the tongue. In April, 1906, her mother has died, and the neuralgia and headache are worse. The tongue is distinctly syphilitic, the *glossite dépapillante* of Fournier. She does not smoke.

This patient was constantly under treatment for the first two years of her disease, and although from October, 1889, to April, 1906, she showed no recognizable symptoms of syphilis, short courses of mixed treatment, together with other remedies, were administered on all the occasions noted above in order to calm her fears. A more continuously treated case could scarcely be imagined.

Case XIV.—Relapse of papules in third and fourth years. Occasional treatment. Pustular syphilid in thirtieth year. Chancre in 1869 at the age of twenty-nine. When first seen, three years later (December, 1872), the left pupil was contracted and adherent from iritis and the right one dilated. On buttocks and shins scars of syphilitic ecthyma, on the arms a symmetrical active papular syphilid. On the scrotum, chromophytosis. General condition poor. Previous treatment not noted. He has been treated for paresis on account of some emotional disturbance.

The patient was put on a tonic and mixed treatment. He gradually gained weight, the eruption was cured, relapsed in April, 1873, and in January, 1874. In the fall of 1874 he had gained over twenty pounds, and was well but for a persistent mydriasis of the right pupil. Was seen again in 1890, 1892, 1893, 1894, and given a little mixed treatment to allay various fears.

In September, 1900, he presented "some small ecthyma"; was given mixed treatment again, and in October it is noted "ecthymatous spots well; each leaves a clean-cut, smooth, round, thin scar, and was clearly a specific lesion." Well in December, 1902.

Case XV.—Relapsing erosive lesions of tongue for twenty years. Lip chancre in 1870, at the age of twenty-one. Glands under jaw enlarged; many early mouth lesions. No skin lesions remembered. Early treatment probably adequate.

When first seen, in January, 1890, he reports that for at least ten years past the tongue has almost constantly troubled him. Treatment irregular. Habits as to alcohol and tobacco not stated. Examination of the tongue reveals thin scars along the edges, with here and there active superficial erosions. Under mixed treatment and cauterization with silver nitrate the tongue heals in a month. Treatment continued four months. Relapse on tongue eight months later, in January, 1891; mixed treatment three months thereafter. No further relapse when last seen, in April, 1892.

Case XVI.—Relapse of squamous syphilid and suppurative onychia after twenty-five years. Chancre in 1871, at the age of twenty, followed by slight secondaries, chiefly buccal. Drinks beer heavily and does not smoke. Has taken very little treatment. When first seen, in February, 1896, he shows a scarred and fissured tongue, without active lesions upon it, a suppurative onychia of one finger, and a circinate squamous eruption on the palms and elsewhere. He remains under treatment only two months, at the end of which time he is much improved by mixed treatment ("hands clean") and the cessation of alcohol. Then he takes to drinking again, and disappears.

Here are five cases showing relapse of typical secondary symptoms from twenty to thirty years after the occurrence of

chancre. If they stood alone, unsupported by many others of less and a few of equal duration, unconfirmed by the reports of other observers, they might all be put down to honest but mistaken enthusiasm. But the weight of evidence is too great to be denied. One or two or three such cases may not be syphilitic; but the great majority of them surely are.

To study the duration of secondary symptoms accurately it is necessary, however, to distinguish the extraordinary from the routine, and not to be misled by such cases as these into the belief that any large proportion of syphilitics have secondaries of more than a few years' duration. A fair estimate of the usual expectancy in this regard may be gathered from the following tabulation of successive cases:

DURATION OF SECONDARY SYMPTOMS IN 280 CASES

Less than 1 year	17 cases
1 to 2 years	82 "
2 to 3 years	79 "
3 to 4 years	25 "
Less than 4 years [1]	49 "
4 years	17 "
5 years	6 "
6 years	2 "
7 years	1 case
8 years	1 "
9 years	1 "
Total	280 cases

Thus in the great majority of cases the secondary symptoms cease to recur within three years, in some ten per cent they recur after the fourth year,[2] in some two per cent after the sixth, and surely very rarely after the tenth year.

[1] Duration not quite accurately known.

[2] Fournier estimating from 19,000 cases places this at about 6 per cent. Tarnowski estimates 80 per cent in the first four years, 17 per cent from fifth to tenth; 2.5 per cent from tenth to fifteenth, and 0.5 per cent thereafter.

On the other hand, in tabulating about 2,500 cases, I find late secondaries (i. e., at or after four years) in 227; about nine per cent.

LATE SECONDARIES AMONG 2,500 CASES

4 years	51 cases	17 years	1 case
5 "	44 "	18 "	2 cases
6 "	34 "	19 "	1 case
7 "	13 "	20 "	4 cases
8 "	11 "	21 "	1 case
9 "	14 "	23 "	2 cases
10 "	9 "	24 "	1 case
11 "	8 "	25 "	4 cases
12 "	11 "	30 "	1 case
13 "	8 "		
14 "	1 case	Tenth to thirtieth years	60 cases
15 "	4 cases	Fourth to ninth years	167 "
16 "	2 "		
		Total	227 cases

Of these cases, one half stopped in less than seven years, three quarters in ten years. About one tenth had secondary symptoms after the thirteenth year. This represents surely less than one per cent of all cases; for freaks, seek the specialist.

From all of which we may conclude that while there is no absolute term to relapses of secondary symptoms, no "secondary period" of syphilis, yet secondary symptoms cease within the first four years in about ninety per cent, within six or seven years in about ninety-five per cent. Secondary symptoms appearing later than the tenth year are the rarest exceptions.

Regions Involved by Late Secondaries.—Excepting one case of double iritis, all the lesions recorded affected the skin or the mucous membranes. In the following list there are many duplicates, owing to the fact that a given case may show simultaneous or successive secondary lesions of different tissues (e. g., Case XIV):

Palms	120 lesions	Throat	26 lesions
Soles	24 "	Mouth	26 "
Scrotum	10 "	Tongue [1]	81 "
Penis	7 "	Double iritis	1 lesion
Nails	7 "		
Other parts of skin	121 "	Total	423 lesions [2]

NATURE OF THE LATE SECONDARIES.—Almost all the skin lesions are squamous, papular, or crusted. Late macular syphilids were noted thrice in the third year, once in the fifth. Condylomata were not seen later than the third year, but mucous papules of the penis were noted once in the sixth year; pustules in the seventh, eighth (twice), eleventh, and thirtieth years (Case XIV); vesicles once in the seventh year.

The late secondary syphilids of the mucous membranes are, as Fournier state, usually of a purely erosive type. The mucous papule is rare. Three fourths of these mucous-membrane lesions (55 out of 75) were noted within the first six years of the disease.

THE INFECTIOUSNESS OF LATE SECONDARY SYPHILIDS.—How rare late infections actually are has already been noted; how often they are possible it would be hard to say. The infectiousness of secondary lesions is unmistakable; yet the impossibility of contagion from a dry skin lesion is equally certain; and it is precisely these dry skin lesions that make up almost the whole tale of late secondary syphilis. The only noteworthy exceptions are the mucous-membrane lesions. That these are a real source of danger is shown by the fact that many of the intimately studied cases of late infection have been traced to—a kiss. Thus does the lapse of time purify even venereal disease.

Among the many instances of late infection cited by Four-

[1] The tongue lesions are so casually recorded that almost half have been rejected as too indefinite for tabulation.

[2] Among 227 cases.

nier, the following, observed by Spillmann, is peculiarly instructive:

Case XVII.—Chancre in 1871, followed by secondary symptoms of skin and mouth. Treatment for four years. He marries in 1876. The first child is born and remains clean to his twenty-sixth year. Shortly after the birth of this child the father becomes an inveterate smoker, and soon his tongue shows syphilitic erosions. Warned on several occasions of the danger to which these lesions expose his wife, he nevertheless continues to smoke, and the erosions multiply. At last, in 1880, the expected happens; the wife develops chancre of the lower lip, followed by secondary lesions. Being pregnant, she aborts, and later bears two syphilitic children.

Note the striking points of this case. In the fifth year of the disease the patient marries and proves that he is not infectious by having a healthy child. Later *he becomes an inveterate smoker* and promptly the tongue relapses. But, *although the tongue is constantly sore, it is several years before he infects his wife* in the ninth year of his disease. Recrudescence of lesions from smoking and relatively slight infectiousness of these lesions—such are the morals of this case.

The Prevention of Late Secondary Lesions.—To a certain extent the recurrence of late secondary lesions is not preventable. That adequate early treatment is not an absolute preventive is shown by Case XVII, while Cases XIII and XIV show relapses in spite of numerous short courses of treatment extending over many years. Indeed, Fournier believes that early treatment may inhibit subsequent *tertiary* lesions or may merely attenuate them and permit them to appear as secondary lesions.

I cannot quite agree with this view. Adequate early treatment does not wholly inhibit late tertiaries or late secondaries. But it does inhibit both in great measure and in proportion to its intelligent application, other things being equal. My clin-

ical impression is entirely in accord with Neumann's theory that every active syphilitic lesion is potentially a focus of incubation for a subsequent outbreak, and hence it is much more important in the early stages of the disease that the symptoms be vigorously combated and promptly conquered than that the patient should follow what is, after all, but a theoretical system of mercurial treatment based on eminently fallible observation of the most elusive and contradictory of diseases.

Such treatment, the detail of which will be discussed later, is surely calculated to inhibit relapses of all sorts, secondary as well as tertiary.

But of even greater importance as regards the late infectious buccal and pharyngeal syphilids is the abuse of tobacco. The constant smoker cannot get an adequate prophylaxis from any treatment, however intense, however prolonged. The irritation of tobacco is a menace, varying very widely and often surprisingly slight; yet it is, of all the causes of late, infectious, secondary lesions, the most important as well as the most unnecessary. Tobacco is to late infectious secondary lesions what alcohol is to late tertiary lesions.

THE TERTIARY SYMPTOMS

Definition.—Those lesions of syphilis which are localized and destructive of tissue are called tertiary. They consist of relatively diffuse infiltrations terminating in the production of masses of scar tissue or of reatively localized masses (gummata) tending to central caseation. The tertiary lesions show little tendency to heal spontaneously, but spread to the surrounding tissue, advancing in a circular or circinate way and destroying every tissue encountered in truly malignant fashion.

A severe outbreak of tertiary lesions may undermine the patient's general condition, and prolonged suffering from such lesions may give rise to amyloid or sclerotic changes in the vis-

cera. But in this regard syphilis does not differ from other chronic inflammations. There is no specific tertiary toxemia.

Occurrence.—The majority of cases of syphilis as we see it to-day do not have any tertiary lesions. Among my 2,500 cases, in 1,024 (forty-one per cent) [1] tertiary lesions are recorded; of these, 106 in women.

Tertiary lesions may appear at any time after the secondary incubation. They may even precede the secondary symptoms. But this *tertiarisme d'emblée*, as the French call it, is most exceptional. (Case XXVII, page 231, in which only a few negligible secondary symptoms preceded the development of a gumma, is an excellent example.) Yet it is by no means unusual for tertiary lesions to appear during the first year of the disease—but the conditions may be best expressed in figures, and accordingly I annex two tables, Fournier's, the larger, drawn from a private practice in Paris; mine, the smaller, drawn from a similar source in New York:

INCIDENCE OF TERTIARISM

	Fournier	Keyes
First year	278	112
Second year	453	149
Third year	471	142
Fourth year	388	79
Fifth year	357	61
Sixth year	326	64
Seventh year	274	39
Eighth year	211	25
Ninth year	195	20
Tenth to fourteenth years	736	80
Fifteenth to nineteenth years	423	26
Twentieth to twenty-ninth years	304	23
After the twenty-ninth year	83	4
Total	4,499	824

[1] Certain authorities record as low as ten per cent tertiaries in cases treated by certain methods. But I am not dealing with syphilis as treated by us, but with syphilis as seen by us—with syphilis as it exists in the United States to-day.

Observe that tertiaries make their appearance more often in the second and third years of the disease than in any other two years; that even in the first year they are by no means uncommon (six per cent, Fournier; thirteen per cent, Keyes); that in half the cases they appear first within three (Keyes) to seven (Fournier) years after the chancre; that the beginning may be deferred for an almost indefinite time. Among my cases the four longest deferred began at thirty (twice), thirty-one, and forty years: Fournier records two at forty-six, and one each at fifty-two, fifty-four, and fifty-five years after the chancre. Manifestly, the tertiary "period," like the secondary "period" of the disease, begins with the chancre and ends with the patient. Yet the rule that secondary symptoms usually cease ere tertiary ones begin is clinically true and is supported by a comparision of the above statistics with those on the duration of secondary symptoms.

Relapses.—The intermittent or relapsing character of syphilis is most evident in its tertiary lesions. Relapses of secondary lesions usually occur at relatively short intervals of a few months; tertiary relapses at longer intervals.

Among 307 cases of relapsing tertiaries which I have analyzed, 115 relapsed once, 77 twice, 30 thrice, 6 four times, 5 five times, 2 six times, 1 eight times.

(The intervals between relapses may be tabulated as on page 98.)

Thus about one third of the cases of tertiarism relapsed, and about half of these relapsing cases recurred no more after two years. Yet in this matter statistics are almost meaningless, for averages do not affect individuals.

How widely the course of tertiarism fluctuates may be inferred from the contrast of Cases IV, VI, VII, and XXVII (pages 19, 231) of early tertiarism with the following four typical examples of late and protracted tertiarism:

INTERVALS BETWEEN TERTIARY RELAPSES

	First Relapse	Subsequent Relapses	Total
Less than 1 year[1]	37	13	50
1 year	99	71	170
2 years	51	42	93
3 "	41	29	70
4 "	13	11	24
5 "	15	5	20
6 "	7	4	11
7 "	10	3	13
8 "	6	4	10
9 "	5	0	5
10 "	8	5	13
11 "	4	2	6
12 "	1	..	1
13 "	3	..	3
14 "	1	2	3
15 "	1	..	1
16 "	2	..	2
17 "	..	1	1
18 "	..	..	..
25 "	1	..	1
26 "	1	..	1
28 "	1	..	1
Total number of cases	307	..	..

Case XVIII.—Tertiary lesions of larynx and bones beginning after forty years. October 21, 1886. The patient is a physician sixty-five years of age. He relates that in 1843, while a medical student, he acquired what proved to be a chancre in the usual way. The sore was burned with nitrate of silver and he took a few five-grain blue pills—not more than six, he thinks—and no other treatment whatever. For forty years he remained entirely well, and, though a practicing physician, noted upon himself nothing attributable to syphilis.

In 1883 his voice became husky, the diagnosis of tuberculosis was made, and he went to the Adirondacks. After a year's residence there his voice had improved, but he suffered from severe nasal catarrh and lost a piece of the septum. Then a large node

[1] Many apparent relapses within a year are merely recrudescence of uncured lesions.

appeared on the lower part of the right tibia, another in the upper frontal region. The latter ulcerated, but he took a little iodid of potassium and it promptly healed. A second ulcer then appeared in the frontal region below the former one. This has been open for a year. There is bare bone at the bottom about one cm. square. He was sent home, taking iodid of potassium, and has not been seen since.

Case XIX.—Malignant relapsing syphilis after twenty years. Chancre in 1855, at the age of twenty-four. Early treatment a few months, chiefly or wholly by iodid of potash. He married and had healthy children, and no further symptoms of syphilis until 1875. Then gummata of tibia and ulna, treated and cured by Dr. Van Buren (who introduced him into catheter life at the same time).

In 1879 enormous gumma of left thigh. Treated by my father. Well in April, 1880.

In August, 1880, gumma of right buttock, then some tubercular skin lesions. Cured in March, 1881.

In July, 1881, gumma of right calf; in November, gumma of skull. Cured in December, 1883.

In July, 1883, acute suppurative nephritis with edema of lungs. He resumed mixed treatment, and in January, 1884, was well so far as subjective symptoms were concerned. But the urine continued purulent, catheter life continued, and in April, 1884, he had another edema. Last seen in July, 1884. No further symptoms of syphilis. He probably died soon after.

Case XX.—Secondary symptoms relapse twenty years: tertiaries from the fourth to the twenty-second year. Chancre in 1870, at the age of thirty-three. Early symptoms light. Quality of treatment not stated. First seen in 1874 with a scaly eruption of the scrotum, a pearly patch on the side of the tongue, a little interstitial thickening of both tonsils, and thin, ribbed, eczematous-looking nails. He gave an eczematous family history, and, accordingly, was tried on arsenic and local treatment. But he began to lose weight, the lesions did not improve, and in June he was therefore put on mixed treatment. This he took quite faithfully, yet the scrotum grew worse, became pustular, and finally ulcerated in July, 1875. A sharp course of iodid healed these ulcers in two weeks.

A month later a typical chancre redux appeared, resembling a chancre in every respect. But there had been no exposure for a year, and there was no adjacent adenitis. It was followed, however, by a few papules on the hands, some squamous patches on the legs, and more pearly spots on the tongue. In January, 1876, all was well except the tongue. Nails still ridged.

Though he kept up treatment, with never more than a month intermission, new spots continued to appear on the tongue, and in January, 1877, after much facial neuralgia, a gumma developed "high up under the left temporal muscle." By March this was under control.

In July, 1877, he complained of drowsiness, diffuse pain over the side of the head, and some nervous excitability. This was followed by alternating diarrhea and constipation and slight left ptosis. Treatment resumed to November, 1878.

In January and November, 1879, and in April, 1880, he took short courses of mixed treatment for facial neuralgia and "the old congestive painful attacks in the abdomen." Yet, in November, 1880, tongue and scrotum (scales) relapsed, and a few weeks later he lost his voice from infiltration about the arytenoids and vocal cords. These symptoms were relieved by February, 1881.

In April, 1881, a relapse of facial pain, tenderness, and edema called for more treatment.

In September, 1882, several gummata appeared in the tongue, followed in January, 1883, by depapillating glossitis. This was cured by the end of 1884.

In September, 1885, tubercles on fingers. Prompt cure.

In June, 1887, interstitial glossitis and erosions on tongue. These continue to relapse until 1890 in spite of treatment.

In 1892 the intestinal attacks returned, a mass was discovered, and operation advised. Accordingly, Dr. Stimson explored, and finding a large growth obstructing the gut, made a lateral anastomosis and gave a bad prognosis. Undismayed by this, and unaided by any antisyphilitic treatment, he promptly recovered from the operation, and when he next reported, in February, 1898, the lump had disappeared, and he was well but for a little gout. And since then the syphilis has ceased to relapse. (Last seen in 1904.)

Was the intestinal tumor syphilitic? I believe it was.

Case XXI.—Interval of twenty-eight years between tertiary relapses. Chancre in 1846, at the age of twenty-nine. Took mercury constantly, until, in 1854, he lost the greater part of both hard and soft palates and was gravely debilitated. Cured in two years by heroic doses of iodids. No relapse until May, 1882. Then some papules appeared on the index finger of the right hand. These spread rapidly, and when first seen, four months later, there was a tuberculo-ulcerative syphilid extending from hand to elbow. Cured in one year by mixed treatment.

THE EFFECT OF TREATMENT.—Such cases surely prove the lawlessness of syphilis. Be it noted, however, that the efficiency of treatment is the determining factor in tertiary relapses. If a tertiary lesion remains untreated it will, sooner or later, be followed by other tertiary manifestations. If inefficiently treated, it may be temporarily controlled, but other relapses soon occur (Cases XIX, XX)—hence the frequency of relapses within three years. If efficiently treated, future relapses will probably be altogether prevented, certainly indefinitely delayed (Case XXI). Happily, our understanding of and equipment for efficient treatment have advanced greatly in the last quarter of a century.

But, if the treatment of a given tertiary outbreak must be so administered as to prevent relapses, it is manifestly even more important that the early routine treatment be so ordered as to prevent any tertiary outbreak whatever—if that be possible.

Let it be confessed at once—such prevention cannot be absolute. In Case XXVII, for example, no conceivable foresight could have forestalled the tertiary lesion, though vigorous treatment earlier applied would have checked it sooner. So, in a lesser degree, many instances of tertiarism in the first years of the disease are unpreventable.[1] But after the fifth

[1] Absolute prevention of tertiaries cannot be achieved by any treatment. The ideal minimum claimed by certain Continental authorities is ten per cent of tertiaries.

year the well-treated case may expect no further tertiary lesions.

What, then, constitutes "good treatment"? Mercury and hygiene. And each of these is twofold, thus:

1. *Mercury* (*a*) sufficient in quantity and administered long enough to control the average case; (*b*) sufficient in quantity and with the aid of iodids, tonics, or whatever else be required to control *promptly* all secondary or tertiary manifestation.

2. *Hygiene* (*a*) especially abstinence from alcohol and such moderation as may be necessary in the use of tobacco; (*b*) in general, the avoidance of such deleterious influences as disease, overexertion—especially the overstrain of so-called "modern life," be the dissipation business by day or pleasure by night—and trauma.

Discussion of these matters does not belong here. But if the rules laid down (page 139) are obeyed, the patient has nine chances in ten of escaping tertiary lesions after the fourth year of his disease. This statement I should gladly support by statistics, but that statistics are lacking. I have no close record of the manner of life followed by my patients. For example, less than one per cent of them are denominated "alcoholic"—surely an optimistic estimate.

Yet I have a general record of the duration of the early treatment in 523 non-alcoholic cases which developed tertiaries. Of these, 292 were under mercurial treatment at least eighteen months,[1] 231 for a less time. The duration of tertiaries in the former class [2] was less than five years, sixty per cent; five to ten years, twenty per cent; ten to fifteen years, four per cent; over fifteen years, six per cent; for the latter class [2] respectively thirty per cent, thirty-three per cent, twenty-one per cent, and sixteen per cent.

[1] Yet many of these were by no means efficiently treated.

[2] Exclusive of known alcoholics.

THE EFFECT OF ALCOHOL.—I have said above that tobacco is to late infectious secondaries what alcohol is to late tertiaries. Let me insist on the converse proposition: Alcohol is to late tertiaries what tobacco is to late infectious secondaries. Every syphilitic smoker does not have an eroded tongue, nor does every syphilitic drinker continue to have relapsing tertiaries. I have known several men to drink themselves to death many years after their last syphilitic lesions had disappeared. Yet close observation will show not only that the man who drinks hard is the worst sufferer from tertiary syphilis, but also that he who merely absorbs constantly—faithfully, one might say—two or three drinks a day is a far more promising candidate for tertiarism than he who drinks nothing at all.

More than this: In sensitive subjects I have several times (sufficiently often to convince both the patient and myself that this was no mere coincidence) noted distinct exacerbation of existing lesions within forty-eight hours after the patient had taken two or three glasses of beer or whisky. By keeping a sharp lookout for such occurrences, one notes them with startling frequency, and one readily convinces the patient of the evil effect of even a little drink. I grow yearly more convinced that the syphilitic who wishes to insure himself against relapses must drink neither habitually nor intemperately. An occasional drink is permissible; but it is safest, all things considered, that he drink not at all.

Statistically, my 113 recorded alcoholics stand almost on a par with the insufficiently treated cases (see above) with semidecennial percentages of thirty-four, thirty-two, nineteen, and fifteen.

REGIONS INVOLVED.—No tissue is exempt from tertiary syphilis. The same is doubtless true of secondary syphilis; but the transitory congestive or exudative secondary lesion passes and leaves no trace; while the persistent and destructive

tertiary lesion, even though it be cured, leaves a permanent scar to attest its passage.

It were, therefore, futile to enumerate in detail the lesions I have observed; another few thousand cases would show altogether different proportions. In the main, however, one may separate them into four classes in the following proportions: Lesions [1] of the skin and subcutaneous tissue, thirty-one per cent (544 cases); lesions of the nervous system, thirty-one per cent (598 cases); bone lesions (including nose and palate), seventeen per cent (302 cases); lesions of the viscera and mucous membranes, seventeen per cent (308 cases).

It is surprising to find lesions of bone so few and lesions of the nervous system so many. Yet the statistics of other observers show about the same proportions.

But in statistics of this sort, which after all chiefly concern the patient, it seems unfair to separate tertiary symptoms from late secondaries and parasyphilids; they are all one to him. I have, therefore, included all these in the following table, made up in percentages from 2,231 recorded lesions (occuring at or after the third year, and including all tertiaries, upon 1,330 cases).

INCIDENCE OF ALL LATE LESIONS

	To 5 Years	5 to 10 Years	10 to 15 Years	15 to 20 Years	Over 20 Years	Grand Average
	Per cent	Per cent	Per cent	Per cent	Per cent	Per cent
Skin, etc.....	43	38	29	31	32	37
Nervous system...	23	27	33	34	32	27
Bones.....	13	13	15	10	12	14
Other tissues	21	22	23	25	24	22
Total number of lesions.....	1,018	642	313	106	152	2,231

[1] Counting an eruption, even though it appears first in one part of the body, then in another, a single lesion.

Thus, although the number of lesions decreases by about half with each five-year period, lesions of the nervous system are proportionately little more and lesions of the skin little less frequent after the tenth year of the disease than before this, while the proportion of bone lesions remains about the same throughout. Bone syphilis owes its horror rather to its disfigurement than to its frequency.

THE PARASYPHILIDS OR SYPHILITIC DYSTROPHIES

Definition.—The parasyphilids or syphilitic dystrophies are lesions or functional derangements for which pathology shows no syphilitic basis and for which the antisyphilitic specifics afford little or no relief. They are apparently toxic in origin, and may be caused by various forms of systemic intoxication. This systemic intoxication is usually, though not necessarily, syphilis—syphilis implanted upon a soil harrowed by an evil heredity or fertilized by debility due to such causes as malnutrition, excessive mental or physical strain or debauch.

Varieties.—Thus *the parasyphilids*, or syphilitic dystrophies, *include all conditions which, without being pathologically syphilitic, are clinically due to syphilis.* Their name is legion. Case XX, showing "eczematous-looking" nails, apparently due to syphilis, and yet persisting after the truly syphilitic lesions had been cured, is an excellent example of a large and entirely indefinite class of conditions bearing no special hall-mark of syphilis, and yet arising during the active period of the disease. In the patient's mind such lesions are truly syphilitic: Thus one man tells me his skin "has never been the same"—another that the texture of his hair or nails has changed—since his syphilis. Another feels his spirit broken or his vitality gone; he never has regained the weight lost during the first months of the disease; he has been afflicted with neuralgic headaches ever since the disease began. An-

other has become hysterical or neurasthenic or syphilobic to an insane degree.

Are these functional or dystrophic conditions truly syphilitic in origin? Clinically, yes. Syphilis is undoubtedly the exciting cause. And yet in many such instances one feels assured that the constitution of the patient is much more at fault than the syphilis. The same rough skin or impaired vitality might have resulted from typhoid fever or from a severe mental shock or from prolonged malnutrition. The disorder is a functional disturbance of the nervous system evoked by syphilis, but whether evoked by terror or by toxin, it is often impossible to say.

1. Such is a large vague group which will or will not be classed as syphilitic dystrophies, according as the physician is materialist or idealist.

2. Again, there are a number of conditions manifestly not neurotic in origin, and of which syphilis, like any other chronic toxemia, may be the cause. Arteriosclerosis comes first to mind, and with it interstitial sclerosis of any viscus. Fournier would add certain cases of epilepsy, glycosuria, etc. Indeed, this class of cases is but little less broad and vague, but little more closely and essentially allied to syphilis than the preceding one.

3. In a third class, and strikingly emphasized by their frequency, their fatality, and their almost constant association with syphilis, are tabes dorsalis, paresis, and Erb's spastic spinal paralysis. These are the parasyphilids *par excellence.*

4. In a fourth class are the defects of development imparted by hereditary and to a less degree by infantile syphilis. These will be considered with the other special lesions of hereditary syphilis (page 532).

5. Finally ascending the scale and reaching lesions more peculiarly and essentially syphilitic, and yet not bearing the full pathological imprint of the disease, we may mention the pigmentary syphilid (page 300) and certain acute neurasthenias

of early syphilis. These were included among the parasyphilids in Fournier's classical monograph; but they scarcely deserve this special attribution. Nor need buccal leukoplakia (page 352) and aneurysm be considered here, for these are truly syphilitic lesions.

Tabes Dorsalis and Paresis.—The wide divergence of modern opinion upon the etiology and pathology of tabes and paresis is most disheartening. We do not yet know whether the two are clinical types of a single pathological condition. We do not know whether they are primarily inflammatory or primarily degenerative. Perusal of recent discussions (especially in relation to paresis [1]) leaves one quite bewildered and destroys all the confidence engendered by reading the clear-cut opinions of any one authority.

Seeing these diseases from a syphilitic point of view, I naturally encounter only those of manifest syphilitic origin, and, accordingly, must side with those who believe tabes to be almost always syphilitic in origin, paresis frequently so.

Etiology.—Syphilis is discoverable in the antecedents of ninety per cent to ninety-five per cent of tabetics and in about fifty per cent [2] of paretics. Asylum statistics of paretics show a relatively low proportion of syphilis because it is difficult to obtain an accurate history after the disease is far advanced. But closer study of the histories of paretics reveals a disproportionate increase of syphilitics. Juvenile paresis and tabes are exclusively syphilitic.

Most authorities agree that syphilis is not the adequate cause of either malady. "Syphilis and civilization," says Krafft-Ebing. And this is the accepted doctrine. Among Asiatics and Africans, though syphilis be common, even en-

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[1] *J. Am. Med. Ass'n.*, 1905, vol. xliv, pp. 1153, 1320, 1413; *N. Y. Med. J.*, 1905, vol. lxxxii, p. 500; *Bull. de l'acad. de méd.*, Paris, 1905, vol. lxix; *La Syphilis*, 1905, vol. iii, pp. 321, 500, 595, 652, 743, 829.

[2] "Not more than one half the total number" (Berkley).

demic, paresis and tabes are extremely rare, and apparently our ancestors could drink wine and debauch women with little fear of tabes at least. But in our own time both diseases grow more and more common. Whereas women used to suffer infrequently (1–10), some recent statistics attribute the disease to them quite commonly (1–4, or even higher). City folk are more liable to these diseases than their country brethren. The age of onset is between twenty-five and forty-five years in the great majority of cases (seventy-five per cent of my tabetics and seventy per cent of paretics began between the ages of twenty-eight and forty-five). These general facts, fortified by observation of individual cases, show that "civilization" has a large share in the etiology of tabes and paresis; but it is not possible to specify more closely than this. Lechery and liquor can scarcely be invoked as exclusively modern causes of disease, though they are undoubtedly contributory in certain cases. Heredity is, of course, blamed by some authorities and exculpated by others. The constant nerve tension of city life, whether in the struggle of business or in the pursuit of pleasure, is often the determining cause.

I have tabulated the relation with syphilis of 123 cases of tabes and 44 of paresis, thus: [1]

7[2]	tabetics	and	1	paretic	denied syphilis.
10	"	"	1	"	were doubtful.
96	"	"	38	"	confessed syphilis.
8	"	"	5	"	began before the fifth year of the syphilis.
30	"	"	13	"	began between the fifth and ninth years.
16	"	"	9	"	began between the tenth and fourteenth years.
8	"	"	0	"	began between the fifteenth and nineteenth years.
13	"	"	7	"	began after the twentieth year.
22	"	"	10	"	had suffered syphilitic lesions of the nervous system.
41	"	"	15	"	had suffered tertiary lesions.[3]

[1] See also table on p. 372.

[2] One other tabetic who denied syphilis had lost his septum and showed a palmar syphilid.

[3] Including syphilis of the nervous system.

The longest "incubation" of tabes was 32 years; of paresis, 25 years; the shortest of tabes, 3 years (6 cases); of paresis, 3 years (3 cases); the average, of tabes, 9 years; of paresis, 8 years.

Tabes occurred in about four per cent of all cases of syphilis, paresis about one third as often. My records do not show the bearing of early treatment or symptoms of syphilis upon the prospect of tabes or paresis. Fournier alleges the early symptoms are almost always light and the treatment brief.

PATHOLOGY.—In both tabes and paresis two pathological conditions commonly coexist—viz. (1) degeneration of certain tracts, and (2) an interstitial sclerosis. The essential lesion of tabes [1] is commonly believed to be degeneration of the sensory fibers, chiefly those of the cord; the interstitial hyperplasia found at autopsy (and the motor degeneration) being regarded as incidental or secondary. Yet a certain few see in the interstitial changes a primary perivascular syphilitic sclerosis. Practically the same divergence exists in the interpretation of the pathology of paresis. Those who believe in the syphilitic nature of the two diseases see in the inflammatory lesions evidence of actual syphilitic change, but the weight of authority is against this view—at least for the majority of cases. Yet the cytologic changes of the spinal fluid so common in syphilis are all but constant in tabes and paresis (page 376).

Yet a certain few typical cases of tabes (or paresis) prove, whether by the success of antisyphilitic treatment or by the post-mortem findings, to be syphilitic conditions pure and simple. They are classed as syphilitic pseudo-tabes (or pseudo-paresis). Though various symptoms have been alleged as differentiating these "pseudo" or syphilitic conditions from true tabes (or paresis), one encounters from time to time cases diagnosed as one of the latter conditions by competent neu-

[1] Marie's suggestion that tabes may be a syphilitic disease of the lymphatics, though very attractive, has not been demonstrated.

rologists which yet show marked improvement under antisyphilitic treatment.

Hence the two important points that concern the syphilologist are (1) to be always on his guard against tabes or paresis and able to diagnose them in their incipient stages, and (2) after making the diagnosis to administer a short, sharp course of antisyphilitic treatment before consigning the patient to the neurologist.

Diagnosis.—This is no place for a clinical study of the symptoms of tabes and paresis. We must take this for granted, and set down here only the essential features of the diagnosis.

The diagnosis of tabes is called for if a syphilitic complains of sharp lancinating (otherwise inexplicable) pains in any part of his body, or of inability to urinate, or of increasing constipation, or of numb feet or stumbling in the dark, or of explosive diarrhea or vomiting accompanied by severe pain, or of any "eye trouble," or of impairment of the sexual powers.

The diagnosis may usually be made from *loss of knee-jerk* and *reflex iridoplegia* (Argyll-Robertson pupil). If one of these be absent and the patient tabetic, there will be severe *fulgurating pains*, areas of *paresthesia* or *static ataxia* (inability to stand with heels and toes together and the eyes shut) to replace it. In many instances the typical ataxic gait practically establishes the diagnosis.

Tabes may be confused with pseudo-tabes, whether due to syphilis or toxic neuritis. Toxic cases usually are distinguished by an actual diminution in muscular power and tender points along the inflamed nerves, neither of which occurs in early tabes. Syphilitic myelitis should be readily distinguished by the increased reflexes (page 403).

The diagnosis of paresis is much more difficult. "Think twice before contenting yourself with a diagnosis of neurasthenia in a man or a woman who in middle life shows well-defined reflex pupillary disturbance," says Berkeley. Neuras-

thenia, indeed, may be indistinguishable from incipient paresis unless by the very closest physical examination. Disorders of the iritic reflexes are among the earliest and commonest of the physical signs of paresis. One or both pupils are myotic and do not react to light, or the consensual reflex (wavering of the pupil when light is admitted to or excluded from the *other* eye) is absent. Slight incoördination and twitching of the face and tongue also occur early, and upon these a probable or even a certain diagnosis may be had if they are associated with the familiar moral irresponsibility, the change of character that makes the paretic so dangerous to the community. The importance of examining the cerebro-spinal fluid to distinguish paresis from neurasthenia and alcoholism cannot be overestimated (page 376).

Prognosis.—Tabes may be controlled (sometimes without antisyphilitic treatment, oftener with it) for an indefinite time, even for many years. When controlled the muscular incoördination may be lessened by systematic gymnastics. The paralyzed bladder may even resume its functions spontaneously (one case) or be improved by operation (two cases). But the stigmata of lost knee-jerk, reflex iridoplegia, and static ataxia do not disappear.

Paresis, though it show brief remissions, can neither be controlled nor cured.

Treatment.—The treatment of either condition should begin with a short, sharp course of antisyphilitic medication (page 148). The older authors depended chiefly upon iodids; nowadays injections of mercury are often used without iodids. I believe both do good and should be pushed to the limit of toleration.

Successive courses at intervals of three to six weeks may be employed to push or hold an advantage already gained. But *if the first thorough trial of antisyphilitics achieves nothing, it should not be repeated.* The tonic, hygienic, educative, and

orthopedic treatments may be conducted simultaneously. They concern the neurologist, not the syphilologist.

Syphilitic Spinal Paralysis.—Erb [1] affirms that certain cases of spastic paraplegia, occurring in syphilitic patients, are due to syphilis, though the pyramidal tracts (lesions of which cause spastic paraplegia) do not show typically syphilitic lesions. Hence the condition is a parasyphilitic one. Erb has entitled it syphilitic spinal paralysis.

This condition differs clinically from other types of spastic paraplegia in only a few features. There is the same slow progress with years during which the disease does not advance (though occasionally the progress is quite rapid, the onset acute); there is the characteristic symptom complex; viz., motor weakness, chiefly in the lower extremities, spastic rigidity of the lower extremities, and exaggerated reflexes, i. e., Babinski reflex, ankle clonus, and exaggerated knee-jerk.

From this general type the syphilitic variety differs chiefly in three ways:

1. History or signs of previous syphilis.

2. Partial or complete bladder paralysis (which is not seen in the non-syphilitic cases).

3. Slight, early paresthesia. The areas of disturbed sensation may be discovered only after careful examination.

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[1] *Deutsche Zeitschr. f. Nervenheilk.*, 1902–3, vol. xxiii, p. 347.

CHAPTER VIII

DIAGNOSIS

THE diagnostic problem of syphilis is twofold: viz., Has the patient syphilis? Is the lesion syphilitic?

To the victim the two questions are one. If he knows he has syphilis every pimple and pain looks syphilitic to him. But the practitioner must be more judicious. If the patient seem syphilitic, but the lesion not so, let him be slow to infer a connection between the two. If the lesion seem syphilitic and the patient not so, let him be even slower.

A few examples will elucidate the necessity for this distinction.

Case XXII.—A patient, in the thirteenth year of his syphilis, having had no symptom of the disease for several years, comes with a small, scabbed ulcer under the right eyebrow. It is superficial and nondescript, and is accordingly watched for two weeks. During this time it increases slowly in size and becomes a characteristic little syphilitic ulcer. It yields promptly to mixed treatment.

Nine months later he returns with a similar spot near the angle of his mouth. He insists on antisyphilitic treatment, and it is accordingly administered, but the sore, instead of healing, remains nondescript for a week or so. Then he disappears for a month, and returns (still vainly under treatment) with a typical warty epithelioma. It is scraped, cauterized, and cured.

Eighteen months elapse, and he returns with what looks like a suppurating hair follicle on the nasal septum just where it joins the lip. Under local applications this improves for a time, then suddenly begins to spread and destroy the surrounding tissue. This is checked in a few days and healed in a month by injec-

tions, inunctions, and iodid of potash. The septum is left slightly deflected.

This very striking example of the caution requisite in diagnosis, even when the patient is known to be syphilitic, requires no further comment. Case III (page 19) is a sad instance of neglect in this regard.

The opposite error, a mistaken diagnosis of syphilis founded on a single lesion which appears to be syphilitic, is wofully common. Every syphilologist sees hundreds of patients who have been condemned to years of treatment and a lifetime of terror by the rash diagnosis of syphilis from the appearance of a genital sore (see page 243). Another common error is to treat headache or neuralgia by iodid of potash, and then, noting some fancied or fleeting amelioration, to infer the existence of syphilis.

Nothing could be more unwarranted; nothing more destructive. For if there is one error worse than to assure a syphilitic he is clean, it is to assure a clean man he is syphilitic.

SYPHILOPHOBIA

The fear of syphilis is much more acute in those who only think they have the disease than in those who have its actual manifestations to check the riot of imagination. The horrors of syphilis pale into insignificance compared with those of syphilophobia, as this condition is called. It assumes every variety of hysteria and neurasthenia. I have known it to drive a man to suicide.

Case XXIII.—I have known a woman, brutally ill-treated and cast off by her husband, to fancy she had syphilis and wander from doctor to doctor, until one ill-advised man, rather in the hope of relieving her mental condition than impressed by her symptoms, told her she had syphilis and gave her mercury. She hailed him as a deliverer, wept upon his shoulder, tortured him

for a year with every conceivable imaginary symptom, and finally came in triumph leading her beautiful little child, in whom she declared she had observed symptoms of the fell disease.

Case XXIV.—I have known a man to marry and to consult a physician the following day on account of a sore which had just appeared upon the foreskin. The doctor, overlooking the obvious cause, pronounced it chancre. The man never saw his wife again, obtained a divorce, and spent the rest of his days bringing out eruptions with iodid of potash and "driving them in" with mercury. When he came under my treatment, some ten years later, it was impossible to separate him from his drugs.

MEANS OF DIAGNOSIS

To avoid such horrors with certainty we should have an infallible laboratory reaction for syphilis. But we have it not. We depend mainly—in most instances entirely—on clinical evidence.

The means of diagnosing syphilis may be classified as follows:

1. Laboratory Diagnosis.—Examination for spirocheta.
   Inoculation of monkeys.
   Specific reaction.
   Examination of the blood.
   Examination of syphilitic tissue (biopsy).
2. Clinical Diagnosis.—History.
   Scars of previous lesions.
   Appearance of the present lesion.
   Effect of treatment.

Examination for Spirocheta.—The opinion of a *real* expert is valuable confirmatory testimony. But even the most expert may fail to find the spirocheta when present, and may think he sees it when absent. Hence such evidence is only

confirmatory. I find it most useful in the differential diagnosis of chancre and of buccal sores (pages 243, 249).

Inoculation of Monkeys.—This can never be generally employed for lack of subjects. It appears to be a reliable test.

Specific Reaction.—The only specific reaction at present available is the *Justus test*, the Wasserman serum not being reliable (page 34).

Dr. Justus asserted (in 1895) that when a syphilitic, uncured, but not under treatment, is given a stiff dose of mercury, preferably by injection or by inunction, his hemoglobin percentage falls from ten to twenty points in from three hours to one day. This change he considered pathognomonic of syphilis.

The question has been well thrashed out [1] with the result that this test is found to be fairly reliable only in the early and more active stages of syphilis. But as it sometimes gives positive results in non-syphilitics, and negative findings even in actively syphilitic cases, it is of no clinical importance. The recognized blood changes of syphilis have no diagnostic significance.

Examination of Syphilitic Tissue.—The pathological changes described on pages 46, 327, should be a help in diagnosis. A snipping from the lesion may help differentiate gumma from neoplasm or tuberculosis. Yet in practice this is rarely employed, perhaps because the actual appearance of a specimen thus obtained is often quite nondescript. The pathologic characteristics of syphiloma are ill-defined and of a negative type, and it is often difficult to get a deep enough section of the growth to show its structure.

In practice I have known this method to fail signally in two instances. In one it led to the excision of a chancre of the lip; in the other to repeated curettage and cauterization of a

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[1] Cf. Feuerstein, *Arch. f. Derm. u. Syph.*, 1903, vol. lxvi, p. 363.

gummatous ulcer of the nose. Both were mistaken for epithelioma. In the diagnosis of an obscure lingual or laryngeal growth, however, I have never known biopsy to mislead.

Clinical Diagnosis.—Whereas the application of the laboratory tests is as yet limited and bound to conform with the clinical findings, these latter are almost universally applicable. We are therefore much better acquainted with the limitations of our clinical tests, and in the present state of our art they are the more reliable.

Yet there is a sharp distinction between the method of applying these two classes of tests. The laboratory tests are applied singly; the success of one of them is almost as convincing as though they all coincided. But the clinical tests must be applied all together. History and scars give us a hint of what *may be* the trouble; this impression is *strengthened or weakened* by the appearance of the lesion, and *confirmed or refuted* by the effect of treatment. When all four clinical tests agree we are certain beyond the certainty conferred by any laboratory report. But if they fail to agree we may remain in doubt, and the lesion may heal and leave us there. For recovery under antisyphilitic treatment is no proof that the lesion was syphilitic unless it was typically syphilitic in appearance or unless there remains a typical syphilitic scar (see Case XIV, page 89).

Thus no one test is absolutely convincing. We must apply them all. In every instance we must endeavor to prove the patient syphilitic and the lesion (the effect of treatment) must confirm our opinion.

The History.—That the patient has had a sore on his penis and has taken mercury is no proof that he has had syphilis; that Dr. X. is convinced should carry little weight; that the patient is convinced carries no weight whatever. The penile sore may have been herpes, the skin lesions acne, the mouth lesions aphthous, the pains neurotic.

Yet the *ensemble* of a syphilitic history is often quite convincing. If the sore was a month coming out and a short while healing,[1] if the groin nodes were not gravely involved, if the skin lesions were slight and the mouth symptoms severe, there will be no scars, yet there is a strong presumption of syphilis.

On the other hand, if the patient speaks of great inguinal adenitis, raves about his diurnal headache, bewails his senile baldness, and triumphantly exhibits a glistening or congested throat, depressing his tongue with professional ability, fear him. He is a true syphilophobe.

THE SCARS.—To confirm the impression derived from the patient's history we look for scars. The whole body should be inspected, and the patient may indicate some special region; but, generally speaking, evidence of past syphilis will be found in the glands, the legs, the bones, the mouth, and the site of chancre.

Enlarged nodes are the exception rather than the rule in syphilis. Within two weeks after the appearance of chancre the typical inguinal adenitis appears, and lasts some three to six months. General syphilitic adenitis (page 260) usually follows close upon the first general eruption, and disappears well within the year. Hence *syphilitic adenitis is extremely rare after the first year.*

The *skin of the legs* is the usual site of destructive skin lesion, and here we may look to find thin, round, depressed (pigmented) scars (page 274) in most severe or neglected cases.

The bones, especially the tibiæ, should be carefully palpated. The exostoses (page 431) left by unsuspected periostitis may thus be discovered.

The *mouth* sometimes bears typical deformity of the pal-

[1] The patient considers the chancre healed when it ceases to be eroded. The lump persisting many weeks thereafter does not concern him.

ate or fauces, though the secondary lesions of these regions, however severe, vanish and leave no trace. But the tongue should be inspected most carefully for erosions or pearl spots. Dr. N. B. Potter [1] finds strong confirmatory evidence of syphilis in destruction of the large papillæ at the base of the tongue (Virchow's *atrophia levis*). These are palpable rather than visible. On passing the finger over the back of the tongue one feels it to be glazed and smooth instead of lumpy, as is the normal tongue. This destruction of the large papillæ by syphilis is common, though by no means constant. It may be of assistance in diagnosing syphilis, but is not an infallible sign.

The *scar of the chancre* may be absent and is often insignificant. A thick round scar on the skin of the penis is suggestive of chancre; but if the scars are multiple or if there are scars in the groin the lesion was probably chancroid. Scars within the prepuce, even if single, usually mean chancroid.

The Active Lesion.—The diagnosis of the various lesions of syphilis will interest us as we take them up in turn.

The Effect of Treatment.—Having decided that the lesion is syphilitic we turn to treatment both for cure and for confirmation of the diagnosis. Indeed, one all too often finds it impossible to make an accurate diagnosis without the test of treatment (page 205). The history may be vague, scars lacking, the lesion itself atypical, and biopsy impracticable. We then administer iodids and mercury, and judge from the result whether the lesion be syphilitic.

No patient should be put to this test of treatment without due deliberation as to its effects upon mind as well as body. If the lesion itself is benign, the syphilis ancient, and the patient neurotic, it is wiser to wait a bit and try such other methods of cure as may suggest themselves, until the failure of these strengthens the probability of syphilis. For unless

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[1] *Boston Med. and Surg. J.*, 1906, March 8.

there is a moral certainty of syphilis, or unless the lesion is destructive or threatening, the shock to the patient's nervous system from the suggestion that his syphilis has relapsed may not be incurred lightly. He must be protected from his own fears as far as possible.

DIAGNOSIS OF DIFFERENT PERIODS OF THE DISEASE

We may summarize here certain diagnostic situations that constantly come up.

The Initial Stage.—To diagnose syphilis by the appearance of a chancre one must find (page 242)

1. Typical chancre,
2. Typical regional adenitis,
3. Negative autoinoculation, and
4. *Spirocheta pallida.*

The Secondary Outbreak.—Various combinations of circumstances lead to a diagnosis at the time of the secondary outbreak. The most common combinations are the following:

1. General macular or papular eruption with the scar of chancre and persistent regional adenitis.

2. Scattered syphilitic papules with the chancre and lymphatic nodes.

3. Chancre and lymphatic nodes undiscoverable; general macular or papular eruption combined either with scabs in the scalp or mucous papules in the mouth, or symptoms of general toxemia (notably nocturnal pains).

4. Mouth lesions combined either with scabs in the scalp or characteristic general adenopathy.

5. Preëruptive fever with toxic symptoms, scar of chancre, and persistent nodes.

In a Period of Calm.—Within the first six months typical, posterior cervical, and epitrochlear adenopathy are usually found. If both sets of nodes are enlarged and the patient's

history is suggestive of syphilis, it is proper to confirm the diagnosis, since the chances are that the patient has the disease, and it is, therefore, safer for him to continue treatment than to stop it.

After the first six months there is often no trace whatever of the disease. In this event we must depend entirely upon the history, and, even though it be not very impressive, it is usually wiser to continue the treatment on the diagnosis previously given; for there is no means of proving that the patient has not syphilis, and the omission of treatment may bring him grave relapses in the future.

Later Relapses.—The diagnosis rests chiefly upon the characteristics of the lesion and its reaction to appropriate treatment. But, as already stated, the patient's history should be searched and his body carefully inspected.

Tertiary lesions of brain, bone, and viscera quite often occur in patients who give no history of previous syphilis and show no scars. In such cases we must, therefore, go by the physical characteristics of the lesion. Yet the treatment instituted must always be regarded as tentative until the resolution of the lesion proves that it is syphilitic. The dangers of overtreatment, especially in obscure lesions of the nervous system which may or may not be syphilitic, must always be borne in mind (page 148).

DIFFERENTIAL DIAGNOSIS

Syphilis simulates almost every disease. Any one of its graver lesions may be confused with cancer or tuberculosis. The early secondary fever, as well as the less easily recognized late tertiary fever (page 269), simulates malaria, typhoid, tuberculosis, and sepsis. Genital chancre may be almost indistinguishable from chancroid, buccal chancres from Vincent's angina. Cerebral and cerebro-spinal syphilis reproduce the clinical picture of epilepsy, apoplexy, brain tumor, paresis, and

tabes. Skin syphilis counterfeits eczema, psoriasis, and lichen planus. But syphilis as a whole simulates only one disease: viz., yaws.

Yaws (*frambœsia tropica*) is so closely allied to syphilis that Hutchinson has alleged them to be identical.

The striking points of similarity in the two diseases are the following: The most obvious lesions of each are in the skin; these skin lesions are circinate in character and are frequently clustered about the mucous orifices; the pains caused by each disease are much more marked at night; the cure in each is effected mainly by mercury and iodid of potash. A spirocheta as nearly as possible identical with *S. pallida* has been identified by Castellani [1] and Wellman in the lesions of yaws.

But the differences between the two diseases are no less striking. "Yaws is a chronic, highly contagious disease, confined to certain tropical countries, and characterized by a peculiar cutaneous eruption which goes through the stages of squamæ, papules, and tubercles"—these tubercles split, and a granulating mass pushes through them—"it is accompanied by a variable (but generally slight) amount of constitutional disturbance and tends to recovery" (Scott).

The contrasting characteristics of yaws and syphilis may be tabulated as follows: [2]

SYPHILIS	YAWS
Pandemic	*Confined to the tropics*
Transmissible by heredity.	Never inherited.
Primary sore constant and pathognomonic.	Primary sore inconstant and no different from other skin lesions.
Reinfection extremely rare.	Re-infection not uncommon.

[1] *Brit. M. J.*, 1905, vol. ii, p. 1330; *J. of Trop. M.*, 1906, vol. ix, p. 1.

[2] This table is founded on that of Jaenseline, *Brit. M. J.*, 1905, vol. ii, p. 1276.

SYPHILIS	YAWS
Pandemic	*Confined to the tropics*
Autoinoculation impossible.	Autoinoculation possible for a long time.
Eruptions polymorphic.	Only one type of eruption.
Tertiary syphilid leaves typical scars.	No scars unless irritated.
Lesions primary, secondary, and tertiary.	No such distinction.
Involves the mucous membranes and viscera.	Affects only the skin.
No itching.	Itches.
Loss of hair.	No loss of hair.
Confers no immunity against yaws.	Confers no immunity against syphilis.

The fact that the patient suffering from either disease may be infected with the other is the best evidence that they are not related.

CHAPTER IX

PROGNOSIS

THE general prognosis of syphilis is a gloomy prospect compounded of various follies amid which madness for drink and carelessness in treatment stand preëminent. All the cases cited must be read with this in mind; not many of them were really well treated, not many abstained from drink.

One is fully justified in assuring the recently infected victim that *if he obey orders* he need expect no *great* trouble after the first year or two of his disease, and will even bear the outward semblance of cleanliness during that time. Syphilis is a series of surprises, to be sure, but the cases which I have personally supervised from beginning to end have been singularly free from grave relapses,[1] a freedom which I believe they owe to the system of treatment followed, and constant insistence upon the fact that relapses can neither be foreseen nor absolutely prevented by any system of treatment. If the patient is made to recognize that he cannot be guaranteed against relapse in his own person (however sure he may be that he is no longer infectious) he is—sanely—on his guard and prepared to report at the outbreak of symptoms when they may be promptly controlled.

In short, a case of syphilis intelligently treated and properly conducted usually terminates with the cessation of treat-

[1] But I have not been able as yet to follow a number of them over a sufficient length of time to give results of statistical value.

ment at the end of three years in all things except the matrimonial guarantee which is habitually to be reserved for two years longer.

But let us consider the elements of the prognosis *seriatim*; they are:

1. The patient's constitution.
2. His environment.
3. His habits, chiefly as regards alcohol and tobacco.
4. His treatment.
5. The lapse of time.

THE PATIENT'S CONSTITUTION

We can never assure a patient that his syphilis is going to be light. From the duration of primary or of secondary incubation no inference may be drawn as to conditions six months hence. And all similar signs are worthless and meaningless. Yet, retrospectively, one can see that such a patient has had little trouble with his disease, has perhaps never really suffered from it at all; while such another has labored and slaved for months and years to keep himself clean. Why this should be we no more know than why one man dies of pneumonia and another does not.

This partial, congenital immunity may even be so great as to lead to a spontaneous cure after the appearance of the chancre. Incredible as this may seem—and I have not seen it myself—it is attested by excellent authority. Yet the fact may be other than the seeming. The patient who without treatment (or with treatment for that matter) skips the early secondary symptoms is by no means quit of his disease. He is just as syphilitic, just as much a candidate for prolonged, systematic treatment as his most intensely infected neighbor —witness the following case, the only male in which I *know* the early secondaries were skipped:

Case XXV.—No symptoms or treatment for six years; then naso-pharyngeal ulcerations. The patient, a thin, rather small, and not very rugged man, aged twenty-one, showed on January 30, 1880, a healing sore upon the penis, which had appeared on January 15th, and was attributed to coitus on December 18, 1879. The sore had been cauterized, but when first seen it was no longer ulcerated, though very hard and sharp at the edge—a typical chancre. The glands in both groins were large, not painful, not very characteristic. Judgment was withheld and the patient closely watched; but the induration disappeared, and no further symptom developed. No mercury or iodids were given. During 1881 and 1882 he was treated for gonorrhea, but still showed no sign of syphilis. He was not seen again until April 15, 1886. "Patient has been perfectly well. He never had any symptoms of syphilis. He never had another sore. He never took a particle of mercury or iodin. Yet now he has a characteristic thickening of the fauces, [and] a sloughy, pultaceous ulcer, distinctly syphilitic. His throat has troubled him for five weeks. He has lost five pounds, and Dr. Bosworth diagnosticates syphilis from the throat." There is also an ulcer on the floor of the nose.

Under mixed treatment the throat promptly healed; he lost a small piece of the vomer, and in August he was clean. Treatment was continued until October 22, 1888, no further symptoms appearing. He has not been seen since.

The case points its own moral: put no faith in aborted syphilis, however mild, however readily relieved; give it the benefit of a full course of treatment. Willingness to take treatment is no less devoutly to be desired than a relative immunity from symptoms. Indeed, morally speaking, the patient with grave or persistent early symptoms has the best prospect of an early cure, for his physical woes demonstrate the gravity of the disease and the necessity of persistent treatment far better than the most impressive lecture. And though relative, congenital immunity is surely the syphilitic's most valuable asset, he can never be sure of its possession, while, practically speaking, good habits and docility to treatment con-

fer a far more tangible assurance of future health than anything else we know of.

Interaction with Other Diseases and Diatheses.—But the effect of the constitution of the patient upon the course of the disease is by no means limited to the bald moderation or intensification of symptoms. Syphilis brings out every latent weakness. It encourages the development of tuberculosis. It plunges the hysterical and the neurasthene into new depths of despair. It feeds every organic neuropathic predisposition. On the other hand, the syphilis itself is colored by every tinge of heredity—diathesis and temperament alike. Persons of gouty tendency run to scaly and tuberculoid eruptions, and are said to be especially liable to syphilis of the nervous system (though I cannot fully substantiate this last assertion). Eczema becomes almost inextricably intermingled with eczematoid syphilids (Lichen planus, psoriasis, and similar skin lesions, though they may be closely simulated by syphilis, can scarcely be said to influence the course of the disease). The anemic or "scrofulous" victim suffers intensely from early toxemia, florid, general early skin lesions, and mucous-membrane lesions of a most persistent relapsing character. The neuropath is stricken in his point of least resistance. In short, syphilis assumes the habit of its victim. Its course depicts his weakness.

THE PATIENT'S ENVIRONMENT

Syphilis, like character, is founded upon heredity modified by environment. Every influence to which the patient is subjected may react upon his disease. It were vain to try to enumerate every shade of environment, but a few of the more striking facts may be noted.

Climate.—The tropics have always borne the evil reputation of producing the vilest forms of venereal disease. Yet,

as already noted, the source of syphilis has no influence upon its prognosis. If syphilis is most virulent in the tropics this is doubtless due to the debilitating effects of climate, or malaria, and to the carelessness or indolence of the sufferer.

Age.—The age at which syphilis is contracted seems to have some effect upon its course. The overwhelming character of hereditary or infantile syphilis is too well known to require demonstration. Yet after puberty age is not a very strong factor. To be sure, the average patient infected after thirty seems to develop tertiary lesions rather more quickly than does he who is infected before that age; but he is through with them sooner, too. And the assertion so constantly made, and supported by such general acceptance, that syphilis acquired in old age is most virulent, depends, in some instances at least, upon the fact that the old man who acquires syphilis is likely to be far more completely saturated with alcohol and debilitated by riotous living than the younger victim. From my cases I am certainly not warranted in inferring any essential evil effect of age.

Debility and Trauma.—Mechanical trauma occasionally induces a relapse of tertiary symptoms (page 38), but striking instances of this are not common. On the other hand, trauma in the widest sense is one of the commonest of all causes of tertiary relapse. I have now under treatment a gumma of the tibia due to a bruise and a syphilitic hemiplegia occurring in a public accountant at a time when he was very run down by overwork. The hemiplegia is doubtless quite as directly referable to intellectual strain as is the gumma to contusion. Indeed, close investigation will reveal some such exciting cause for a surprisingly large proportion of relapses. Any disease, excess, or other debilitating influence weakens the patient's powers of resistance, and so indirectly permits the relighting of a smoldering syphilis much more frequently, though not quite so obviously, as a blow may excite a gumma.

THE PATIENT'S HABITS

Since the patient's native immunity to syphilis is his most important asset, to fortify that immunity should be his constant effort. During the first years of the disease he should keep "in training." Regular hours, simple food, plenty of sleep, fresh air, and exercise should be his rule of life in so far as his position permits. A regard for the elementary rules of hygiene is always helpful, and is in some cases of syphilis as important as in tuberculosis. Unfortunately, the majority of syphilitics may grossly neglect the rules of hygiene, and yet come to no immediate, manifest harm. Consequently they treat lightly the warning that bids them look to a more or less distant and problematical future; they neglect to make every effort to stamp out the disease while it is yet young, and in later years they return groaning under afflictions that were quite preventable.

Alcohol.—The reaction of alcohol upon syphilis epitomizes the effects of bad environment and evil habits. Alcohol is the commonest as well as the most active enemy of the syphilitic. Malignant early syphilis is most frequent among those who have been alcoholic, malignant late syphilis among those who remain alcoholic. These two are the current causes of syphilis of the nervous system, and the various visceral and vascular fibroses.

In debating the effect of alcoholism in syphilis a strict definition of the term is essential. By alcoholism is meant chronic intoxication by alcohol. This does not imply drunkenness. A man may die of alcoholism without ever being drunk. Indeed, the man who boasts that he cannot get drunk has the opportunity of becoming far more alcoholic than he who is laid low by a stein of beer; and it is precisely among these hardy and incessant drinkers that the most pronounced evil effects may be looked for. Persistent drinking may make it impossible to prevent or to cure tertiary relapses. I have

recently witnessed the most striking evidence of this in a young man with a tuberculo-ulcerative infiltration of the lips. The lesions lasted, now better, now worse, for almost two years, and almost every exacerbation could be traced to a few, a very few, drinks. Close questioning reveals similar instances quite frequently.

But the essential evil of alcoholism is not that it so often makes the early lesions of syphilis virulent and hard to cure, but that it, combined with inefficient treatment (and the two go hand in hand), is almost the whole cause of late syphilis. I have already cited my personal statistics in this regard, and my clinical impression is even stronger—much stronger—than my statistics.

On the other hand, even in its relation to alcohol, syphilis plays strange freaks. One of my patients died of drink in the thirteenth year of his disease, having had no symptom since the early secondary outbreak; another died drunk in the twenty-fourth year of his disease and twelve years after his last symptom (ulcer, scaly palms); still another took treatment faithfully, yet drank equally faithfully. He had a very light roseola and absolutely nothing else. Yet after four years he developed various ocular paralyses, requiring the most vigorous treatment for their relief. Thus the alcoholic *may* run any conceivable course; yet statistics show that he is not a good risk if he drinks constantly—even a little (page 103).

Tobacco.—The ill effect of tobacco is immediate, local, and obvious. If it excites mouth lesions it must be stopped, but otherwise it may be used in moderation. Excessive smoking must always be prohibited, for the more a man smokes the more liable he is to mouth lesions; and since it is precisely the excessive smoker who cannot be persuaded under any circumstances to give up his habit altogether, he is the man of all others who should avoid getting into a condition where he ought to stop. I try to keep all my patients down

to two or three cigars a day; but alas! I often fail, for it is harder to stop smoking than to stop drinking. Yet only the other day I saw a man who said he had smoked from six to ten cigars a day for twelve years following his chancre, and the disease had never relapsed in the mouth or anywhere else; to prohibit such a man from smoking would be ridiculous.

THE EFFECT OF TREATMENT

What is the effect of treatment? In other words, Does treatment cure syphilis? There are good reasons for doubting it. No treatment gives an absolute guarantee against relapse. Exceptionally, a case does badly in spite of the most energetic and intelligent treatment. Impressed by these isolated and exceptional facts, the pessimist cries "Syphilis can never be cured!" As well say malaria can never be cured. It, too, may relapse after years of apparent health. It, too, may prove malignant and intractable. Yet what could be more essentially curable than the ordinary attack of chills and fever? The same rule holds good for syphilis. The ordinary case is perfectly amenable to and curable by treatment. The exceptional case is refractory.

But the distribution of yellow tablets does not constitute the treatment of syphilis. The art of curing syphilis is like the art of killing salmon. The skill of the fisherman and the wiles of the fish are quite as important elements in the game as the strength of the tackle. Some fish are child's play; others tax the most experienced wrist. And the joy—but that is another story.

THE LAPSE OF TIME

Time and treatment are the enemies of syphilis. Age certainly does wither her though time cannot be said to stale her infinite variety. If the disease relapses appropriate treatment

diminishes by fully eighty per cent the prospect of further relapse, though under this, as under any other geometric progression, an infinite number of relapses is possible.

And, be it noted, syphilis does not necessarily shorten life. Because a man has syphilis his days are not lessened. Scarred he may be; but unless he be careless in his habits or unless he be the one marked for destruction in spite of all that man can do to aid him, his life will be spared.

Destructive visceral syphilis, amyloid disease from prolonged toxemia, the riddling of bone and brain and skin with a thousand lesions—these horrors of the ancients are all but inexcusable at the present day. Cerebro-spinal syphilis, paresis, and aneurysm still claim their victims, and so does tabes, though so brilliant have been the results which I have seen from vigorous antisyphilitic treatment in incipient (syphilitic) tabes that I must confess a great optimism in regard to that malady.

My own statistics give no clew to the active death-rate of syphilis, but the insurance statistics quoted below place that as accurately as possible; though I confess they impress me as unduly pessimistic.

I have studied 2,300 cases in regard to the average duration of the disease without reference to sex, age, treatment, or habits. The findings are expressed in the following table:

DURATION OF 2,300 CASES

3 years or less	1,511 cases
4 "	102 "
5 "	83 "
6 to 10 years	298 "
11 " 15 "	162 "
16 " 20 "	60 "
21 " 25 "	43 "
26 " 30 "	30 "
31 " 35 "	5 "
36 " 40 "	5 "
43 years	1 case

Thus 3 out of 4 cases of syphilis, as we encounter it clinically, end within five years; 7 out of 8 within ten years; while only 1 in 30 lasts over twenty years.

That these inferences are fairly accurate is proven, I believe, by the following table, showing the duration of the disease in cases watched five, ten, and fifteen years since the disappearance of the last symptom. The numbers are far fewer, but the proportions remain the same:

Watched	5 Years	10 Years	15 Years
Duration 5 years or less	155	75	36
" 6 to 10 years	30	13	5
" 11 " 15 "	18	10	4
" 16 " 20 "	8	2	2
" 21 " 22 "	2	1	1
Total number of cases	213	101	48

LIFE-INSURANCE ESTIMATES[1]

In recent years the influence of syphilis on mortality from a life-insurance standpoint has been discussed frequently by the medical directors of life-insurance companies, and at a recent International Congress of Life Insurance Medical Directors, at Berlin, as many as five original papers on the subject were presented. One of these, by Dr. Blaschko, is replete with statistics as to the prevalence of syphilis in some of the European companies, comparing urban and rural districts, classes of the population, occupations, etc., etc., and illustrating its influence as a cause for morbidity as well as mortality. But Dr. Blaschko also shows how greatly these statistics vary—how untrustworthy and useless they are as a guide to determine the assurability of this class of risks. The reason for

[1] Communicated by Dr. W. M. Bross.

this is that the main effort of these statistics is directed to broaden our scientific knowledge of syphilis in general, to determine the importance of syphilis as a cause of illness and death, while for the life-insurance medical directors it is of main importance to determine the influence of syphilis on the duration of life. In view of the deficiency of these statistics from the insurance man's standpoint, Dr. Blaschko was greatly pleased to be able to refer to the report of Dr. Tiselius, presented at the Sixth Scandinavian Life Insurance Congress, "an excellent report," as Dr. Blaschko puts it, and "of great importance, because it is the first statistical work on this subject compiled on a basis which is entirely flawless." This report, which contains the experience of Scandinavian companies with policy holders who had had syphilis, gives, among other information, that which is most essential for our work —the number of expected deaths on the basis of standard mortality tables and the number of actual deaths in the class. The Scandinavian companies had an actual loss of 128.5 deaths for every 100 deaths expected by the 17 English offices tables.

Dr. Blaschko did not refer to the experience of the Actuarial Society of America. They found that a history of syphilis gave a mortality of one hundred and thirty-three per cent, a result which coincides closely with the experience of the Scandinavian companies. These two reports, the one of the Actuarial Society and the other by the Scandinavian companies, contained the only trustworthy information as to the influence of syphilis on longevity which we had until very recently, when the Gotha Insurance Company published its own experience with syphilitic risks. This report is contained in the paper read by Dr. Gollmer at the Berlin Congress. It contains not only a statement of the Gotha's expected and actual losses, but also highly interesting tables, showing the influence of syphilis on causes of death. I quote

here the most important results of this investigation, and also those of the American and Scandinavian companies:

Scandinavian Companies		American Companies		Gotha	
Ages of Observation	Mortality	Ages of Observation	Mortality	Ages of Observation	Mortality
	Per cent		Per cent		Per cent
20–30.....	102.8	15–28....	105	15–35....	138
30–40.....	106.7	29–42....	134	36–50....	186
40–50.....	143.3	43–56....	153	51–70....	161
50–60.....	141.3	57–70....	102	71–90....	140
60–70.....	132.4				
70–80.....	100.0				
20–80.....	128.5	15–70....	133	15–90....	168

These three experiences deal with material of considerable size. The Scandinavian experience comprises 848 deaths; the American, 397 deaths; the Gotha, 487 deaths. They show uniformly a considerable excess mortality, the highest occurring between the ages of forty and fifty. The American and the Scandinavian companies give results that are very nearly alike; the Gotha's experience is decidedly the most unfavorable. The experience of the American companies, and also of the Gotha, tend to show that the excessive death-rate persists through the entire life, independent of the time elapsed since infection. To some extent this fact may be deduced from the above table, but it becomes more clearly apparent from the following:

	Insurance Years 1 to 5	Insurance Years 6 to 30	All Years
	Per cent	Per cent	Per cent
American companies' mortality.....	121.4	142.3	133.3
Gotha.....	143.5	172.4	168.6

Even as long as thirty years after infection the mortality continued to be fifty per cent in excess of the normal.

GOTHA MORTALITY BY YEARS ELAPSED SINCE INFECTION

	Entrants	Expected Deaths	Actual Deaths	Per Cent
5– 9 years	550	3.74	6	....
10–19 "	5,899	52.93	106	200
20–29 "	5,915	87.92	140	159
30 years and over	3,134	112.74	169	150

The Gotha also gives us a highly interesting table of causes of death among those who were infected with syphilis, and compares the deaths occurring in this class with those of all its policy holders who insured their lives between the years 1852 and 1895:

Causes of Death[1]	Expected Deaths	Actual Deaths Syphilitic Lives	Mortality Per Cent
Infectious diseases	20.00	22	110
Tuberculosis	51.72	25	48
Cancers	29.32	47	160
Diseases of the metabolic process	8.61	8	....
Alcoholism	8.98	22	245
Progressive paralysis of the brain	12.53	63	503
Disease of the spinal marrow	3.45	23	667
Disease of the respiratory organs	27.23	27	99
Cerebral apoplexy, aneurysm	70.24	152	216
Cerebral apoplexy alone	19.30	44	228
Aneurism alone	1.91	13	....
Disease of the kidneys	12.77	21	164
Disease of stomach and intestines	11.45	21	184
Disease of the liver	8.68	9	....
Accidents	7.39	11	....
Suicides	9.90	22	222
Old age	1.10	3	....
Other diseases	6.67	11	....
All causes	290.04	487	168

[1] The list contains several duplicates so that the totals do not sum up accurately.

Causes of Death	Expected Deaths	Actual Deaths	Mortality Per Cent
Mental diseases, Paralysis of the brain, Other brain diseases, Spinal diseases, Alcoholism, Suicide,	34.86	180	373
Circulatory diseases, inclusive of cerebral apoplexy, and kidney diseases	83.01	173	208
All other causes	172.17	184	107
All causes	290.04	487	168

My personal experience consists of 1,658 cases, and an analysis among these shows a syphilitic mortality of one hundred and forty-five per cent. The causes of death run so close to the above table that I think it unnecessary to mention them in detail. Among the number who have been rejected for life insurance because of syphilis, we found the mortality to have been 166.2 per cent.[1]

From the few facts given above, I think the conclusion is justified that syphilitics may be insured at any time after the symptoms have ceased to show themselves, but not at standard rates. Syphilis no doubt shortens the expectancy of life, and there is a largely increased mortality among the class during life. Much could be learned regarding this subject if the life-insurance companies of America would coöperate for a joint investigation conducted on a uniform basis.

Regarding the treatment, it can be safely assumed that those who are well enough off to assure their lives, are, in the vast majority of cases, treated to the best of the ability of the medical man employed. The indications are, however, that a syphilitic should always remain under the observation of a competent medical man.

[1] This percentage doubtless represents an exceptionally large number of neglected syphilitics (Keyes, Jr.).

CHAPTER X

PRINCIPLES OF THE TREATMENT OF SYPHILIS

PREVENTION

Prophylaxis.—The prophylaxis of syphilis has been discussed in Chapter I.

Abortive Treatment.—To abort syphilis is impossible. Syphilis may abort itself; but even though no symptoms follow a true chancre we have no means of judging whether the disease is ended or whether we may look for an outbreak five or ten years hence. *Excision of the chancre does not abort syphilis,* clinically or experimentally, in man or in monkey. Indeed, Metchnikoff and Roux have shown that excision of the site of inoculation after twenty-four hours does not prevent the development weeks later of true chancre or true syphilis. They have prevented the development of syphilis by the application of mercurial ointment within eight hours of inoculation, but this is the only abortive treatment they have found of any service. Hence, *if the patient is seen within twelve hours of a suspected contact, mercurial ointment should be well rubbed into the suspected part.* After twenty-four hours the ointment may be removed.

Beyond this nothing—neither cauterization, nor excision, nor internal medication will abort syphilis.

GENERAL PRINCIPLES OF TREATMENT

In the treatment of syphilis the physician has two ends in view: viz., to prevent the appearance of symptoms and to cure

them when they do appear—to control the disease and to cure its lesions.

These two ends are entirely distinct, and unless this distinction is kept constantly in mind a discussion of the treatment of syphilis becomes a mere muddle.

For example: It is routine practice to administer mercury without potassium iodid during the first year of syphilis. But if the patient develops a gumma during that first year, we promptly administer iodid, at the same time continuing the routine mercurial treatment. We are attacking the disease by mercury, the lesion by iodid.

Again, suppose in the course of the first year of syphilis there appears an obstinate squamous syphilid. The routine treatment fails to control, and we have recourse to more forceful measures, to higher doses or a more active method (e. g., injections) of administering mercury. These we employ solely on account of the lesion, and having cured this we drop back to our routine treatment.

But if the lesion persists in relapsing, then we change our routine treatment of the disease in some way, in order to check this tendency, to prevent the outbreak of further symptoms.

Thus there are two treatments of syphilis, the one, routine to prevent relapse and control the disease, the other, symptomatic, to cure lesions.

THE ROUTINE TREATMENT

The routine treatment of syphilis is hygienic, tonic, and specific. The specifics are often ineffective unless aided by the others. No one of them may be depended upon alone. They form component parts of one rational system.

No two authorities employ precisely the same routine treatment of syphilis. In the matter of hygiene we are all more

or less neglectful. In the matter of drugs, a generation back one year of treatment, perhaps by iodid alone, was the most a patient need expect; to-day mercury is universally employed for two or three years. Iodid is generally administered during the last year of treatment (and often for some time thereafter), and the mercury is given by mouth, by inunction, or by hypodermic.

In such a confusion it is impossible to do justice to every man's opinions. One can only describe one's own practice and the reason for it, leaving to objectors the privilege of following any other system that gives them better results.

But first a word of explanation as to the cause of this Babel. In the routine treatment of syphilis we are attempting to come as near as possible to what is confessedly an unattainable ideal by any of the systems at present in vogue; viz., the absolute and permanent prevention of all relapses of syphilis. Now syphilis is no exception to the rule: the less curable a disease, the greater the number of "sure" cures for it. For the vision of man is short and the cold statistics by which he attempts to widen its scope are notoriously misleading. If I carry a patient through the first few years of his disease, I send him off "cured" with a confidence in my method of treatment little dampened by the relapse of another whom I treated in the same way five years ago. For some patients relapse no matter what you do for them, and others are permanently cured under any form of treatment.

But there are one or two rules upon which the routine treatment may be based; rules often broken nowadays.

Hygiene is of the First Importance.—The brilliant effect of drugs often blinds us to the need of hygiene in syphilis. Yet hygiene is all-important.

The hygienic treatment of syphilis includes all the ordinary laws of health. Regularity of meals, of sleep, and of the bowels is necessary. No special diet is required. Air, exer-

cise, and light, essential to all animal well-being, are particularly so in obstinate chronic disease.

In obstinate cases change of air is most useful. The nature of the change is not important: from seaboard to mountain, from inland to shore, sea trip or dry and high. Six weeks in any locality is long enough for the maximum beneficial effect. This effect is most strikingly illustrated by the patient's ability to take iodids. I have more than once observed that a patient who could not take enough of this drug to control his symptoms in New York could readily do so out of the city. I have remarked the same fact in patients sent me from Chicago and elsewhere; they could with ease and advantage tolerate heavier medication in New York than at home. Many obstinate cases that fail to respond to treatment at home, especially if that home be in the city, make rapid strides toward recovery as soon as the air and surroundings are improved. Mercury and iodid do not cure all syphilis. The old chronic cases, relapsing endlessly in the dark and crowded tenements of our large cities and returning year after year to our clinics, often need hygiene far more than medicine.

The Hot Springs of Arkansas.[1]—This Mecca of syphilitics requires a word of serious comment. That it has positive value I am sure. But its positive value is reducible to precisely this: Under the stimulation of changed surroundings and aided by the hot baths, the old broken-down syphilitics, the alcoholics, those who have ruined their digestive functions by inundating themselves with iodid of potassium—in short, all those whose lesions fail to improve at home because they cannot tolerate the high doses of mercury and iodid required to cure them, can take such doses at the Springs.

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[1] The following remarks apply equally to the syphilis cure at Aachen, Germany.

Whether this improvement is due chiefly to the greater willingness of the patient to attend to his cure, or to some thermic or electric or chemical property of the water, or merely to the change of climate and the hot baths, I do not know. But the fact remains: at the Springs certain desperate and unmanageable cases do better than elsewhere.

But this is not to say that all syphilitics should go to Hot Springs; far from it. In the past five years I have sent only two patients there, and of those who have come to me from there, nine out of ten were no better off than they would have been from intelligent treatment at home; while some were greatly harmed, utterly dilapidated from overtreatment, and requiring months or years to recover from the effects of their experience.

The statement that the relapse of syphilis is rarer after a treatment at Hot Springs than after any other course is absolutely untrue.

The Curative Effect of Hygiene.—The average patient does well enough regardless of his surroundings just as the exceptional patient gets well regardless of his treatment. But the fundamental fact remains that *every* syphilis *may* relapse, and that the good health of the patient is the one thing that stands between him and relapse, mercury or no mercury. Mercury controls the disease, and doubtless prevents relapse by attenuating the spirochetæ; but it does not destroy them all, and it should be our constant effort to hasten and assure a complete cure by keeping the patient in the best possible physical condition.

Extreme temperance in the use of alcohol and tobacco (pages 103, 129), the avoidance of every excess in work, worry, and pleasure, plenty of fresh air, exercise, and good food—such are the fundamental rules. They are not universally applicable, but we must keep them in mind and din into our patients the necessity for keeping "in training."

Tonics must not be Forgotten.—If the patient is anemic, and this anemia does not improve under mercury, a little iron helps. The proprietary organic iron compounds are useful, but I often use the following:

℞ Sodii arsenat. .... gr. ss to j (.03 to .06 gm.);
Ferri reducti..... ʒj (4 gm.);
Pulv. rhei ..... ʒj to jss (4 to 6 gm.).
M. Fiant capsules No. xxx.
S.: One capsule t.i.d.

The mercury may be embodied in this capsule and strychnin may replace the arsenic. For neurotic patients hypophosphates and glycerophosphates also help; while, if they have lost much weight cod-liver oil and other fatty emulsions are appropriate.

But it is in treating syphilis of the nervous system that tonics and hygiene are peculiarly applicable. Mercury and iodids may fail signally—worse than this, if persistently pushed, they may do great harm by impairing the patient's vitality. Hence the rule in such cases to push the specifics to the limit of toleration, then to stop them entirely and institute a course of tonics amidst the best surroundings available, after which return again to the specifics (page 148).

The Specific Tonics.—I think that *Zittmann's decoction* is a remedy of positive value, especially in late syphilis, when there is cachexia, anemia, irritable stomach, loss of appetite, moderate constipation, and particularly when the stomach will not take the iodids kindly. The senna encourages intestinal action, the sarsaparilla undoubtedly has an influence (if left out the remedy is decidedly less effective), and the mercury is presented in a dose calculated to exercise its tonic effect. But the old-fashioned Zittmann's decoction was full of unnecessary ingredients in its composition, and was

troublesome to make, difficult to take on account of the quantity required as a dose, and its administration was surrounded by unnecessary rules and precautions. Starting with the original decoction, and then modifying it by McDonnell's formula, I have gradually dropped one thing after another until I now use the following formula, regulating the dose somewhat according to the purgative effect:

℞ Hydrarg. chlorid. corros ... gr. j (0.06 gm.);
Aluminis ..... ℥ss (2 gm.);
Extr. sarsaparilla ..... ℥ij (64 gm.);
Glycerin ..... ℥j (32 gm.);
Syr. sennæ ..... ℥iss (48 gm.);
Spts. anis ..... ℥j (4 gm.);
Extr. glycyrrhizæ ..... ℥j (4 gm.);
Aquæ feniculi ..... q. s., ad ℥viij (250 gm.).
M.
S.: ℥ss t.i.d.

As to the other so-called specific tonics, such as *succus alterans*, *mercauro*, etc., some practitioners cling to them and attribute excellent results to their use. But apart from some little tonic value I think they owe their reputation to being employed when the patient has been drowned in mercury and iodid, and is ready to improve as soon as he recovers from the poisonous effects of these drugs. Under such circumstances any tonic (or no tonic) will work wonders, and whatever tonic happens to be used gets the credit.

Such a condition is a striking indication of the effect of hygiene. The patient at the end of his course of "mixed treatment" is apparently far worse than when he began it; his lesion is uncontrolled, while he has gone to pieces completely under the treatment. But in a few weeks *without* iodid or mercury he improves and his symptoms diminish with incredible rapidity (Case XXVII, page 231).

Whether atoxyl[1] belongs among the specific tonics the future must decide.

Iodids do not Prevent the Relapse of Syphilis.—Most of us have been educated in the "mercurialization" school. We do not remember how many treatments used to be devised in order to avoid giving mercury. We rarely see a patient who has dragged along under some "sarsaparilla" or "decoction" or perhaps under heroic doses of iodid in utter misery from which only mercury could save him. My father's greatest claim to scientific fame is the fact that before the International Medical Congress at Philadelphia in 1876, he struck the first blow for freedom from the error that mercury properly administered is a poison. Since that time iodid has played a less and less important part in the routine treatment of syphilis. But though it is now generally recognized that iodin does not prevent relapses of syphilis, this drug retains its hold on the last years of the routine treatment of most physicians.

The precise reason for this practice I do not see; hence I do not accede to it. I employ iodin only for the cure of lesions, never in the routine treatment of syphilis.

Mercury does Prevent the Relapses of Syphilis.—Mercury certainly does not cure syphilis. It hastens a cure by destroying the spirocheta (page 33), by curing certain lesions, and by preventing relapses of the disease. This much everyone concedes. Those who go further and claim that mercury actually cures syphilis must admit that it does not cure all cases; therefore, it seems more prudent to believe that the disease is cured by time and hygiene, but only ameliorated by mercury.

MERCURY, ADMINISTERED IN SMALL DOSES, IS A TONIC.—I quote my father's words upon this subject: "This demonstration of the harmlessness of mercury, of its tonic influence,

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[1] Cf. Lesser, *Deutsche med. Wochenschr.*, July 4, 1907.

I think I have clearly made.[1] I have shown that moderate doses of mercury continued for any length of time (up to several years, a time amply long so far as syphilis is concerned) not only do not debilitate but act as a tonic, augmenting the number of red blood cells in health or in syphilis. I have amplified the subject, showing that mercury cannot be held responsible for late lesions.[2] Finally, upon this foundation, I devised a method of treatment which I called the tonic treatment of syphilis,[3] so naming it because in it mercury can be used in such a way as to exercise its tonic influence, while at the same time it is controlling the disease. I have been persistently misunderstood in this matter. *It is not as a tonic, or because it is a tonic, that mercury cures syphilis* or alleviates it; but mercury may be so used in the treatment of syphilis that, over and above its specific influence, it may still not only do the patient no harm, but may be actually a tonic to him, doing him good—a point that no one has hinted at before, and many do not yet believe; and on this account only I have called this method the tonic treatment of syphilis."

In the Routine Treatment of Syphilis Small "Tonic" Doses of Mercury are More Effective than Poisonous Doses.—This statement is obviously true, and is fortified by the experience of past centuries when mercury was administered in doses almost incredible to a less heroic age. Yet these furious assaults were utterly inefficient in controlling relapses.

So Long as a Sufficient Amount of Mercury is Exhibited, it Matters not Whether it is Given by the Mouth, by the Skin, or by Injection.—The attitude of many syphilologists is this: We find injections (or inunctions) far more effective in curing symptoms than the administration

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[1] *Am. Journ. Med. Sci.*, January, 1876.

[2] "Treatment of Syphilis," Trans. Int. Med. Congress, 1876.

[3] "Tonic Treatment of Syphilis," New York, 1877.

of pills. Hence the same discrepancy must exist for the *prevention* of symptoms. The premise is not absolutely accurate, for certain symptoms are cured quite as quickly by internal medication as any other way, though in many instances injections and inunctions are certainly far more effective. But the reason for this effectiveness is that by these methods larger doses of mercury are promptly absorbed with less danger of poisoning than if the drug were given by mouth. But this quality of treatment, though excellent for the cure of symptoms, is unnecessary for their prevention, *so long as a sufficient amount of mercury can be absorbed by the stomach.*

The contention for injections and inunctions in the routine treatment is, therefore, valid only when the patient cannot, without poisoning, ingest enough of the drug to control the disease.

As a matter of clinical experience, so far as my records and my recollection go, there seems little to choose between the three methods of administering mercury for the *routine* treatment of syphilis, except in those patients who cannot digest enough mercury, or who cannot bear the pain of injections or the irritation of inunction.

THE SYMPTOMATIC TREATMENT

The principles governing the symptomatic treatment are generally accepted and merely require brief enumeration.

Excepting the Early Painful Symptoms, the Secondary Lesions of Syphilis Call for Mercury.—This is true even of the latest secondaries.

The Early Painful Symptoms Call for Iodid.—Accepted doctrine.

Gummatous Lesions May be Cured by Iodid alone but are Better Managed by "Mixed" Treatment.—Iodid of potassium may cure gummatous lesions by its own virtues, even

though no mercury has been administered. Thousands of cases have proven this in the past, yet nowadays when all patients take mercury, it is the fashion to belittle the effects of iodid, and to attribute its virtues to "setting free" the mercury. But this is a misrepresentation of the facts. Recognizing that iodin has little or no effect in preventing relapses of syphilis, and that its destructive effect upon the spirocheta is but slight, we must see the necessity of enforcing its action by mercury. Yet for the cure of gumma iodid is the prime requisite; mercury is accessory and preventive of relapse (Case XXVI, page 202).

Sclerotic Tertiary Lesions and Lesions Difficult to Classify Require "Mixed" Treatment.—For such lesions, for instance, as tuberculo-ulcerative, serpiginous syphilids, both iodid and mercury are required. Some do better on high doses of the one, some on the other.

Lesions of the Nervous System, if Gummatous,[1] Require High Doses of the Iodid, if Arterial, High Doses of Mercury (with Iodid), for Short Periods, with Intermissions for Hygienic Treatment.—The two factors which differentiate the treatment of syphilis of the nervous system are the importance of scar tissue in causing symptoms and the importance of hygiene in effecting a cure.

We attack, let us say, a syphilitic hemiplegia. At first it yields to treatment; later the improvement ceases. Then one is tempted to inundate the patient with mixed treatment in the hope of clearing up these rebellious last traces. But this often results in destroying the patient's health without improving his symptoms. Thousands of sufferers are thus tortured with useless medication. How shall we avoid this? How distinguish what is curable syphiloma from what is in-

[1] It is often clinically impossible to distinguish gummatous from arterial lesions, and it is accordingly good practice to push *both* iodids and mercury in short courses.

curable scar? The only solution is to give short, sharp courses of specific medication interrupted by periods of purely hygienic treatment (page 143). Thus we can form an estimate of the patient's condition and desist from specific medication when it proves futile.

The Symptomatic Treatment Should Always be Vigorous.—The one thing probable (yet not quite certain) about relapses of syphilis is that they are caused by spirochetæ, which spirochetæ multiply in the active lesions of the disease. Hence, as an important adjunct to our routine treatment of syphilis, the symptomatic treatment should be vigorous in order to destroy as rapidly as possible these nests of infection. This is peculiarly true of the very lesions one is most apt to neglect. The superficial secondary syphilids of the mouth and upper air passages are among the commonest and the most infectious lesions of syphilis. Yet they are so obstinate and so benign that the patient neglects them if his physician permits, and flatters himself on his good health when his mouth is covered with active ulcers and mucous papules.

This Vigorous Treatment Must be Continued Even After the Lesion has Apparently Healed.—Neumann very justly insists upon this point: viz., that the scars of apparently healed syphilitic lesions often contain areas of tissue which have the microscopic appearance of active syphilis. Hence the necessity of continuing vigorous treatment after the lesion has apparently healed (page 36).

Vigorous Treatment is that Treatment which Effects Prompt Improvement.—We judge the treatment by its effects. If a gumma melts away under a few drops of iodid and a mild dose of mercury given internally, it is quite unnecessary to have recourse to harsher methods. Yet another case, apparently similar in every other respect, may not yield until 50 minims a day of potassium iodid are given; another may require 100, another 200, while still another may be obstinate

until the mercurial effect is intensified by using inunctions or injections. Each lesion thus has its requirements, which can be learned only by experiment. One can never tell beforehand. An ancient destructive syphilid often yields as rapidly to light doses as to heavy ones. The only rule to follow is this: Each lesion has its "dose," to which it yields promptly. Find this dose as rapidly as possible, and do not be misled by previous experience with other lesions into giving too small a dose to control the lesion or too large a dose for the needs of the case.

Local Treatment Avails Little.—Local treatment, whether by salves or powders or by electricity or X-rays or any of the newly discovered rays, avails little in syphilis. To this rule there are two notable exceptions. Secondary mucous papules and ulcers in and about the mucous membranes heal rapidly under light cauterization (page 347) and condylomata wither if kept dry (page 346).

The local treatment of chancre and all other open lesions is palliative and cleansing. Some form of mercury is usually applied for its slight local effect,[1] but for real results we employ systemic treatment.

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[1] Experiment has proven that inunction or injection of mercury encourages healing of adjacent syphilitic lesions; but clinically this local action is of little importance.

CHAPTER XI

THE ROUTINE AND SYMPTOMATIC TREATMENT

THE ROUTINE (TONIC) TREATMENT

MERCURY, given in physiologic or "tonic" dose increases the number of red blood cells and the percentage of hemoglobin; it accelerates vital changes and increases the urea excreted. It is a true tonic. This tonic action is limited by no time, but only by the amount of mercury administered. In syphilis we habitually give a rather high dose in order to control the disease, and thus we may mildly poison the patient without salivating him; he then loses weight and again becomes anemic. Yet this is not due, as is currently believed, to the long-continued course, but to the long-continued administration of a mildly poisonous dose.

Exceptionally we even encounter a patient who is salivated by very minute doses of mercury, whatever the method of administration. Such a one cannot take the drug to any purpose in a tonic dose. He must be poisoned that his disease may be controlled.

Moreover, the poisonous dose of mercury varies not only with the dose of the drug, but also with the salt employed and with the method of administration. Most patients are more readily poisoned by internal treatment, for example, than by injection.[1]

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[1] Taking a given syphilitic lesion as a test, we can often cure it by hypodermic medication without poisoning, whereas it resists even poisonous doses given by the mouth.

Therefore, the dispute between Fournier and most other European authorities, on the one hand, the the followers of Hutchinson and Keyes, on the other, resolves itself into this: Can syphilis be properly treated by administering mercury in truly tonic doses, or not?

We maintain that this can be done in the average case; our opponents dissent. After all, the question is a relative one, and would be of no great moment were we not convinced of the fact that *the most important ally the physician can invoke in the treatment of syphilis is habit.* If you put your patient on a treatment calling for interruptions of six weeks here and six months there—interruptions of which he must recognize the purely arbitrary nature—he promptly becomes careless and forgetful. But if you can tell him: "Take so many pills three times a day, every day, winter and summer," he will, under the shadow of his first fear, form the habit which will make him almost regret the loss of his treatment when he stops it. Or, if injections are used, it is much easier for him to remember to come once a week or twice a month than to be bothered with courses of irregular length and frequency.

Therefore, when we can, we employ the continuous tonic treatment of syphilis. But if the dose required to check the disease is poisonous to the patient, then we use interrupted courses perforce.

HOW TO ADMINISTER TREATMENT

The Vigorous Interrupted Method.—Our forbears had the pleasant habit of inundating the victim with mercury until he was wretchedly salivated, and then setting him by until an outbreak of the malady gave the signal for another mercurial onslaught. In those days the cure was worse than the disease.

The only relic left us of this disastrous system is the practice of returning annually to the Hot Springs for a severe

"cure." This is an extravagant and ineffectual way to treat syphilis, which, be it said, the physicians at the Springs do their best to discourage.

The Modern Interrupted Method.—Throughout the Continent of Europe it is still customary to give mercury in interrupted courses. In part this is an inheritance of the ancient heroic system; in part it is due to the feeling that relapses of syphilis occur with some degree of constancy and are, therefore, to be met by an equally constant recurrence of treatments; in part to the belief that mercury, constantly administered, becomes poisonous; and sometimes to the exigency of the treatment employed.

If one uses inunctions or soluble injections in the routine treatment of syphilis, the treatment must be intermittent.

In Germany and Italy intermittent mercurial treatment is given somewhat as follows:

During the first year three courses of injections (usually soluble) or inunctions, the first two lasting four to six weeks each, the last about four weeks.

During the second year, two courses of about four weeks each.

During the third and the fourth year, one four weeks' course.

In France mercury is usually administered (internally) for two months at the onset, then during alternate months until the end of the second year, every third month during the third year, every six months during the fourth year.

Fournier's course is the following: At the onset a sharp course of eight weeks, then (whatever the patient's condition) an interval of six weeks. Then six weeks of treatment followed by six weeks' interval, then six weeks of treatment followed by eight weeks' interval (twice).

During the second year three courses of six weeks each.

In the third year the administration of iodid is begun;

two iodid courses and two mercurial courses of the same length being given alternately.

During the fourth year no mercury, but three iodid courses.

During the fifth and sixth years two courses of iodid (in each).

In England and America treatment is more nearly continuous, and usually by the mouth.

It were futile to be more explicit or to multiply examples. No two authors preach precisely the same doctrine. One can only judge the merits of the different systems in the light of experience, comparing published results with cases witnessed, and thus weighing the practicability of each. Any one of them is, doubtless, efficient if the patient can be made to follow it through.

The Continuous Tonic Treatment.—My chief reason for preferring this method I have already stated. The objections to inunctions and to insoluble injections, the only two forms of treatment that may be said to require interrupted treatment, need not be repeated here. Thirty years of additional experience have but confirmed the thesis defended in 1877 [1] that syphilis may be successfully treated by mercury in tonic doses.

Method of Administering Mercury.[2]—The intelligent treatment of syphilis means the treatment of each case according to its needs. No two are precisely the same. Therefore, in laying down rules for a system of administering mercury, one must bear in mind that exceptions to these rules are constantly cropping up, and that under different circumstances it is conceivable that these exceptions should overwhelm the rule itself. Thus I have no quarrel with those who prefer

[1] "Tonic Treatment of Syphilis," Keyes, 1877.

[2] The explanation for many statements in the following paragraphs will be found in the preceding chapters.

interrupted courses to the tonic treatment, nor yet with those who employ inunctions or soluble injections in the routine treatment. I follow the system that gives me the best results.

I prefer internal medication or insoluble injections. I leave the choice with the patient. If he can and will take a sufficient dose internally, and if this controls relapses adequately, and he waxes fat, let him do so. But if he cannot digest the necessary dose, or if the symptoms incessantly relapse, or if he remains under weight, then some change must be made in the system, and injections for a time at least are useful. Moreover, certain patients prefer injections. I respect the preference, though I do not believe that in the average case injections are a whit more effective for routine treatment than internal medication.

THE TONIC TREATMENT

When the patient comes with his early lesions upon him he requires (*a*) that these lesions be controlled and (*b*) that their relapse be prevented.

First, he is given a preliminary instruction, covering the following points:

1. The gravity of his disease, the duration of treatment (page 157), the probability that his symptoms will be mild if he attends to treatment (page 124), the fact that he must continue treatment while well, just as though he were sick.

2. The great importance of hygiene (page 140), the necessity for abstinence from alcohol (page 129), and moderation in the use of tobacco (page 130).

3. How others are infected, and how to avoid this infection (page 53).

4. His need to have his teeth put in good order and kept so (page 163).

5. The necessity of reporting every little while that he

may be weighed (page 84) and his throat (page 336) and urine (page 165) examined.

The mercurial treatment is then instituted by an injection if the lesions are grave, but usually with G. and L. granules (page 191).

Fifty of them are ordered, and (if it is a Monday morning) he is given this list:

Monday	..	1	1
Tuesday	1	2	1
Wednesday	2	1	2
Thursday	2	..	..

This means he is to take one granule after lunch and one after dinner on Monday; one after breakfast, two after lunch, and one after dinner on Tuesday, etc., and to report on Thursday.

If all goes well, his list is then continued to the following Monday, thus:

Thursday	..	2	2
Friday	2	3	2
Saturday	3	2	3
Sunday	3	3	3
Monday	4	..	..

with instructions that if he has two colicky, watery stools a day, he is to report immediately.

The dose is thus increased one granule a day until one of two things occurs: the colicky diarrhea or the subsidence of the lesions.

If the diarrhea comes first, the dose is dropped to half this maximum dose, and held there until the symptoms subside, when *the regular "tonic" dose*

2.....2.....2

is instituted. If the lesions disappear before diarrhea occurs, the "tonic" dose is instituted at once.

If the lesions refuse to subside within a few days, they are treated *secundum artem* (page 159).

If the patient cannot take the tonic dose of G. and L. granules, gray powder, bichlorid, or mercury and iron are tried (page 170). If these fail, turn to injections (page 174). If these are too painful, fumigation (page 188), (black oxid, two drams once a week), or inunction (by the German method described on page 153).

This is the fundamental principle of the old "tonic" system, shorn of its "full dose," "reserve dose," and "tonic dose." We only retain the notion of a minimum dose. If the patient cannot take the dose stated above, his treatment is not adequate. No matter how clean his skin or how perfect his general health, he must take a certain minimum dose of mercury to minimize the danger of late relapses. This dose is arbitrary—theoretic, if you will;[1] yet without it there is no adequate prospect of cure.

After several months of treatment the digestion of the patient may begin to resent the mercury. Interrupt treatment a week or two and this will usually pass.

In the second year treatment is continued as in the first.

In the third year no treatment during the first six months. Resume routine treatment during the last six months.

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[1] One instinctively looks upon the relapse of symptoms as the only index of inefficient treatment. Yet we daily meet vicious relapses in late years in patients whose lack of early symptoms led them to take no treatment at all. Without a theoretical minimum of mercury we have no standard to go by except the patient's general health. Under well-managed tonic treatment he should slowly gain weight until he regains or even exceeds his normal weight; his blood should become normal and, within six months or a year, he should be in the pink of condition. This tonic effect keeps pace with the antisyphilitic effect of mercury (though, as already observed, they are by no means identical) and is the best omen for the future.

In the fourth year no treatment if the patient has remained clean for two years; if not, three months (or more) of treatment.

In the fifth year as in the fourth.

In the sixth year matrimony permitted if the patient has been two years without lesions and without treatment (page 63).

Such is the routine treatment I prefer. I believe it as efficient as any, and more convenient than most.

Tonic Treatment after Late Relapses.—After the lesions are cured, two courses of six months, with six months' interval and a final course of three months, suffice. Even though the patient has had no early treatment, I see no virtue in prolonging the preventive course. But the patient should be under observation for full two years, and if during that time any further relapses occur, two more courses of six months should be given.

WHEN TO BEGIN ROUTINE TREATMENT

Treatment Should be Begun as Soon as the Diagnosis is Made.—Of this there can be no question. Discussion centers about the diagnosis. Shall we begin treatment as soon as we *suspect* syphilis, shall we wait until we are morally certain, or shall we await physical *objective certainty*?

At the onset of the disease, when the patient has only the primary lesion to show, *nothing less than a physical certainty justifies the beginning of treatment.* Four things are required for a positive diagnosis of chancre: viz., a characteristic sore, typical lymph nodes in the groin, negative autoinoculation and positive finding of spirocheta by an expert microscopist (page 26). Lacking any one of these we must await the confirmation of secondary symptoms, for so grave a diagnosis must depend, not upon any opinion,

however expert, but upon the facts: physical certainty is required.

Later in the disease, if the patient comes clean, unscarred, and with an unconvincing history of early symptoms and treatment and if the period of ordinary routine treatment has not yet elapsed, treatment should be instituted on the ground that, inasmuch as there is no immediate possibility of perfecting the diagnosis, *it is prudent to take his syphilis for granted* and put him through a regular course of treatment rather than to await developments, and thus increase his risk of grave lesions: moral certainty suffices.

The same reasoning applies to an apparently clean wife impregnated by a husband in florid syphilis. To save the child she should follow a routine treatment through the first seven months of pregnancy.

Finally, *if the patient known to have syphilis comes with some suspected secondary or tertiary manifestation, this mere suspicion justifies treatment*; the diagnosis is made certain by a test course of treatment (page 205); then a routine course of preventive treatment is instituted.

Thus under different conditions we may require a physical certainty, a moral certainty, or a mere suspicion of syphilis before instituting routine treatment. But at the onset of the disease the strongest possible certainty is always required.

TREATMENT OF THE LESIONS OF SYPHILIS

No two cases of syphilis can be treated alike, for in no two do the lesions of the disease appear in like rotation or with like severity. And even if they did, that would not make general rules any more applicable; for the treatment that cures one lesion may not have the least influence upon its counterpart in another patient or in the same patient at another time.

Hence it is impossible to generalize in discussing the treat-

ment of the lesions of syphilis. Each must be attacked with all the resources of hygiene, tonics, and specific medication at our command. The treatment must be vigorous, yet not vigorous beyond the patient's needs (page 149). The dose of mercury or iodid required to control each lesion must be discovered and administered as soon as may be; yet this dose may not be recklessly exceeded.

That new lesions appear from time to time during the routine treatment is no indication that this treatment is inefficient; and, on the other hand, the mildest mucous papule is not too slight a lesion to merit active treatment for its cure.

One special point, too, must be borne in mind. Certain lesions must run their course in great measure uncontrolled. I have just cured a chancre redux which I began to treat six months ago. I attacked it first with mixed treatment. The patient's gums were touched, he was completely iodized, yet the lesion persisted. After a short respite I gave a course of gray-oil injections. Again the gums were touched and the lesion much reduced; but it was not cured. And now a second course of gray oil has cured it. The cure has been singularly slow, yet to have proceeded more viciously would have so undermined the victim's health as to make him worse rather than better.

Certain other points of treatment will come up for consideration as we deal of the lesions *seriatim*.

CHAPTER XII

PHYSIOLOGICAL EFFECTS AND TOXICOLOGY OF MERCURY

WHETHER administered by the mouth, by the skin, by inhalation, or by injection, mercury enters the lymph channels and is distributed throughout the body by the blood stream. Experiments on animals show that the metal is deposited everywhere in the body, but chiefly in the kidneys, the liver, the large intestine, and the spleen.

Mercury is eliminated in all the secretions, but chiefly in the urine and feces.[1] When given by injection it appears in the urine within a few hours. Excretion takes place very slowly. Indeed, certain German authorities have reported the identification of mercury in the urine after incredibly long intervals. Thus, Lang[2] cites Landsberg's case (interval ten months) and Welander's case (six to twelve months) and then four of his own in which mercury could be identified in the urine respectively, seventeen months, twenty-three months, three years, and ten years after the last treatment.

BENEFICIAL EFFECTS OF MERCURY

Mercury in small doses is an excellent tonic, quite apart from its effect upon syphilis. It has also a specific tonic effect upon patients debilitated by early syphilis; it increases

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[1] Schuster believes it is excreted chiefly in the feces, but most authorities agree that the kidneys are the chief agents.

[2] "Pathol. u. Therap. d. Syph.," 1896, p. 831.

the number of red blood cells and the percentage of hemoglobin. Moreover, it cures the lesions of syphilis, and in great measure prevents their relapse.

The above points are all discussed elsewhere.

TOXICOLOGY

It so happens that to control syphilis we often have to give mercury in more than the tonic dose. To cure the lesion we have, in some measure, to poison the patient. This poisoning may show itself in various ways: There may be salivation, entero-colitis, nephritis, besides evidences of local over-irritation and general debility from prolonged overdosage. The susceptibility to mercurial poisoning varies widely in different persons.

But there are a great many other ways in which the mercury does not poison. It does *not* "corrode the bones" or "make the hair fall out," and there is no such thing as "mercurial neuralgia." The popular belief in these horrors is a relic of the days of the antimercurialists.

Salivation.—Salivation is the commonest form of mercurial poisoning. It may occur from the use of mercury under any form. The great *predisposing cause* to salivation is inflammation of the gums, whether due to tartar, to neglect of the teeth, or to Riggs's disease. General debility may be a contributory cause, while the patient's native susceptibility to the drug is the underlying element, causing one person to become salivated much more readily than another.

Symptoms of Mild Salivation.—In many urgent syphilitic conditions mercury is pushed until "the gums are touched," i. e., until mild salivation is produced. This is the signal for stopping treatment.

The first symptom noted by the patient is a *coppery taste* in the mouth. This is accompanied by a very heavy *disagree-*

able odor to the breath; the *tongue becomes edematous* and shows the marks of the teeth along its edge; the *gums bleed*; the *teeth feel sore* and elongated.

Examination of the gums reveals localized areas of congestion, *especially about the lower incisors* or about any decayed tooth. When the lesions are more pronounced the gums actually ulcerate. The *pathognomonic mercurial ulceration appears behind the lower incisors and back of the lower wisdom teeth.* Ulcerations may occur elsewhere in the mouth, and it may be almost impossible to distinguish the lesions due to the medicine from those caused by the disease (page 343).

Actual *salivation* is usually slight at first, though in some cases the excessive flow of saliva is the earliest symptom.

Symptoms of Severe Salivation.—Nowadays one does not see "good, old-fashioned" salivation, the patient depressed, feverish, stupid, his tongue swollen beyond recognition and gangrenous in spots, a constant stream of ropy saliva drooling from his swollen lips, his teeth loosened and falling, even his jaws becoming necrotic, and his breath permeating the atmosphere with an intensely disgusting fetid odor.

Yet conditions intermediate between this and the mild type of salivation described above are often seen.

Treatment of Salivation.—So important is the care of the mouth in the *prophylaxis of salivation* that the patient about to take a course of mercury should be carefully instructed how to keep his mouth in good condition.

He must first have his teeth filled and thoroughly cleaned by a dentist.

He must brush his teeth morning and night.

He must rinse his mouth morning and night with chlorate of potash, gr. xv (three 5-grain tablets), dissolved in half a glass of water or with an alkalin mouth wash.

He must smoke little.[1]

He must report the instant his gums become swollen or sore.

The *treatment of mild salivation* consists in painting the gums with one of the following solutions:

℞ Tr. myrrh.,
Tr. iodi comp. } .....āā ʒj (4 gm.).
Aquæ

S. Apply to gums once or twice a day.

Or

℞ Acid lactic ..... ʒj (4 gm.);
Aquæ ..... ʒij (8 gm.).

S. Apply to gums once a day (Tennesson).

Or

℞ Acid chromic ..... gr. xv (1 gm.);
Aquæ ..... ʒv (20 gm.).

S. Apply to gums every other day (Berdal).

The ulcerations may be touched once a week with liq. hydrarg. nitratis, fifty per cent, or with the solid stick of nitrate of silver. Meanwhile, let the patient suck during the day two or three tablets (āā gr. v) of chlorate of potash.

The bowels must be kept open (they are usually loose, anyway) and the *mercury stopped.*

For severe cases free purging and diuresis, hot baths, a milk diet, and *suspension of the mercury* constitute the most important treatment. The local applications may be used as in mild cases, but cauterization had better be omitted. Ulceration of the cheeks may be lessened by separating them from the teeth with pledgets of cotton dipped in boric-acid solu-

[1] I am not sure that the irritation of tobacco is an important contributory cause of salivation, but I know it predisposes the patient to syphilitic mouth lesions.

tion or with a piece of raw turnip. Belladonna or atropin theoretically ought to stop the salivary flow, but they are not very efficient. Chlorate of potash, four per cent, administered internally in doses of one or two ounces a day is highly prized, but I cannot say I have seen it do much good.

Entero-colitis.—A sharp diarrhea with colic usually accompanies severe salivation. This is due to the elimination of the mercury into the bowel.

But internal administration of mercury sets up an irritation of the bowels by direct local action. Hence result griping pains and diarrhea, which may even go on to chronic colitis with constant diarrhea and passage of blood and mucus, marked debility and anemia.

To prevent this is the art of managing the internal treatment.

To cure it stop the mercury, put the patient on a fluid diet without milk and, after giving a sharp purge, administer bismuth (gr. x), beta-naphthol bismuth (gr. x), castor oil (ℳx), and salol (gr. v), or opium.

Mild cases are promptly relieved by stopping the mercury.

Nephritis.—The normal kidney secretes mercury in small doses perfectly kindly. But mercury may produce congestion of the kidney or nephritis either.

1. When the kidneys are diseased, or
2. When mercury is given in poisonous dose.

Any form of acute or chronic nephritis makes the kidneys a poor filter for mercury, and since mercurial nephritis is the rarest form of mercurial poisoning, there is always room for suspicion that the kidney is chiefly at fault. This is further emphasized by the fact that there occurs an acute secondary syphilitic nephritis which may be mistaken for mercurial nephritis (cf. Ledermann [1]).

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[1] *Dermat. Zeitschr.*, 1905, vol. xi.

The *symptoms* of mercurial nephritis are those of acute congestion of the kidneys from whatever cause.

The *treatment* consists in stopping the mercury. The *prognosis* is excellent.

The chief practical point in regard to nephritis is that *the urine should be examined when mercurial treatment is instituted, and should be repeatedly examined during any severe course of mercury.*

Albuminuria or nephritis does not contraindicate the administration of mercury, but does give warning that it must be given in small doses and with constant attention to the condition of the renal function.

Dermatitis.—Some skins are very sensitive to inunction, and in general the hairy portions of the skin become irritated most readily.

Mercurial dermatitis begins as an erythema which, in severe cases, goes on to an acute eczematous condition (mercurial eczema).

There is also an extremely rare dermatitis, due to a peculiar individual susceptibility to the internal administration of mercury. The eruption is scattered, erythematous, urticarial, or eczematous. It burns or itches; in fact, is a typical toxic eruption. Though said to be commonest after internal administration, the only case I have seen was a sharp urticaria following injection.

The *treatment* consists in stopping the mercury (for a time, and then using it in small doses), soothing lotions, and catharsis.

Ill-effects of Prolonged Mercurialization.—Mercury can be given quite indefinitely in non-toxic doses. But prolonged courses, even if only moderately severe, produce emaciation, anemia, and general vital deterioration, such as would ensue upon any chronic poisoning. Nowadays such a condition is seen only in overtreated cases of syphilis of the nervous system.

If the medication has been much prolonged, or extremely severe, the patient may be left with chronic nephritis or gastro-enteritis, from which he will never recover.

CONTRAINDICATIONS

There are no *absolute* contraindications to the occasional use of mercury; witness the universal employment of calomel as an occasional cathartic, even in nephritis.

But certain conditions constitute *relative* contraindications to its continued use. Such are the acute infectious diseases, tuberculosis and other wasting diseases, and nephritis.

When such conditions complicate syphilis they contraindicate the use of mercury only thus far: the drug must be given with extreme caution.

During an acute infectious disease it may usually be stopped entirely.

If there is tuberculosis or other chronic disease, the organism is peculiarly susceptible to mercurial poisoning, and, accordingly, the drug must be given in small doses, and its effects carefully watched, for the slightest mercurial poisoning is likely to lessen the patient's resistance and to encourage the progress of any complicating disease. Thus the unnecessarily vigorous treatment of Case V (page 19) terminated in salivation and diarrhea; the diarrhea persisted after the cure of salivation, and examination revealed the presence of extensive, inoperable rectal carcinoma. I have no doubt the progress of this disease was greatly encouraged as well by the syphilis as by its overenergetic treatment.

When syphilis and tuberculosis coexist, conditions are precisely similar. The tuberculosis is fed both by the syphilis and its overtreatment. But a moderate treatment, calculated to overcome the patient's syphilis without impairing his vitality, benefits also the tuberculosis. Under such circumstances, if

an intense mercurial effect is desired, it is safer to use soluble injections, the poisonous effects of which are more immediately appreciable and more short-lived.

Complicating nephritis produces almost identical conditions. It does not absolutely contraindicate the use of mercury (page 165), but it does require great caution and constant estimations of the urinary and circulatory conditions (urinalysis, estimation of blood pressure).

CHAPTER XIII

ADMINISTRATION OF MERCURY

MERCURY is administered in syphilis internally, by inunction, by intramuscular or subcutaneous or intravenous injection, and by fumigation. It may also be applied locally, but is rarely so used. Mercurial baths and mercurial shirts or sacks are no longer generally employed.

Internal administration of mercury remains the most popular form of treatment, though of late years intramuscular injections are constantly gaining in popularity. This latter method appeals to the physician on the ground of precision and efficacy; it forces the patient to keep under the eye of his adviser. Moreover, it spares the patient's digestion. Small wonder, then, that this method is employed exclusively by certain physicians, beloved above others by certain patients. Yet its inconveniences are such as to preclude its universal acceptance until it is proven as peculiarly effective in preventing relapses as it often is in curing lesions. This proof is not yet; indeed, so far as present evidence goes, a routine cure by injections is no more essentially curative than one by inunctions or by internal treatment. It is necessary constantly to remind oneself that many lesions heal quite as rapidly under other methods of administering mercury, and that many of the failures in cases treated by internal medication are due to the carelessness with which their cure is conducted, not to any fault inherent in the method.

INTERNAL ADMINISTRATION

The preparations commonly employed are protiodid, gray powder, bichlorid, and blue pill.

Protiodid (Hydrarg. Iodid. Flav.).—If the patient can take them I usually employ for the routine treatment Garnier and Lamoureux's granules of (green) protiodid; not because they contain iodin, for this is not present in therapeutic quantity; nor because they agree with the patient, for they are not peculiarly easy to digest. But their double virtue consists in this: they control the symptoms of syphilis very efficiently, and in case of poisoning they strike the bowel rather than the mouth—they purge before they salivate. Hence they possess the great practical virtue of being the safest form in which to confide mercury to the patient's own hands. With any other preparation he may produce grave salivation before he realizes what is wrong; with these granules he produces only a sharp diarrhea.

The granules contain a centigram each of protiodid. The minimum tonic dose is six granules a day.

Other iodids of mercury I do not employ for internal treatment. The biniodid is too poisonous. Yellow protiodid tablets, the favorite American remedy, in the routine treatment of syphilis are so vilely irritating to the bowel that the most hidebound constipation scarcely resists the explosive effects of the minimum tonic dose; viz., one grain a day.[1]

Gray Powder (Pulv. Hydrarg. Cum Creta).—This is an excellent preparation, preferred in England to all others. Six grains a day is the tonic dose. It may be administered in one- or two-grain tablets. Some patients take this far better than the protiodid (G. and L.). Indeed, I have often given as

[1] It is a great pity that in therapeutic efficiency no other manufacturer has been able to equal the G. and L. granules. They are chemically impure; that is manifest, yet they work better than the purest yellow protiodid.

much as fifteen grains of gray powder a day in attempting to control symptoms; yet very, very few patients can take even ten G. and L. granules a day for any length of time.

Bichlorid (Hydrarg. Chlorid. Corrosiv.).—If given alone, this preparation must be put in pill or capsule, for in tablet form it is likely to irritate the throat and esophagus. It is not much employed in early syphilis, but in the later stages of the disease is the favorite mercurial ingredient of "mixed" treatment (page 202). The minimum tonic dose is one sixteenth grain.

Blue Pill (Pil. Massæ Hydrarg.).—This is another preparation of mercury popular in the late rather than in the early days of the disease. The minimum tonic dose is three grains a day. But twice or three times that amount may often be given. A patient using this preparation should be under constant observation, as it strikes the gums more suddenly and severely than any other form of internal treatment.

Calomel (Hydrarg. Chlorid. Mitis).—This is no longer used in the internal treatment of syphilis.

Tannate of Mercury.—This is favored by certain practitioners. The tonic dose is three to six grains a day. I have not been able to discern its peculiar virtues. It appears to vary in strength in different preparations.

Many *other preparations* are fancied by certain individuals, but their vogue is slight, their merits ill-defined. It is proper, however, to mention two old and familiar mixtures containing mercury. The one is a *pill of mercury and iron*:

℞ Massæ hydrargyri,
Ferri reducti,
Gum tragacanth, ... āā ʒj (4 gm.);
Glycerin ..... q. s.

M. Fiant pil. No. lx.

S. One pill after each meal.

The other is often called *Sir Astley Cooper's tonic*:

℞ Hydrarg. chlorid. corrosiv. gr. i–ij (.06 gm.);
Tr. cinchonæ co..... ℥iv (100 c.c.).
M.
S. Teaspoonful in water after meals.

These form admirable combinations for the exhibition of the iron tonic or the bitters in conjunction with mercury. Either may be employed in the routine treatment. Zittmann's decoction (page 143) is only employed in late syphilis.

Rules for Treatment.—Besides the care of the mouth and the occasional urinalysis (page 166), the following precautions are necessary:

Do Not Give Opium With Mercury.—The callousness with which Continental authorities prescribe opium in the treatment of syphilis is quite inexplicable. Syphilis confers no immunity to this drug. The opium habit is as readily formed by a syphilitic as by any other.

Happily, we need insist no further. On this point American practice is eminently sane. Though recognizing that opium so constipates the patient as to permit him to take mercury internally in larger doses than is otherwise possible, we bow before the greater evil, the danger of forming an opium habit. The constipating effect of iron, though less than that of opium, makes it doubly useful in the mercury and iron pill mentioned above.

Do Not Begin With a Full Dose.—In order to permit the patient to take a full dose comfortably, it is safe practice to begin with half the dose and work up gradually; thus for protiodid, begin with three granules a day, and increase one daily until six are being taken; for gray powder, begin at three grains a day, and increase a grain a day; for blue pill, begin at one and a half grains a day.

To Spare the Patient's Digestive Organs an Intermission of a Few Days may be Occasionally Necessary.—The advent of summer and fresh fruit introduces in many patients an inability to take the full dose of mercury. Regulation of diet may help overcome this; but it is often necessary to intermit treatment a week or so. Such intermissions, even though occurring as often as six times a year, do not invalidate routine treatment. It is no more necessary that a patient should take so many pills *every* day than that, under the theory of interrupted treatment, he should respect certain definite intervals.

Merits of Internal Treatment.—Continuous internal treatment has the merit of being easy to remember and little trouble to follow. But it may be impossible for the patient to digest mercury in sufficient quantity for the control of symptoms or for their cure. It is the least efficient way to administer the drug; yet it is often quite efficient enough. If, in the routine treatment, it can be taken in the necessary minimum dose, and so prevents relapses, or, if in the symptomatic treatment it causes the lesions to heal promptly, there is no need to employ other and less convenient methods.

Yet, in employing internal medication, we must guard against the patient's carelessness. We must not permit him to neglect even mild symptoms or to become indifferent as to the regularity of his treatment.[1] He must be seen, examined, and encouraged at least every three months.

The *theory of the minimum dose* must be clearly understood. We recognize that mercury prevents relapses. We recognize that in certain cases even without mercury (or any other preventive) no relapses occur, or many years elapse be-

[1] One might infer that this excludes internal treatment from the clinic. But I cannot say that I have found any way of adequately treating syphilis in the poor: they simply will not take any care of themselves after their symptoms are relieved.

tween relapses. Hence we are bereft of any clinical evidence to prove that we are giving the proper preventive amount of mercury; we have to depend upon statistics and clinical impressions as to the proper average dose. We know that the dose that will prevent relapses in one case will not do so in another. But we know this only after the fact; i. e., after relapse has proven a given dose inefficient. Thus we must deal in generalities; we must insist that a patient take a definite amount of mercury whether he seems to need it or not. Hence the minimum tonic dose.

But one concession may be made to the individual patient. This minimum dose must be given in such a way as to be a tonic dose; the patient must thrive upon it. He must become free from syphilitic lesions; he must be in good trim; he must not lose weight; and if he has lost weight, he must regain it, and his blood must show a normal red cell and hemoglobin count. If under the minimum tonic dose of a given salt, the patient does not answer satisfactorily (in the main) to the above tests, some change must be made either in his hygiene or his medicine.

INTRAMUSCULAR INJECTIONS

The preparations of mercury employed for injections are either soluble or insoluble. The chief *soluble preparations* are the bichlorid, the benzoate, and the biniodid.

The chief *insoluble preparations* are the salicylate, gray oil, and calomel.

Bichlorid.—This salt is used in one per cent or two per cent solution with salt solution, various quantities of salt being used by different authorities. The following is an excellent combination:

℞ Hydrarg. chlorid. corrosiv. gr. xv–xxx (1–2 gm.);
Sodii chlorid. ..... gr. x (0.6 gm.);
Aquæ destillat. ..... ℥iij (100 c.c.).
Dose: 1 c.c. (ℳxv).

Benzoate:
℞ Hydrarg. benzoat. ..... gr. xv (1 gm.);
Ammonii benzoat. ..... gr. lxxv (5 gm.);
Aquæ destillat. .....q. s. ad ℥iij (100 c.c.).
Dose: 1 c.c. (ℳxv).

Biniodid.—The red iodid of mercury is employed either in oily solution or in so-called "serum" or mixed with potassium iodid. It appears under various proprietary names. I have employed cypridol, the Hannam's and Lafay's sera. These preparations are popular on the Continent, but are little used here. I prefer the following:

℞ Hydrarg. biniodid ..... ℥ss (2 gm.);
Potass. iodid. ..... gr. xv (1 gm.);
Aquæ destillat .....ad ℥iij (100 c.c.).

Among the soluble preparations the succinamidate, formamidate, oxycyanid, hermophenyl, and enesol deserve mention as being well spoken of, but they have not gained general favor.

Salicylate.—This may be put up in five per cent or ten per cent strength. The mixture I employ is:

℞ Hydrarg. salicylate ..... gr. xlviii (3 gm.);
Albolin (sterilized) ..... ℥j (30 gm.).
Shake. Dose: ℳx (0.7 c.c.).

The salicylate settles to the bottom and has to be distributed by vigorous shaking. The admixture of a little lanolin makes a better suspension but a thicker mixture.

Gray Oil.—Gray oil is an emulsion of metallic mercury. It is usually employed in fifty per cent strength, and is in this form a thick mixture which has to be heated before injecting (and requires that needle and syringe be heated as well), and can be prepared only by expert chemists. After employing Lafay's gray oil for about a year, I find that the following formula is equally effective, quite as painless, is easily prepared by any chemist, and, being fluid, requires no heating. Its only disadvantage, the increase in dosage, does not seem to make it more painful.

The formula is that of Lafay (in which the amount of mercury and of lanolin may be varied if the physician so desires):

℞ Hydrarg. bidestillat.[1] ..... ʒijss (10 gm.);
Albolin ..... ʒiij (13.5 gm.);
Lanolin ..... ʒjss (46.5 gm.).
Shake. Dose: ♏ij–vj (0.1 to 0.4 c.c.).

Calomel.—Calomel oil is made by pulverizing the drug, washing in boiling alcohol, drying in an oven, and then thoroughly mixing with ten parts of sterilized albolin. Dose: 0.5 c.c.

Method of Administration.—*The soluble salts may be given subcutaneously*, but this method is unnecessarily painful. They are best given—and *the insoluble preparation must be given—into the substance of some thick muscle.* Injection of an insoluble preparation subcutaneously invites abscess or gangrene.

The *site* usually selected for injection is the buttock. The pectoral muscles or the thick muscles of the interscapular region or loin may also be utilized. Successive injections are given on alternating sides, and no two injections should be put within an inch of each other on the same side.

[1] This is the "dentists' mercury."

Various landmarks have been suggested in order to prevent injury to any important structures. The simplest rule is that of Galliot: Run a vertical line, dividing the gluteal region into an inner third and an outer two thirds; lay a horizontal line two finger-breadths above the great trochanter. Where the lines intersect is Galliot's point. Injections may be safely made in this region and to the upper and outer side of it.

Grosz,[1] in an exhaustive monograph, disapproves of Galliot's point, and recommends the following:

1. A point midway between the ischial tuberosity and the upper edge of the trochanter on a horizontal line at a level with the latter.

2. A point directly above this and midway to a line joining the anterior superior spines.

3. A point on the same (horizontal) level as No. 2 and halfway between the trochanter and the intergluteal fold.

The implements are a sterilizable, hypodermic syringe (preferably all glass, as metal instruments are amalgamated by mercury), and a needle two inches long and of ample caliber;[2] for very stout persons the needle should be two and a half inches long.

Technic.—Boil the implements, wash your hands, and, making the patient lie upon his face, rub the buttock well with alcohol. Then plunge the needle up to the hilt in the appointed spot; if the pain warrants it,[3] withdraw the needle and plunge it in an adjoining spot.[4] Then fill the syringe with the solution to be employed, and note whether any blood is oozing from the needle; if so, change its location; if not screw on the

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[1] *Archiv f. Dermat. u. Syph.*, 1904, vol. lxxii, p. 1. Cf. also *La syphilis*, 1906, vol. iv, p. 71.

[2] I use a No. 19 standard wire gauze.

[3] This one can judge only by experience.

[4] If you strike bone withdraw a little.

syringe, aspirate the minute bubble of air from the needle, and inject the solution.

Authorities are divided as to the advantage of rubbing the spot vigorously after injection. If it bleeds, a square of adhesive plaster may be clapped on with instructions to remove it in a few hours; otherwise no dressing is required.

The reason for leaving the needle stuck in the patient's buttock for a moment before connecting the syringe is that its point may be in a vein, injection into which would result in embolus (see below). This precaution is not essential for soluble injections (for they do not form emboli), and is neglected with apparent impunity by many who use insoluble injections.[1] Moreover, embolism may occur in spite of the greatest care in this regard. Yet it is surely prudent to take this little precaution.

Dosage.—It will be found that any of these preparations may be given to certain patients in very high doses.

But equally good results, nay better ones, may be obtained by the ordinary doses given above, and *an apparent toleration on the part of the patient should never tempt us to excessive hypodermic medication.*[2]—I know no rule more important than this—for excessive medication (especially of the insoluble salts) by injection produces a distinctly cumulative effect.

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[1] Indeed, Berdal has seen emboli as often in spite of this precaution as when it is not employed. Of late I have omitted it with no apparent evil results.

[2] Thus I gave Case IV in eleven days three injections of salicylate, 2 c.c. to the dose, while he was taking six grains of gray powder a day; and Case V took on January 13th, 0.7 c.c. of gray oil; on the fifteenth, 1.3 c.c.; on the seventeenth, 1.6 c.c.; on the nineteenth, 1.3 c.c.; on the twenty-second, 2 c.c.; and on the twenty-fifth, 1.6. c.c.; in all, 8.5. c.c. (4.25 c.c. of pure mercury) in twelve days. This vigorous treatment was given only because the oculist in charge insisted that sight would be lost unless the lesion were immediately controlled. It was controlled, but the patient was salivated for a month thereafter. Case IV, however, suffered no ill effects from his treatment.

Yet such cases as these are exceptional and I now believe such doses both unnecessary and harmful. Even the ordinary dose of gray oil or salicylate

The mercury continues to be absorbed for many days after its injection, and I have twice (once in my own practice) seen *severe and prolonged salivation begin in the second or third week following cessation of treatment by insoluble injections,* and continue for many weeks thereafter.

The injection method is a wonderfully effective, but a dangerous, weapon. It should never be employed carelessly or in excessive doses. *If the lesion does not yield to the usual doses, the way to a cure is not* through an increase in dose or prolongation of treatment, but *by attention to hygiene and stopping all medication.* Case XXVII (page 231) is a brilliant example of the results to be expected from such a course.

Frequency of Administration.—For Routine Treatment.—*Insoluble injections* may be given, one every two weeks, for a year, or until such time thereafter as there shall have been no symptoms for six months; then one every three weeks for the second year; then skip six months; then resume at one a month for six months. Relapse of symptoms should be the signal for resumption of injections every two weeks for three months after their cure.

Soluble injections (page 153) daily or every other day.

For Symptomatic Treatment.—*Insoluble injections* may be given once or twice a week in courses of not more than three or four weeks. Then an interval of at least three weeks should elapse before treatment is resumed.

In order to control a lesion otherwise unmanageable, insoluble injections may be given until the lesion begins to yield; then, especially if they are distasteful or distressing to the patient, they may be exchanged for some other form of treatment.

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sometimes purges a patient vilely for a day or two. With injections, as with internal medication, exceptional persons can—but should not be permitted to—take incredibly large doses. Cf. Klieneberger, *Zeitschr. f. klin. Med.*, 1906, vol. lvii.

Soluble injections may be administered daily or every other day for as long as three or four weeks. When there is an emergency requiring prompt mercurialization (e. g., syphilis of the nervous system) I prefer to begin treatment with three or four daily injections of a soluble salt, and then (on the next day) begin a course of insoluble injections.

Merits of Various Injections.[1]—*Among the soluble injections*, bichlorid is generally preferred, though the benzoate is alleged to be less painful and the biniodid more efficient. I have personally given quite an extensive trial to the bichlorid succinamidate, oxycyanid, biniodid (in Hannam's and Lafay's sera), and enesol, and cannot say that I find any great difference in their efficiency, though I prefer the biniodid and iodid of potassium mixture (page 175) because I have found it usually the least painful.

Among the insoluble injections, calomel is said to be the most efficient, but is too painful. My preference lies between gray oil and salicylate, of which the former is usually less painful, the latter, perhaps,[2] more efficient.

The choice between soluble and insoluble injections is largely a matter of taste. Both have their champions for the routine as well as for the symptomatic treatment of syphilis. But since the insoluble preparations have been perfected, and since the necessity for actually injecting them into the muscle has been generally understood, the vogue of soluble injections has diminished. *None of these injections is absolutely painless* (despite what their votaries say to the contrary), and, since soluble injections have to be given every day for a

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[1] The most extensive discussions of this subject are Lévy-Bing, "Les injections mercurielles," Paris, 1902; also *La Syphilis*, 1905, vol. iii, pp. 28, 34, 41, 46; also *J. Am. Med. Ass'n.*, 1907, vol. xlix.

[2] For the past two years I have been trying in vain to determine this point. The difference in efficiency is very negligible in most cases and Lévy-Bing (arguing from scientific rather than from clinical premises) prefers the gray oil.

"course" of ten to thirty injections, the pain caused by them is cumulative, and in the end may be less tolerable than the more severe [1] but less often repeated pain from insoluble injections.

A more important distinction is this: *Insoluble injections are the more efficient*, and, since efficiency is the one claim made in favor of injections, this fact is gradually forcing insoluble injections to the fore.

On the other hand, *the action of insoluble injections is relatively slow to begin and long continued.* For this reason, as already stated, it is sometimes wise to begin with a few soluble injections to hurry on the effect of the drug while relying in the main upon the more efficient insoluble injections.

On the other hand, we have to consider the relatively grave accidents that may result from insoluble injections.

Disadvantages of Injections.—Soluble.—The disadvantages of *soluble injections* are the cumulative pain, the relative inefficiency, and the necessity for daily visits to the physician.

Insoluble.—The disadvantages of *insoluble injections* are general and local.

The *general disadvantages of insoluble injections* are poisoning and embolism.

Acute mercurial poisoning may follow within twenty-four hours of a single injection of mercury. It is characterized by cramps, explosive diarrhea, depression of spirits, even fever; it may last several days. If the usual doses are employed such an attack follows the first injection once in every thirty or forty cases. It is not prohibitive. Lessen the dose at the next injection, and thereafter return to the full dose.

Grave salivation follows any overdosage with mercury, but that following injections of insoluble mercury has two very

[1] Not always, but usually.

annoying characteristics: It is slow to appear and slow to disappear. Thus I have known two patients (as already noted) to become salivated three weeks after their last injection. And in certain cases, such as the one related on page 178, the patient has so much mercury under his skin when he becomes salivated that he keeps on absorbing this and prolongs his salivation indefinitely. The moral of which is: *Stop injections as soon as the gums are touched.*

Embolism is due to a drop of the oily injection whisking off into the general circulation. It is caused by the perforation of a vein, and is to be guarded against, as far as possible, by the maneuver already related. The embolism is almost always pulmonary; its symptoms may appear the day after injection, though they usually follow it immediately.

After a more or less marked premonitory stage of slight fever and malaise, the attack begins with a sharp pain in the side, which interferes greatly with respiration. The temperature and pulse rise; the patient is much prostrated. The physical signs are those of a circumscribed pneumonia. The whole attack lasts from a few hours to a week. Curiously enough, the injection which causes the embolism is often remarkably painless.

The frequency of embolism is estimated at about one in a thousand injections. I have had but two in my practice and my father one; this in several thousand injections. One or two fatal cases of mercurial embolism have been reported. As I have seen it, embolism is a serious complication, quite serious enough to prohibit my employing insoluble injections in all cases; yet it is a petty inconvenience in comparison with the benefits sometimes to be derived from their use.

The *local disadvantages of insoluble injections* are pain, induration, abscess, sloughing.

The *pain* varies greatly. Soluble injections are painful for one to three days; during this time they may cause agony.

Insoluble injections give no immediate pain unless a nerve is actually punctured by the needle; but in from six to twenty-four hours the spot begins to ache, and this ache increases for a day or two longer, then gradually diminishes, but may last ten days or more.

The first injection is likely to hurt more than subsequent ones, but no two of them are alike. One may set up an intense sciatica, lasting a week, and the next one be almost painless. Some persons feel the pain much more than others. Indeed, it is practically prohibitive in certain cases. Yet I shall always remember one of my first experiences with mercurial injections. The patient was a neurotic woman; she was losing the ala of her nose; she had tried every treatment except injections—inunctions, fumigations, high doses of iodid, all in vain. Small wonder that she accepted injections at any cost. Yet every one I gave her made her vomit for hours and kept her in bed a day. But six injections cured her. It was worth the price.

On the other hand, I have a number of patients who prefer injections to internal medication even for routine treatment, and, curiously enough, one of my most neurotic patients has been on injections of salicylate for the past year, and asserts that they cause no pain whatever.

Indurations may occur even from insoluble injections. I have had a patient come to me with both buttocks a solid indurated mass from injections of bichlorid. As a rule, however, even insoluble injections should leave no induration. But if the injection is not made deep enough, or if any of the injected fluid leaks back along the track of puncture a red, tender, edematous swelling in the subcutaneous tissue results. After several weeks this subsides, leaving an induration, somewhat sensitive to pressure, and over which the skin may remain for a long time bluish and glossy.

Such reactions are rare. They are apparently unavoid-

able in certain fat, soft subjects, but they can almost always be prevented by proper technic. Not one in ten of my injections cause them.

In the muscle itself the injection leaves a small induration which may be pierced by the needle at the time of a subsequent injection. A second charge may be shot into this mass without causing any untoward reaction.

Abscess and gangrene are usually due to dirt or carelessness. Neither accident has occurred in my practice; yet I believe there must have been necrosis, if not slight suppuration, in some of the indurated lumps I have caused. Such suppuration may be due to the debilitated condition of the patient.

A few cases of gangrene have been reported (Nicolsky [1]) as caused by the occlusion of an artery by the injection. Though extremely rare, such an accident could neither be foreseen nor avoided.

Advantages of the Method.—Such hideous possibilities, were they not extremely remote, would be enough to deter one from using insoluble injections. In my office practice I administer from four to a dozen injections of insoluble mercury a week, and, but for two embolisms, a few cases of salivation and an occasional sharp local reaction, I have had no accidents, and others who use the injections on thousands of cases in their clinical practice report equally good results.

Yet even such relative immunity from grave accidents would not warrant the use of this method were not its results superlatively good. They are no less. Insoluble injections will not, to be sure, cure every lesion of syphilis; but where mercury will work at all, these injections are always as efficient as any other method, and often much more so.

I have seen lesions yield to them after resisting every other

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[1] *Bull. de la soc. de derm. et syph.*, 1906, vol. xvii, p. 42.

form of treatment, even soluble injections. I have met but one exception, a palmar syphilid, that yielded better to fumigations than to injections.

Such being the case, insoluble injections are surely the method of choice for the cure of the lesions of syphilis unless they be readily curable by other means or the patient too sensitive to bear the pain of injection.

Let us state the case another way. Most lesions are eminently curable without injections, and to certain patients the pain of injections is almost, if not quite, unbearable.

In most instances, therefore, the physician may select the method of treatment he pleases; in a certain few he is driven to use injections by the failure of all else.

INUNCTION

Preparations Employed.—The *officinal blue ointment* (ung. hydrargyri) can be improved by the substitution of one of the proprietary ointment bases, such as *resorbin or vasogen.* These are less greasy and odoriferous. One part of mercury may be rubbed into one or more parts of the base, or the preparation may be obtained ready made in twenty per cent and fifty per cent strengths. Put up in dram capsules (one for each rub) they are convenient to carry about.

Another new preparation is *calomelol ointment,* white in color, put up in graduated glass tubes, two of the "marks" to be used for each rub. It is cleaner than the blue ointment, and seems to be about as efficient and somewhat less irritating.

Mercurial plasters and mercurial shirts are no longer generally employed in the constitutional treatment of syphilis, though the plasters are sometimes used for a local effect.

The Technic.—The best method is that employed at the Hot Springs. The patient takes a hot soap-and-water bath,

is well rubbed down with alcohol, and then is ready for his inunction. He sits astraddle a chair with his face toward the back, his arms folded upon it and his chin resting upon his arms. An attendant now rubs in broadly and with a vigorous circular motion of the hand over the entire back a given quantity of mercurial ointment, generally one dram at a rub. The friction continues for twenty minutes. (To prevent mercurialization the frictioner may protect his hand with a rubber glove.) The ointment thus rubbed in is left upon the back, and the patient puts on first a thin gauze undershirt (which he wears a week as a "mercurial" shirt), and over this his ordinary undershirt and customary clothing. On the following day the patient takes another hot soap-and-water bath, is thoroughly washed, rubbed with alcohol, and then takes another friction as on the previous day, resuming his mercurial shirt.

In case the patient has to do his own rubbing he cannot reach his back, and as no other part of the body is covered with skin sufficiently thick to bear daily rubs, he is obliged to shift from place to place. The hot bath, the alcohol rub, and the mercurial shirt are employed as in the above-described system, and the rubs are made in the following regions on successive days:

1. Right arm and forearm, internal aspect.
2. Left arm and forearm, internal aspect.
3. Right thigh, internal aspect.
4. Left thigh, internal aspect.
5. Right side of thorax and loin.
6. Left side of thorax and loin.
7. Abdomen.

The purpose in thus distributing the course is to make it occupy an even week. Hairy subjects may have to modify the plan.

The ointment must be rubbed into the skin until every bit

of greasiness has disappeared. This takes about twenty minutes, and is a duty the patient is inclined to shirk.

Duration of Treatment.—Inunctions are usually given daily in courses of two to four weeks. With the ordinary dose (1 dram (4 gm.) of fifty per cent blue ointment) the patient is usually slightly salivated in two or three weeks. When salivation occurs the rubs must be suspended.

Merits of Inunctions.—In efficacy inunction ranks below injection and fumigation. In convenience it may rank higher than either, in that it can be performed by the patient himself. But the dosage is uncertain, for we do not know how much mercury is absorbed by the skin (or whether the absorption is not chiefly by inhalation of the mercury that evaporates from the surface of the body [1]), and very few patients rub in the ointment thoroughly.

But the chief objection to inunction comes from the patient. The treatment is dirty and disgusting; it often irritates the skin, and may even provoke a local eczema; it is tedious in the taking.

For these reasons I find that when I ask a patient to choose between inunction and injection he almost invariably selects the latter. And unless he is oversensitive to injections I much prefer he should.

Actually I employ inunctions very rarely; only when a patient needs vigorous treatment, and cannot or will not take injections or fumigations. But many physicians employ them frequently, even for the routine treatment of the disease. The choice is largely a matter of taste.

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[1] This question has never been absolutely settled; but it would seem that with the ordinary method of inunction, fully as much mercury is absorbed by the skin as is inhaled.

FUMIGATION

Salts Employed.—The salts of mercury most suitable for fumigation are calomel and the black oxid. The former is the more popular, but I prefer the latter (which is less irritating). The dose of calomel is gr. xx (1.3 gm.), of the black oxid, ʒij to ijss (8 to 10 gm.). The fumigations are usually repeated twice a week.

The Technic.—If the fumigation cannot be taken at a bath establishment, it may be taken at home with the aid of the following apparatus: a piece of tin, 10 × 3½ inches, bent to a right angle at 3 inches (more or less) from each end; a spirit lamp or Bunsen burner; a large pan of boiling water; a chair; a couple of large blankets or a vapor-bath cabinet. The patient strips and sits on a chair surrounded by the blankets or the cabinet, with his head out. Under the chair are placed the pan of steaming water and beside it the spirit lamp covered by the bent tin, upon which the mercury is spread.

As soon as the body becomes warm and moist the spirit lamp is lighted, and the salt begins to volatilize. After fifteen minutes the heat usually becomes unbearable; the lamp may then be extinguished, but the patient should sit in the mercurial fumes ten minutes longer. Then he arises, wraps himself in a blanket, and lies down until he is cool. He should not take a bath until the following day.

Calomel fumes are often irritating to the mucous membranes, and should, therefore, not be inhaled; but if the black oxid is used, the patient may take an occasional whiff with impunity and perhaps with some benefit.

Let him also take care not to burn himself to death.

Merits of the Method.—The difficulties surrounding fumigation are many. Very few patients will spend the time and trouble to take their baths at home, and I know of no bath establishment in New York where they are properly given. Yet the

method is not painful like injection (and usually not quite so efficacious), more powerful and more cleanly than inunction. The danger of salivation is the same as with injections.

I use it quite frequently and with very satisfactory results in the most stubborn cases.

INTRAVENOUS INJECTION

Since its introduction by Bacelli this method of administering mercury has been taken up repeatedly by various investigators in the hope that, in spite of its manifest perils, it might prove peculiarly useful. But no great virtues have been found in it. The salt thus introduced into the circulation has a more immediate action,[1] but also a briefer period of activity than when injected into the muscle. But, barring accidents, intravenous injection is entirely painless. Hence it has the vices of the soluble injections, less their pain, and would be advantageous only for the production of an instant mercurial effect in a patient so sensitive as not to bear any form of intramuscular injections—a most improbable combination, and one I have not yet encountered.

Solutions Employed.—Bichlorid.—This salt, though subject to the criticism that it coagulates albumin, was employed by Bacelli and has been more used than any other.

Bacelli's formula is:

Hydrarg. bichlorid. ..... 1 gm.;
Sod. chlorid. ..... 3 gm.;
Aq. destillat. ..... 1,000 gm.

Inject 1 c.c. daily.

Other Salts.—Barthélemy and Lévy-Bing have also employed the oxycyanid (1 : 1,000) and the biniodid (1, 2, or 3 : 1,000). They prefer the last.

[1] Berdal denies this.

Technic.—Abadie, one of the few avowed partisans of this method, advises the following technic:

1. He employs a syringe whose barrel and piston are both of glass (thus assuring absolute freedom from specks of dirt) and a fine needle. These he sterilizes by boiling.

2. He employs the oxycyanid (1:1,000), injects 1 c.c. every other day for twenty days (10 injections); then, after an interval of a fortnight, resumes this twenty-day course as often as necessary.

3. "Before injecting I distend the veins at the elbow by a ligature about the arm. I choose the (most prominent) vein (at the elbow) and, after careful antisepsis, insert the fine needle gently and obliquely into the center of the vein. The entrance of the needle into the vein imparts a characteristic sensation.[1] The patient does not feel the least distress. The injection is made slowly after removal of the ligature. An antiseptic collodion dressing is applied and the patient returns to his work" (Abadie).

Complications.—The method appears to be less difficult and dangerous than one would expect. Such local inconveniences as ecchymosis, periphlebitis, nodosities, and subcutaneous edema would be pardonable were there any great advantage in the method; but this does not appear to be the case.

OTHER METHODS OF ADMINISTRATION

Intratracheal Injection.—Described by Landis.[2]

Administration per Rectum.—Andry[3] has successfully employed suppositories of gray oil, each containing gm. 0.02

[1] Marcus and Wielander (*Archiv f. Dermat. u. Syph.*, 1906, vol. lxxix, p. 213) aspirate a drop of blood to be sure they are in the vein. They give as many as thirty daily injections of bichlorid.

[2] *Gaz. des hôp.*, 1905, April.

[3] *Ann. de dermat. et syph.*, 1905, vol. vi, October.

to 0.04 of metallic mercury. He recovered mercury from the urine, found that one suppository a day for twenty-five days in the month was perfectly well tolerated for an indefinite period, and believes that the action of the mercury upon lesions is as efficient as when it is administered by the mouth, while being free from every inconvenience.

COMPARISON OF VARIOUS METHODS

In the preceding paragraphs I have endeavored to make clear my own preferences, and the reasons for them, while showing due deference to those who differ from my views. There are many excellent methods of treating syphilis, and, inasmuch as every method finds its application somewhere—inasmuch as I myself employ them all—it is often a matter of no great moment what method is employed so long as it is efficient and pleases the patient. My only plea is that internal medication be employed as much as possible for the convenience of the patient, and that hypodermics be fearlessly used in stubborn cases or in cases where an immediate effect is required (e. g., brain syphilis).

The relative merits of the various methods of administering mercury may be summed up as follows:

For Efficiency.—Insoluble injection, soluble injection, fumigation, inunction, internal medication.

For Comfort.—Internal medication (or insoluble injection or soluble injection)—then the others in different order for each case.

In the routine treatment comfort should always be considered first; in the cure of symptoms it may have to bow before efficiency.

CHAPTER XIV

THE IODIDS—MIXED TREATMENT

WHEN we speak of the iodids, we think of the iodid of potassium. This drug is worth more than all the others together. Occasionally the patient is so susceptible to its influence that some substitute has to be employed, but no effort should be spared to make the patient digest potassium iodid, so infinitely superior is it to all the other remedies claiming to be "just as good."

Therapeutic Indications.—Iodids are indicated in the treatment of:

1. Tertiary lesions.

2. Neuralgic or painful lesions, especially the pains of early secondary periostitis, arthritis, headache, etc.

3. Cases in which, on account of idiosyncrasy or severe nephritis, mercury cannot be borne.

Unfortunately, though grave toxemia from iodid is rare, mild inconveniences are extremely common. It is therefore essential to understand the toxic effects of the drug and how to combat them.

IODID POISONING (IODISM)

Iodid poisoning shows itself in many forms. Among the commoner ones, often occurring in conjunction, are:

1. The metallic taste.

2. Coryza.

3. Indigestion.
4. Acne.

Among the rarer forms may be mentioned:

5. Various skin lesions.
6. Toxemia of the nervous type.
7. Salivation and congestion of the salivary glands.
8. Neuralgia.
9. Localized edema.
10. Epistaxis, urethritis, albuminuria.

METALLIC TASTE.—The metallic taste, as though the patient had a copper cent in his mouth, is usually the first symptom of iodic poisoning. Many patients state that every dose of the drug leaves this taste in the mouth.

IODIC CORYZA.—This resembles a common cold in its milder forms; but when severe simulates the most violent hay fever; the "head is stopped up"; the eyes and nose run copiously.

A still more violent phase is the *iodic grippe*, characterized by sudden onset of the most violent naso-pharyngeal catarrh, fever, intense headache, and utter prostration.

Happily, these various phenomena do not succeed one another in order of intensity. The average patient, having reached an iodic coryza, or an "iodic hay fever," goes no further. Let the iodid be increased, and he actually passes through this, and at the higher dose shows none of the phenomena produced by lesser amounts. Yet, let the iodid be discontinued for a few weeks, and, on resuming its use, the patient has to go through precisely the same misery before reaching the higher dose at which he no longer suffers.

Iodic grippe (if it occurs at all) is usually caused by the very first doses of the drug. It subsides, as it arises, almost simultaneously (on withdrawal of the iodid), and is evidence of a peculiar sensibility.

INDIGESTION.—This is the most annoying of the commoner forms of iodic poisoning, since it cannot very well be borne for any length of time, nor can it, like the coryza, be lived down.

This indigestion may merely amount to a little abdominal discomfort and a disgust for food. If the remedy is pushed, the patient's digestion goes all to pieces; he is nauseated, has cramps and diarrhea, and the iodid has to be stopped. If it is, nevertheless, brutally pushed, the patient's digestion may be damaged for years or forever.

ACNE.—Pustular acne, usually upon the face, neck, shoulders, back, or buttocks, is extremely common. The eruption comes out in crops. The pustules may attain the proportions of little boils. Though annoying, the eruption is bearable. No treatment appears to influence it; but the pustules should be left undisturbed. The more they are pinched and squeezed the longer they last. I have sometimes thought that combining the acetate with the iodid lessened the acne outcrop.

OTHER SKIN LESIONS.—The rare skin eruptions due to iodism are *erythema*, which may be papular, commonly occurring upon the nose, cheeks, and forehead, and may become eczematoid; *papules or tubercles* (very rare); *purpura*, usually affecting the legs and occurring in the old and debilitated; *bullæ*, which usually occur on the face and extremities, and may be so umbilicated and so numerous as to suggest variola; a *pustulo-crustaceous* dermatitis (simulating syphilis), and various combinations of these varieties.

TOXEMIA.—Iodic nervous toxemia occurs under two types: In the *acute form*, due to a short course of heroic medication, the patient is overwhelmed by the drug and lies, stupidly drooling, unable to take nourishment, perhaps absolutely unconscious. Yet on stopping the drug the patient is up and about in a fortnight (see Case VI, page 20).

In the *chronic form*, due to prolonged administration of

large doses, the symptoms are commonly mistaken for those of syphilis of the nervous system with the disastrous result of encouraging the use of more and more iodid. I have seen several patients long under treatment by iodid for severe nervous lesions due to syphilis gradually become anemic, restless, nervous, despondent, tremulous, unable to eat, and apparently too weak to do any work. I have known two of them to give up work, and one of them prepare to die. One could not sign his name, so weak and unsteady was he, and yet both recovered in a few weeks after leaving off the iodid, going away from town, and resorting to general tonic measures.

It is to prevent the occurrence of such mishaps that syphilis of the nervous system is nowadays treated by intermittent courses of iodid.

SALIVATION.—Salivation due to iodid is never so severe as the true mercurial salivation, nor is it accompanied by any grave lesions of the mouth. *Enormous swelling of the salivary glands* may be caused by a single dose of iodid.

NEURALGIA.—Distressing facial neuralgia may be the first evidence of iodism. Pains elsewhere are rare.

EDEMA.—Edema, localized about the face, or a much more grave edema of the upper air passages, threatening asphyxiation, are fortunately extremely rare.

OTHER LESIONS.—*Urethritis* and *albuminuria* and *epistaxis* may be caused by iodism. They are unimportant.

METHOD OF ADMINISTRATION AND DOSAGE

The susceptibility of various patients to iodism differs widely. I have seen a single dose of iodid of potash (gr. v) cause the submaxillary glands to swell to the size of pigeon's eggs, and have record of several cases of facial edema and iodic erythema or urticaria following a similar dose. My father has recorded the experiences of two physicians, one

of whom "at one time could not touch his tongue to the moistened cork of a bottle containing an iodid-of-potash solution without feeling the effects for a day upon the mucous membrane of his nose"; while the other "experienced mild symptoms of iodism if the tincture of iodin touched his skin."

But such cases are most exceptional. *As a rule*, the metallic taste, mild snuffles, and acne may be expected at a dose of gr. x to gr. xxv (0.6–1.6 gm.) three times a day, and indigestion at double these doses. On the other hand, the patient *may* take an ounce (30 gm.) a day with no inconvenience except a strong metallic taste.

Yet there is always some doubt at first as to how well the patient will digest the iodid, and, accordingly, its administration is always surrounded with many precautions.

The One Hundred Per Cent Solution.—The preparation commonly employed is the "saturated solution" of iodid of potassium given in drop doses. But there are two terms here which may be misinterpreted: *What is a "saturated solution"? what a "drop dose"?*

If you simply prescribe "a saturated solution," the pharmacist may take you at your word and compound a mixture containing one hundred and twenty per cent (or more) of potassium iodid. This is precisely what you do not wish. *You wish a one hundred per cent solution.* To avoid error, therefore, always write as follows:

℞ Potass. iodid ..... ℥j (30 gm.);
Aquæ .....q. s. ad ℥j (ad 30 c.c.).

The Minim Drop.—In the second place, the droppers of commerce vary in size as well as shape, and none of them drop minims. Therefore, the drug must be dispensed in a *dropper bottle* (which drops minims) or accompanied by a *minim graduate*. The dropper bottle is better adapted to small doses,

the graduate to large ones. The patient will find it convenient to measure out in the morning such doses as he is to take during the day while away from home, and carry them about in small vials or in a graduated bottle.

It may be more convenient to use *tablets* (gr. v) of iodid, but the solution seems to be less poisonous.

The Time of Administration.—Iodid is better borne if administered on a relatively empty stomach. Three hours after meals (e. g., at 11 A.M., 4 and 9 P.M.) is the ideal time. But this is so inconvenient an arrangement that I have most of my patients take it on arising, half an hour before lunch, and on retiring (or a fourth dose may be inserted half an hour before dinner). But many persons can take it at any time.

The Dilution.—The iodid must always be diluted. The administration of an undissolved tablet will give most people heartburn for hours.

As far as possible,[1] one should make it a rule to dissolve no more than ʒss (2 gm.) of the salt in a single tumblerful of fluid, while even the smallest dose merits at least half a tumblerful.

The Diluent.—If the patient can digest milk, this is much the best diluent, gaseous waters are the second choice, plain water the third choice. For a continuous course, it is well to suggest an occasional change of vehicle.

Any other fluid, such as coffee or tea, may be employed if the patient prefers, and if it may be drunk in sufficient quantity to effect the desired dilution.

Prevention of Iodism.—So common are the annoyances of iodism that innumerable remedies have been devised to prevent them. These may be divided into two classes:

1. Methods of taking potassium iodid.
2. Substitutes for potassium iodid.

[1] The limit with high doses being the number of glasses of fluid a patient can drink.

Methods of Taking Potassium Iodid.—Little can be done beyond taking the drug well diluted, not during or after meals, and in a suitable medium (see above).

The taste of the drug may be disguised by syrup of orange peel (syr. aurantii cort.), or by a few drops of lemon juice. Milk, coffee, and carbonated waters also disguise the flavor.

In order to aid the digestion of iodids, ten drops of dilute hydrochloric acid after meals is often helpful. The combination of Tr. nucis vomicæ (or of tincture of belladonna or of arsenic) with each dose of iodid is well spoken of; but they have been of no use at my hands.

A favorite English prescription is:

℞ Potass. iodid.,
Sodii iodid.,
Amonii iodid., ...āā gr. cc. (āā 14 gm.);
Tr. nucis vomicæ ..... ʒij (8 gm.);
Infus. quassæ .....ad ʒiv (ad 130 gm.).

M.

S. Ten minims t.i.d., and increase arithmetically (20 t.i.d. on the morrow, then 30, etc.).

Some patients prefer this to plain iodid, yet, as a rule, it is no better.

The best way of all is to put the patient in the best possible hygienic surroundings, on a light, simple diet, give the iodid in small doses at first, and increase the dose slowly. If mild iodic acne or coryza occurs, the dose should be still further increased, and the patient will doubtless pass through it.

Iodid of potassium may be administered per rectum: ʒij–iv (8–16 gm.) in ʒvi (200 c.c.) of peptonized milk. But this method is not very efficient, and can be used for only a short time. The iodid is not suitable for hypodermic medication.

Doevenspeck [1] has used daily intravenous injections of five per cent solution (injecting 2 c.c.).

Substitutes for Potassium Iodid.—This is a weary tale. Each year brings new iodid compounds "quite as good." A few scientists report wonderful results from their use—and they die a natural death.

Sodium iodid is about half as strong as potassium iodid. It has to be dissolved in twice its weight of water, and thus forms a fifty per cent solution. Hence to get the same effect, four times as much of this solution is required as of one hundred per cent potassium iodid. And even at that it is not as efficient, though somewhat less poisonous.[2] I very rarely use it.

Ammonium iodid, calcium iodid, strontium iodid, iodid of starch, and iodoform are no longer employed.

Tincture of iodin is too feeble to be of service.

Syrup of hydriodic acid is a mild tonic. It is useless for vigorous treatment.

Sajodin is highly spoken of by Fischer and Mering,[3] Roscher,[3] and Mayer.[4] It is a colorless, tasteless powder insoluble in water. It is said to contain twenty-six per cent iodid. Dose, in powder or capsule, the same as that of potassium iodid, which it is said to equal in effect without producing any poisoning. It is therefore to be used by patients who cannot take the potassium salt. I have not tried it.

Iothion [5] is a form of iodin alleged to be absorbable by the skin, and thus sparing the stomach. Two to three c.c. are painted upon the normal skin daily.

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[1] *Therap. der Gegenwart*, 1905, vol. xlvi, No. 12.

[2] Cf. Distefano, *Riforma med.*, 1905.

[3] *Med. Klinik.*, 1906, No. 7.

[4] *Dermat. Zeitschr.*, 1906, No. 3.

[5] Lipschutz, *Arch. für Derm. u. Syph.*, 1905, vol. lxxiv, p. 265; Ravosini and Hirsch, ibid., p. 295; Wesenberg, ibid., p. 301.

Among the other modern organic compounds I have found no help. One of them looks like brick dust and is quite as active.

Iodipin and Lipiodol.—Iodipin for internal administration contains ten per cent iodin in oil; a twenty-five per cent solution is used for intramuscular injection (dose, ℥ss–i = 2–4 gm.). Lipiodol is a similar but stronger preparation; it contains forty per cent iodin. Both of these preparations may be used freely by injection. Iodipin is employed constantly at Aachen. I have not tried it fairly; but have used the lipiodol in doses of ℥ss–iss (2–6 gm.). These enormous doses of a thick oil [1] when plunged into the gluteal muscles actually cause far less pain, on the average, than any injection of mercury I know. A distinct iodic effect may be obtained by these injections, but I have yet to prove that they are as effective as potassium iodid by mouth. In case of gastric intolerance, however, they are valuable.

Dose.—What is the dose of potassium iodid? It is never twice the same. Fournier is surely wrong in asserting that less than ℥ss (2 gm.) of iodid a day is always futile, and equally wrong in the statement that "what a dose of ten grams (a day) will not achieve no higher dose will achieve." Five grains three times a day may perfectly well cure a lesion, and it is the proper dose to begin with, for one should always be on the lookout for idiosyncrasy. But if the patient can take five grains, he can take ten, and usually twenty. Accordingly, *ten to twenty grains three times a day is the average dose required.*

But to conquer the lesion it may be necessary to go higher, and if the lesion is in the nervous system, to go very rapidly higher. Where shall we stop? at a theoretic ten grams?

[1] So thick that an especially large needle (sixteen wire gauge) is required and a syringe the piston of which may be forced down by screw-pressure—otherwise one's fingers are quite bruised by the pressure required.

No, nor at twenty. Under urgent conditions the dose must be rapidly increased even in the face of severe poisoning. Case VI had to be almost killed to control the progress of her disease, and I have recently seen a tertiary ulceration of the lip, which for years had resisted every kind of mercurial and iodic medication, cured in a few weeks (by Dr. Chetwood) by the administration of a dose of iodid (℥ss a day), which gave him a most intense hay fever and closed both his eyes from facial edema.

Herein lies the great difference between the use of iodid and that of mercury. *If mercury poisons, it must be stopped at once. But if iodid poisons, it may still be pushed—in some cases it must be pushed*—for iodism is but a slight and temporary inconvenience compared with the destructive syphilitic lesions against which iodids are employed. Generally speaking, therefore, *the limit to the dose of iodids is marked by an improvement in the lesion; they must be pushed until the lesion begins to yield.*

Duration of Treatment.—For stubborn lesions, however, this doctrine may be followed too literally. Lesions of the nervous system, because of their peculiar stubbornness, are likely to be overtreated unless a time limit is set to the course. This subject is amply discussed elsewhere (pages 141, 148). Suffice it to say that a *severe course of iodid should not be continued more than three or four weeks.* Whether the lesion has yielded or not an intermission of at least two or three weeks for purely hygienic treatment should then follow, after which another short, sharp course of iodid may be begun.

How to Increase the Dose.—One usually begins with five minims of the "saturated" solution three times a day. This is increased one or two minims a day[1] until fifteen minims is reached. Thereafter the increase may be more rapid, from

[1] If one minim, ♏vi, t.i.d. the second day; ♏vii t.i.d. the third day, etc.

three to five minims a day; and after the doses reach twenty-five minims each, from five or ten minims a day. By this course it takes from three to five days to reach a ten-minim dose, and from six to twelve days to reach a twenty-minim dose.

But if there is need for haste such slow and cautious measures must be discarded. Suppose you are called to treat a case of syphilitic hemiplegia that has just occurred. It may be that low doses would cure, but you have no right to take the chance. You must begin with 10-minim doses at least four times a day, and increase 10 or 15 minims a day until 40- or 50-minim doses are reached. Here you may pause for a few days, hoping the symptoms will improve. If they do not, push on, increasing by 10-minim bounds until either the lesion yields, or the patient is utterly iodized, or the time limit of four weeks is up. Then, after an interval, a secondary course is begun, but it need rarely be made as severe as the first.

MIXED TREATMENT

Thus far I have spoken of iodid as though used alone, and as a matter of fact it is often so employed. But it is my practice *never to use iodid without mercury.* My reason for this is that, *though the iodid may be somewhat efficient alone, its efficiency is greatly enhanced by the use of mercury.* This fact I have often seen exemplified, but never more clearly than in the following instance:

Case XXVI.—A young man came to me in the summer of 1906 with the following history: In 1900 he was infected with syphilis, but the chancre was insignificant and the subsequent lesions consisted of only an occasional sore throat and mouth. After a year or so these disappeared, untreated, undiagnosed even. In 1903 he married, and promptly impregnated his wife (mother and child uninfected). A few months after his mar-

riage he consulted a physician in reference to a persistent, sluggish sore which had appeared above his left elbow three or four months before and was slowly spreading. To the patient's horror the physician diagnosed syphilis and prescribed iodid of potassium. Up to this time he had taken no medicine whatever; since then he has taken iodid continually, but never at any time a grain of mercury.

Before beginning the iodid he was some ten pounds off weight; after beginning it his lesion rapidly improved up to a certain point, then began to grow larger. To combat this the dose of iodid was increased to the limit of toleration (about ten grams a day) and the lesion again checked, but not cured. But continued taking of iodid in maximum doses soon began to tell on his digestion; he lost more weight, and another sore broke out on his right knee (in 1905).

Disgusted, he changed his physician; but his new adviser improved his *régime* only by the substitution of interrupted courses of iodid for the continuous treatment he had been receiving. Under this system he was now better, now worse; but on the whole gradually failing until he consulted me.

At that time he was thirty-four pounds off weight, so feeble he could not longer follow his occupation as a bookkeeper, and his digestion so completely gone he could not digest iodid or much of anything else. Covering his right knee was a large, active, syphilitic ulceration some four inches in diameter, on his left elbow a smaller similar ulcer, and above this several scars and partially healed ulcerations.

He was put upon a diet, instructed in hygiene, and given an injection of gray oil. This caused him so much suffering, however, that it seemed wiser to continue treatment by inunction (internal treatment being out of the question). This he continued (one week in three) for six months, taking no iodid whatever.

At first he improved rapidly, gaining ten pounds in six weeks and resuming his work. The lesions on the elbow all healed, and after three months the ulcer on the knee was reduced to one quarter its size when I first saw it. But there his improvement stopped (he lived out of town and I saw him very rarely), and in January he returned with the leg ulcer showing marked

activity. But in the six months his digestion had greatly improved. Iodid was therefore resumed and run up to one gram t.i.d. Under this the ulcer melted away, and was entirely healed in three weeks.

Here were lesions, uncontrolled by three years of iodid, improved by six months of mercury and hygiene, and cured by three weeks of iodid *after* the mercury. The moral is fourfold: viz. (1) without any treatment the disease was apparently cured for three years (and a healthy child begotten); (2) the iodid of itself had some effect in controlling symptoms; but (3) this effect was greatly enhanced by the administration of mercury, though (4) the mercury alone could not effect a cure.

This treatment of syphilis by mercury and iodid together is called mixed treatment. The two are ordinarily given together in the following formula:

℞ Hydrarg. chlorid. corrosiv., gr. j–ij (0.6–1.2 gm.);
Potass. iodid. ..... ʒij–ʒj (8–30 gm.);
Syr. sarsaparillæ comp. . . ad ℥iv (ad 130 c.c.).
M.
S. Teaspoonful two hours after meals.

The dose may be and usually is given immediately after eating, but it is better digested if given two hours later.

In the above formula the *mercury biniodid* may be substituted for the bichlorid, in half the dose. *Sodium iodid* in twice the dose may replace the potassium salt.

If the patient refuses to take a fluid medicine he may take his iodid as a tablet half an hour before meals and the mercury pill or tablet after meals. The mixed treatment pills or tablet put up by various drug houses have not given me much satisfaction.

But when the gravity of the lesion demands hasty or ener-

getic treatment, or when, as in the case just recited, the condition of the patient's digestion prohibits internal treatment, the mercury must be given by injection, fumigation, or inunction, the iodid by saturated solution or injection (iodipin, lipiodol).

THE TEST COURSE

We shall often have occasion to speak of a "test course," administered more or less tentatively to prove whether a lesion is or is not syphilitic. It were ideal to have a definite course upon which one could invariably depend for a verdict; but no two lesions yield to precisely the same treatment, and, consequently, it is impracticable if not impossible to apply the same test to every case.

The general rule for a test course is this: *Give the treatment as though the lesion were known to be syphilitic and you were trying to cure it.*

Thus for a suspected secondary lesion give mercury; for a dubious or tertiary lesion, mixed treatment.

In any case the treatment should begin mildly; but in case it does no good should be rapidly pushed to the point of salivation or iodism, or both, in order to reach a diagnosis as soon as possible.

It is, therefore, usually preferable to employ one of the more vigorous methods of mercurial treatment; i. e., injection, fumigation, or inunction, and to give the iodid in drop doses by the increasing scale (page 201). Such a course, lasting three weeks, should be ample for diagnosis.

CHAPTER XV

CHANCROID

Custom in America has adopted the name "chancroid" (originated by Clerc) for that form of contagious venereal ulcer which is not accompanied by constitutional syphilitic infection. Chancroid (or soft chancre) and hard chancre are no more akin than measles and leprosy; and it is unfortunate that the ancient confusion of the local and the general infection has left us this legacy of misleading terms; but it is now too late to change them.

Definition.—*Chancroid is a specific, local, contagious, autoinfectious venereal ulcer.*

Fig. 4.—Streptobacillus of Ducrey (highly magnified). (J. L. Davis.)

It is specific in that it is caused by a specific microörganism, the streptobacillus of Ducrey [1] (Fig. 4).

[1] *Riforma medica*, 1889, vol. v, p. 98.

This bacillus is dumb-bell shaped, thick and rounded or square at the ends, constricted in the middle. It varies in length from 1.5 to 2 μ. It groups in parallel chains, and occurs

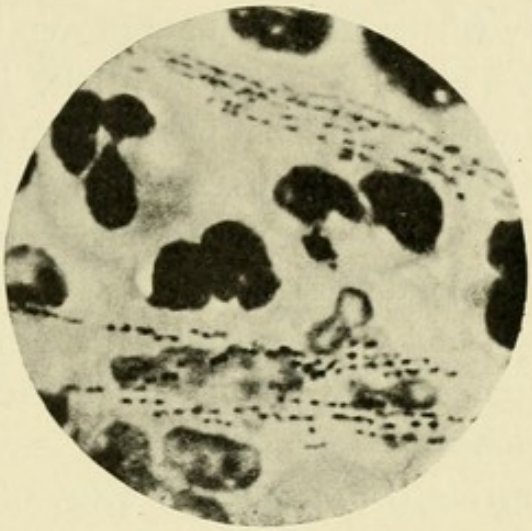

FIG. 5.—STREPTOBACILLUS, SHOWING CHAIN FORMATION. (J. L. Davis.)

both inside and outside the cells (Fig. 5). It stains readily with the ordinary dyes (methyl-blue, or violet, or fuchsin), and is decolorized by the Gram stain.

For many years after its discovery in 1889, in spite of the confirmatory observations of Unna, Kretling, Dubreuilh, and Lasnet, etc., and in face of the manifestly specific character of chancroid, the pathogenic action of the streptobacillus was doubted until proven by the culture and inoculation experiments of Istamanoff and Askpianz,[1] Lincoln Davis,[2] Lancret,[3] and Tomasczewski.[4] But chancroid is peculiarly liable to mixed infection. A smear taken from the surface of the ulcer usually shows numerous pyogenic and other bacteria, and few if any of the pathogenic bacilli. Hence such a smear cannot be depended upon for diagnosis.

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[1] *Jahresbericht d. Path.—Microörg.*, 1898, vol. xiv.
[2] *J. of Med. Research*, 1904, vol. ix, p. 401.
[3] *Bull. med.*, 1898, vol. xii, p. 1051.
[4] *Zeitschr. f. Hygiene u. Infect.*, 1903, vol. xiii, p. 327.

Chancroid is a local lesion, causing no systemic infection or reaction. *It is,* therefore, indefinitely *autoinoculable.* Indeed, the marked tendency to autoinoculation is one of the most striking clinical characteristics of the ulcer. That *it confers no immunity* whatever was amply proven by the disciples of syphilization. Lindemann, for example, inoculated himself 2,700 times with chancroidal pus in the vain hope of immunizing himself against syphilis. But a local and temporary immunity does exist; for after many inoculations a given region becomes immune, though the virus will still take on other parts of the body, and after several months' respite the first region loses its immunity. Clinically, however, such immunity has no significance.

Finally, *chancroid is a contagious venereal ulcer.* Of the three distinct venereal diseases, gonorrhea, chancroid, and syphilis, gonorrhea is, strictly speaking, the most venereal, being scarcely ever acquired except in sexual intercourse. Chancroid, though probably more virulent, is less venereal, since it recognizes many methods of infection other than sexual congress; while syphilis is the least virulent (the least easily acquired) and the least venereal.

Chancroid is an affection perpetuated only by contagion; but sexual intercourse is not essential. Wherever upon the human body a chancroid is found, there, it may be positively affirmed, pus from some other chancroid has been deposited under conditions favorable for its absorption. No amount of sexual excess, no degree of uncleanliness, no irritation, traumatic or chemical, however prolonged, no simple or infected ulceration, nothing, in short, can produce chancroid except chancroid.

Frequency.—In the clinic chancroids outnumber true chancres three or four to one. In private practice the preponderance is reversed, and we see five, or even ten, chancres to one chancroid. The reason for this is twofold. In the

first place, chancroid can never make headway among cleanly persons, for it is so foul and disgusting that no decent citizen infected with it would attempt sexual intercourse until it is cured, and half a cure—such as so commonly conceals the infectiousness of syphilis and gonorrhea—is here impossible. In the second place, in most instances, a little soap and water at the time of exposure is an absolute safeguard against it; so that it flourishes only where soap and water are not esteemed.

Methods of Contagion.—Not once in a thousand cases does one see a chancroid except about the genitals. This is as much as to say that it is practically always acquired by sexual contact. Although many cases of mediate contagion have been reported (the virus being usually conveyed by the hand), such cases are proportionately extremely rare. Indeed, chancroid will not "take" upon the integument unless it is abraded. Cullerier's experiments show that this must oftentimes be true of the vagina as well. In two cases he deposited pus from a chancroidal bubo in a clean vagina, and let it remain there between half an hour and an hour. Then some of the vaginal secretion was collected and inoculated upon the thigh, after which the vagina was thoroughly cleansed. In both instances typical chancroids developed on the thigh while the vagina remained clean.

Hence, be it noted, a woman may convey chancroid from one man to another without herself becoming infected.

Yet autoinoculation of chancroid upon the healthy skin or mucous membrane lying in contact with it is extremely common. But it takes many hours of intimate contact to effect the inoculation.

Situation and Number.—Chancroids upon the male genitals are most common in the coronary sulcus, especially in the little pocket on each side of the frenum. Persons with long foreskins may be inoculated anywhere within the preputial cavity, though urethral chancroid is extremely rare.

In women the sores usually occur about the introitus, rarely higher up.

From these regions the ulcers may spread by contact or by direct extension over external genitals, anus, thighs, abdomen, and even farther afield.

Chancroid commonly begins as a single ulcer; but no sooner has this appeared than secondary ulcerations begin, so

FIG. 6.—CHANCROIDS OF PREPUCE, PREPUTIAL FRENUM, AND GLANS PENIS, IN VARIOUS STAGES OF DEVELOPMENT. (Kaposi.)

that by the time the physician is consulted several sores are usually present.

SYMPTOMS AND COURSE

Incubation.—While Bumstead affirmed that chancroid has no period of inoculation in the sense that the pathological

process begins from the moment of infection, it is, nevertheless, true that, clinically, the disease has an incubation period varying from one to ten days, usually three to five. When the incubation is protracted, this is probably due to the fact that

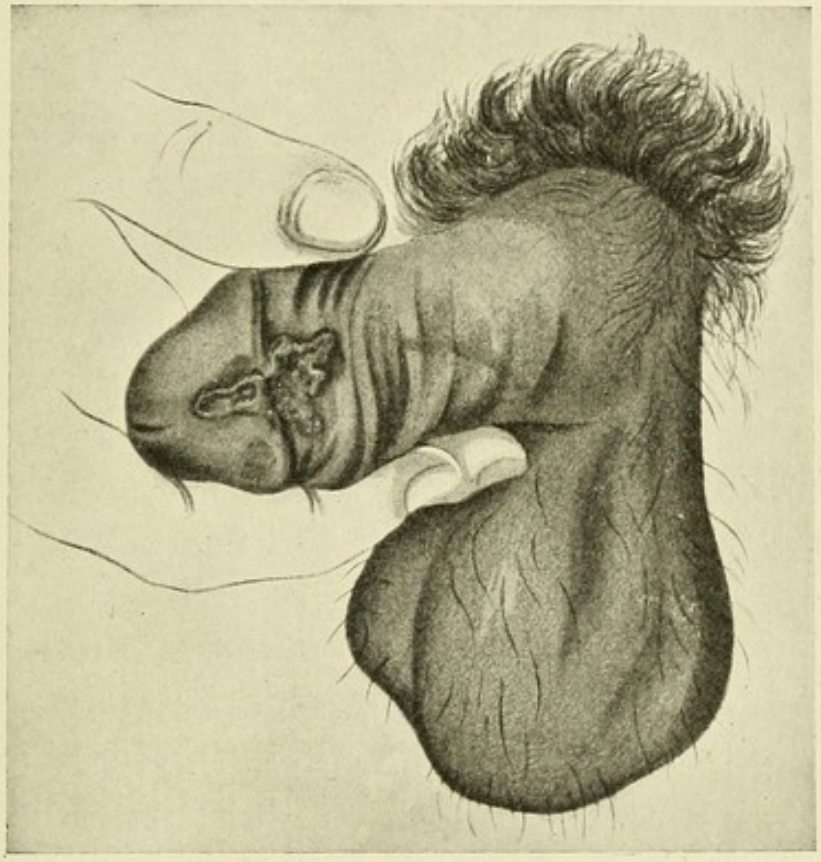

FIG. 7.—CHANCROID THAT HAS DESTROYED THE FRENUM. (Kaposi.)

the virus is retained some days within the prepuce (or vagina) before inoculation occurs.

Onset.—The symptoms of chancroid are best observed by studying the course of the artificial ulcer produced by inoculation. Within twenty-four hours after such an inoculation a reddish blush surrounds the puncture. This soon changes to an inflamed areola which, on the third day (rarely sooner or later), becomes a pustule. This extends quite rapidly, and within a few days breaks and becomes a characteristic chancroidal ulcer (Figs. 6, 7, 8, and 13).

The Ulcer.—The typical chancroid is a round ulcer with abrupt, perpendicular edge, as though it had been cut by a sharp punch. This edge may be undermined or everted. The ulcer is usually rather deep. Its base is irregular, grayish yellow, and covered by a pultaceous false membrane. It is surrounded by a non-indurated, inflammatory areola. The secretion is abundant and purulent. It bleeds readily. Though usually painless, when very active or phagedenic it may be intensely painful.

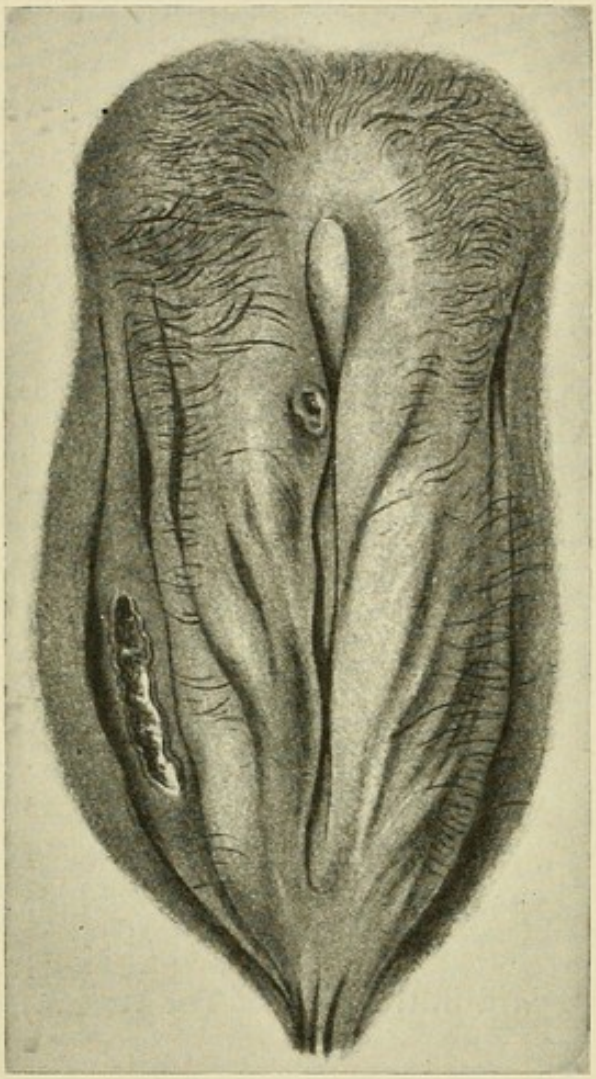

FIG. 8.—CHANCROIDS OF VULVA. (Kaposi.)

Course.—If untreated and uncomplicated the ulcer increases in size for a week or two. Then, having attained a diameter of about 1 cm., it remains stationary for about two weeks, and then gradually heals by cicatrization from the edges toward the center. So rarely, however, is the ulcer both uncomplicated and untreated that the stage of spontaneous healing is not seen in the clinic.

Complications.—The common complications of chancroid are mixed infection with syphilis (mixed sore), mixed infection with pyogenic microbes (inflamed chancroid), destruction of the frenum, phimosis, gangrene and phagedena,

balano-posthitis and venereal warts, lymphangitis, adenitis (bubo).

THE MIXED SORE.—*So long as your patient has chancroid you may never be sure that he has not chancre.* This rule is without exception. Its details and explanation, the necessary insistence upon the fact that a chancroid may readily conceal a chancre from the most experienced eye, as well as that other often-forgotten fact that, when the chancroid refuses to heal, it may be because of complicating *gumma*—these matters are discussed elsewhere (page 246).

THE INFLAMED CHANCROID.—While every chancroid is more or less irritated by its own secretions and contaminated by ordinary pyogenic microbes, the resulting inflammation is often insignificant unless due to friction, e. g., when the sore is on the edge of a long prepuce and subject to friction by the patient's clothes, or unless there is phimosis.

The chancroid at the preputial orifice is usually an interminable while getting well. The friction of shirt and drawers, or even the rubbing of the softest dressing, so irritates the sore that, though it may lose all its chancroidal characteristics and become a simple, sluggish abrasion, it lingers on week after week, taxing to the utmost the victim's patience and the physician's skill.

Chancroid underneath a long prepuce, however loose, usually excites so acute a cellulitis in the connective tissue of the foreskin as to cause *inflammatory phimosis.* This retains the secretions of the chancroid which, bathed in this irritating pus and protected from effective treatment, promptly invades both glans and prepuce, and instead of healing tends rather to eat its way through the glans penis into the urethra or through the foreskin, at the same time setting up an intense inflammation, which may terminate in abscess, erysipelas, or gangrene. If the prepuce is retracted in a desperate effort to get at the suppurating cavity, paraphimosis complicates matters.

Destruction of the Frenum.—One of the commonest complications of chancroid is destruction of the preputial frenum. Indeed, the scar of this lesion may in after years prove most valuable in distinguishing chancroid from true chancre.

The frenum is destroyed in the following manner: a chancroid appears in the sulcus at one or both sides of the frenum; as it enlarges it eats a hole in the frenum, leaving a narrow string, which soon gives way as well.

The more widespread destruction of tissue that results from inflammation or phagedena requires no special mention.

Gangrene and Phagedena.—The terrible phagedena which, until the era of antisepsis, was an imminent possibility for every case of chancroids, has almost passed to oblivion with hospital gangrene and such ancient horrors. One can no longer imagine such a case as Ricord treated for several years, and which, commencing as a chancroidal bubo fourteen years before, was still an open ulcer at the knee.

On the other hand, *gangrène foudroyante* is still occasionally encountered as the complication of stricture, or of chancroid. Thus Martin [1] relates and depicts the case of a man who lost two thirds of his penis by gangrene in thirty-six hours. Happily, such cases are now extremely rare.

Lymphangitis.—Acute inflammation of the lymphatics running along the dorsum and sides of the penis toward the inguinal glands is a rare complication. Suppuration therein is much rarer.

Inguinal Adenitis.—Chancroid of the genitals causes inguinal adenitis or bubo. This complication occurs about once in every three cases. The bubo when bilateral is usually more severe on the side upon which the sore lies; but, on account of an abnormal lymphatic supply, the glands in the

[1] Morrow's "System," p. 875.

opposite groin may be the more inflamed. This is the so-called "crossed bubo."

The chancroidal bubo follows one of three clinical types.

It may be a simple inflammatory adenitis, the glands becoming large and quite tender, so remaining for a week or more, and then slowly resolving.

It may go on to periadenitis. The glands become matted together in irregular, tender masses adherent to the skin and to the subjacent tissues. When the inflammation reaches this stage it often terminates by suppuration; and when the abscess is incised or breaks, it heals in a way no different from suppurations elsewhere.

Finally, the inflammation may go through the same stages of adenitis, periadenitis, and suppuration; but when opened it forms an ulcer which soon assumes the aspect of a chancroid, the pus from which is autoinoculable. This is the so-called *virulent bubo.*

The obvious explanation of these variations in virulence is that the milder forms of bubo are due to the absorption of pyogenic microbes, and that virulent bubo is due to the absorption of the specific bacillus. But it has frequently been observed that, whereas the pus obtained from buboes at the moment of incision is very rarely chancroidal, it becomes so a few days afterwards.[1] Hence, it has been supposed that such buboes are infected secondarily with the chancroidal bacillus.

Recent bacteriological investigations have upset this theory. Tomasczewski[2] found that, although the microscope

[1] Thus Dubreuilh (*J. de med. de Bordeaux*, 1893, vol. xxiii, p. 573) has reported an epidemic of chancroids producing 136 buboes. Of these, 27 did not suppurate; 43 were incised; the pus was sterile to culture and they healed rapidly; 51 became virulent, though sterile when incised; only 3 were virulent at the time of incision; and 12, which were open on admission to the hospital, were all virulent.

[2] *Arch. f. Derm. u. Syph.*, 1904, vol. lxxi, p. 1

showed nothing in the pus from a freshly incised chancroidal bubo, culture on blood agar revealed streptobacilli of very slight virulence, while in older lesions the same bacteria were found with renewed virulence.

Now it has long been known that chancroidal pus, although it would remain virulent for many weeks if kept in a sealed tube, promptly lost its virulence when heated to a temperature of 105° F. It is, therefore, to be inferred that both "simple" and "virulent" buboes are due to infection by the streptobacillus, but that the heat engendered in the inflamed gland is sufficient to reduce its virulence sometimes to the point of rendering it temporarily inocuous; so that it only requires a few days of cooling off, as it were, in the open ulceration to regain its primitive vigor.

DIAGNOSIS

The diagnosis of chancroid may be made by the microscope (which gives no negative assurance), by culture on blood agar, by the ancient and very trustworthy method of autoinoculation, or by confrontation (examination of the person from whom the sore was contracted).

To perform *autoinoculation*, cleanse a spot on the outer side of the patient's thigh with alcohol; then with a clean bistoury or pin wipe a little pus from the suspected sore; twirl the point of the instrument into the skin at the point to be inoculated just deep enough to draw the most minute drop of blood, smear the spot well with the virus, and clap on a vaccination shield. A "take" is announced by the appearance of a typical chancroid on the third day. This should promptly be destroyed by cauterization (see below).

The mere appearance of a pustule or an ulcer after inoculation proves nothing. It must be chancroidal in type to be considered trustworthy evidence. If, after inoculation, there

is still some doubt of the nature of the "take," *its secretions may be examined for the Ducrey bacillus with better prospect of success than in the original sore*, or the diagnosis may be confirmed by heteroinoculation (inoculation of another person). Gangrenous chancroid is said not to be inoculable.

Generally speaking, however, no test is required to establish the diagnosis of chancroid. The multiple, virulent sloughing ulcers, spreading by contact inoculation, the characteristic bubo, and the history of very recent exposure form a typical clinical picture. Inflamed herpes or simple ulcer may, however, very closely simulate beginning chancroid. Moreover, the chancroid may originate in the orifice of a sebaceous gland of the scrotum or penis (follicular chancroid), and so be mistaken at first for a small boil or an acne pustule. Bullous and ecthymatous forms of chancroid are extremely rare.

But the really important point in the diagnosis of chancroid is its differentiation from true syphilitic chancre. The details of this differentiation are considered elsewhere (page 246); but it is not amiss to repeat once again that *no matter how sure you may be that a given sore is a chancroid, you cannot thereby make a negative diagnosis of syphilis*; for the same coitus that transmitted the chancroid may have transmitted syphilis, the chancre of which might not develop until after the chancroid was cured, or might develop on the chancroid, and be so insignificant as to escape the most careful scrutiny.

TREATMENT

Abortive Treatment.—Any chancroid not more than three days old may be cured by adequate cauterization. Chancroids from three to seven days old may often be thus aborted; but if the sore is more than a week old success is so rare that

cauterization should not be employed; for, if it fails, it leaves the sore larger than ever.

It is an absolute condition to success that *every* sore be cauterized; not only those sores that have already developed, but also those that are only just beginning as minute pustules, or ulcers, but which, however small, can recontaminate their larger brethren, however well these be cauterized. Hence, nothing more futile could be imagined than cauterization of sores on the outside of the frenum so long as an inflammatory phimosis prevents access to those within the preputial cavity, or cauterization of balanitic chancroids in the presence of inaccessible urethral sores.

The simplest *method of cauterization* is the following:

1. Wash the ulcer and surrounding region with peroxid of hydrogen and water, equal parts; then dry.

2. Anoint the immediate neighborhood of the ulcer with vaselin to keep it from contact with the acids (not absolutely essential).

3. Cut several narrow strips of blotting paper. With one of these dry the surface of the ulcer.

4. Dip the pointed end of a glass rod into pure carbolic acid and apply it over the ulcerated surface. If there is any excess of acid running over the edge of the sore, quickly catch this with one of the strips of blotting paper. If there are several sores, carbolize them all before proceeding further.

The object of this step is *not cauterization, but anesthesia.* Carbolic acid will not destroy the bacteria in the tissues, but will, after the first sting, anesthetize the sore. If the patient is extremely sensitive and nervous, a ten per cent cocain solution may be used instead of the carbolic acid. Its action is much slower and no more effective.

5. After the whole surface of the ulcer has been fully whitened by the carbolic acid, dry again with blotting paper

and apply, very cautiously, with another glass rod pure nitric acid.[1] Apply this in droplets until the whole surface is stained yellow or brown.

6. Wash again with peroxid and water and cover with calomel or nosophen.

Iodoform is by far the best dressing for chancroid, but its odor and reputation are prohibitive.

If the first cauterization fails, it is futile to repeat it.

Palliative Treatment.—Since every uncomplicated chancroid tends to get well spontaneously, it may be treated expectantly, if cauterization is prohibited by the age of the lesion or the refusal of the patient.

The lesion must be kept mechanically clean and covered with some antiseptic. To do this, wash twice a day with peroxid and water and keep it dusted with calomel[2] (iodoform is still the best, but the most impossible application, and nosophen is the only one of its many "substitutes" that has given me any satisfaction). Never put grease on a chancroid. An occasional application of silver-stick or of pure carbolic acid stimulates the granulations.

If the chancroid lies within the preputial cavity, cover it solely with the powder. If it lies outside, protect it from friction by a large pad of cotton held in place by a bandage or a bag.

Prevention of Bubo.—Warn the patient to avoid all violent exercise, and to walk about as little as possible in the hope of preventing suppurating bubo. Watch the groins carefully, and with the first sign of periadenitis (matting together of the glands) clap him into bed with a hot-water bag on his groin.

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[1] The actual cautery is preferred by some, but is too terrifying a sight for most patients.

[2] Many efforts have been made to diminish the virulence of the bacteria by external applications of heat but without any great success.

Do not, under any circumstances, paint the groin with iodin. It does no good and irritates the skin; so that, if the bubo does eventually suppurate and burst, the surrounding skin is ready for inoculation.

Treatment of Complications.—*Cellulitis and suppuration* call for wet dressings, rest in bed, elevation of the penis, and incision, *secundum artem.*

Phimosis, whether congenital or inflammatory, is the most annoying complication of chancroid. A chancroid under a tight foreskin, unless it can be aborted by early cauterization, demands prompt liberating incision.

Incision of Foreskin.—This little operation is performed under infiltration anesthesia, particular care being taken to anesthetize the mucous membrane. A grooved director is then inserted under the foreskin (not in the urethra, if you please), and upon this the skin and mucous membrane are split with scissors [1] or knife from the free margin well back to the corona.

It is better practice to make two such incisions, one on each side of the penis, as recommended by Taylor, rather than to make the single dorsal incision, which does not thoroughly expose the pockets on each side of the frenum.

Do not suture, but protect the incision by 1 : 5,000 bichlorid wet dressings in the hope of preventing chancroidal inoculation of the wound.

After the chancroids have healed a secondary circumcision is usually required.

If the patient refuses operation, the best that can be done for him is to inject the preputial cavity with ten per cent argyrol solutions frequently—or, better still, refuse to treat him at all; for it is quite impossible to foretell what complications may ensue, and no physician can afford, even occasion-

[1] Bandage shears are the best.

ally, to be responsible for a pathological amputation of the glans penis.

Above all things, do not pull back a tight prepuce. The *paraphimosis* which will probably result is not easy to reduce, and is the most fertile cause of gangrene.

Phagedena demands general anesthesia and extensive cauterization of every recess with the actual cautery, followed by wet dressings and iodoform. Do not fear to cauterize too much.

Delayed healing is the commonest and most annoying complication of chancroid outside the preputial cavity. To encourage healing, protect the sore with a voluminous dressing of cotton, and cauterize it once a week with nitrate of silver, applying in the meanwhile some stimulating lotion, such as red or black wash, or boric-acid ointment.

Partial erosion of the frenum forms a pocket which is very hard to clean. Tie a thread tightly around the remaining band, and it will cut through within forty-eight hours.

Suppurating bubo should be drained by very small incisions, almost punctures, multiple if need be, followed by injection of a ten per cent iodoform-in-glycerin mixture. Then apply wet dressings (or iodoform) topped by a hot-water bag. If after several weeks the indurated masses of periadenitis show no tendency to decline they must be excised. Usually the mass softens immediately and leaves a large chancroidal ulcer. The treatment of this is detailed above.

Excision of the Inguinal Lymph Nodes.—This, though a minor surgical operation, is a long and tedious one, and usually leaves a wound that takes several months to heal.

The one important precaution in operating is to avoid the femoral vessels around which (at the saphenous opening) the nodes are usually clustered.

Further detail need not be attempted here.

CHAPTER XVI

THE INITIAL LESION

THE *initial lesion* (primary lesion) *of syphilis consists of the chancre and the adjacent adenitis.* It must not be forgotten that the inflamed lymph nodes form as essential and characteristic a part of the initial lesion as does the chancre itself.

THE CHANCRE

Synonyms.—Hard chancre, Hunterian chancre, indurated chancre, syphilitic chancre, primary sore.

Description.—The chancre is an eroded or ulcerated, painless neoplasm, arising at the site of syphilitic inoculation.

The chancre is primarily a neoplasm. By bearing this in mind we distinguish it instinctively from chancroid, which is primarily an ulcer. The one is a lump, the other a hole.

This neoplasm is commonly called the *induration.* This induration may be very extensive; it may form a large hard lump, projecting markedly above the surrounding tissues and having a diameter of perhaps an inch. But usually (in eight cases out of ten) it is small—one might almost say minute—and instead of projecting above the integument it is embedded in it. Thus, it may be felt rather than seen, and in appearance is rather insignificant than impressive.

The surface of this insignificant neoplasm is almost always eroded and moist, but it may be ulcerated, or it may be covered by an unbroken reddened integument.

Its *consistency* is *peculiarly hard and elastic*, as though a piece of cardboard were embedded in the integument. To appreciate this one must pick it up from the surrounding tissues and palpate it from side to side.

Morbid Anatomy.—The chancre has the general characteristics of syphiloma. So brilliant a pathologist as Virchow said "chancre is but a gummatous ulcer." Accordingly, we find in a connective-tissue framework a mass of plasma cells, leukocytes, deformed epithelial cells—all the elements of an acute localized exudative inflammation. The vessels, especially the arteries, are infiltrated, irregularly thickened, and occluded. The surface of growth is more or less necrotic, whence the erosion or ulceration. Proper staining shows an occasional spirocheta in the substance of the chancre.

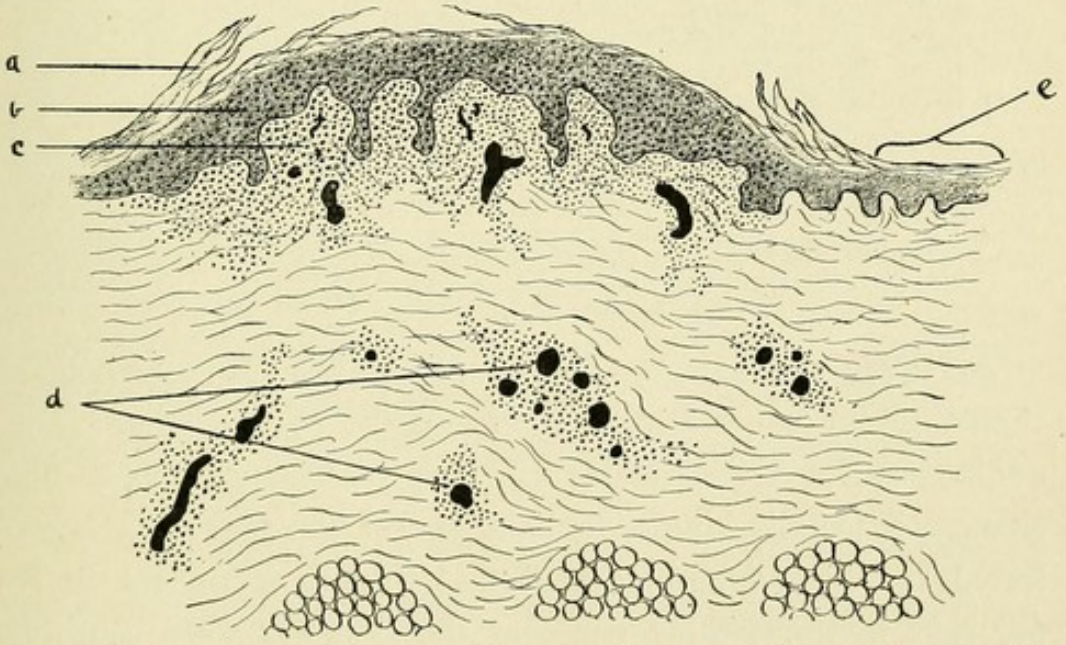

FIG. 9.—PATHOLOGY OF ERODED CHANCRE (diagrammatic). *a*, Desquamating epithelium (erosion). *b*, *c*, Infiltration of epidermis and derma (induration). *d*, Perivascular exudate at a distance from the chancre. *e*, Normal skin.

This inflammation diminishes insensibly toward the border of the induration and extends into the surrounding tissue far beyond the apparent limits of the growth. The infiltrations in the vessel walls, in particular, extend beyond the pal-

pable seat of the disease. Yet the depth of the chancre is almost nil. It occupies chiefly the epidermal layer of the skin, encroaching but little on the true derma and the subjacent tissue (Figs. 9, 10).

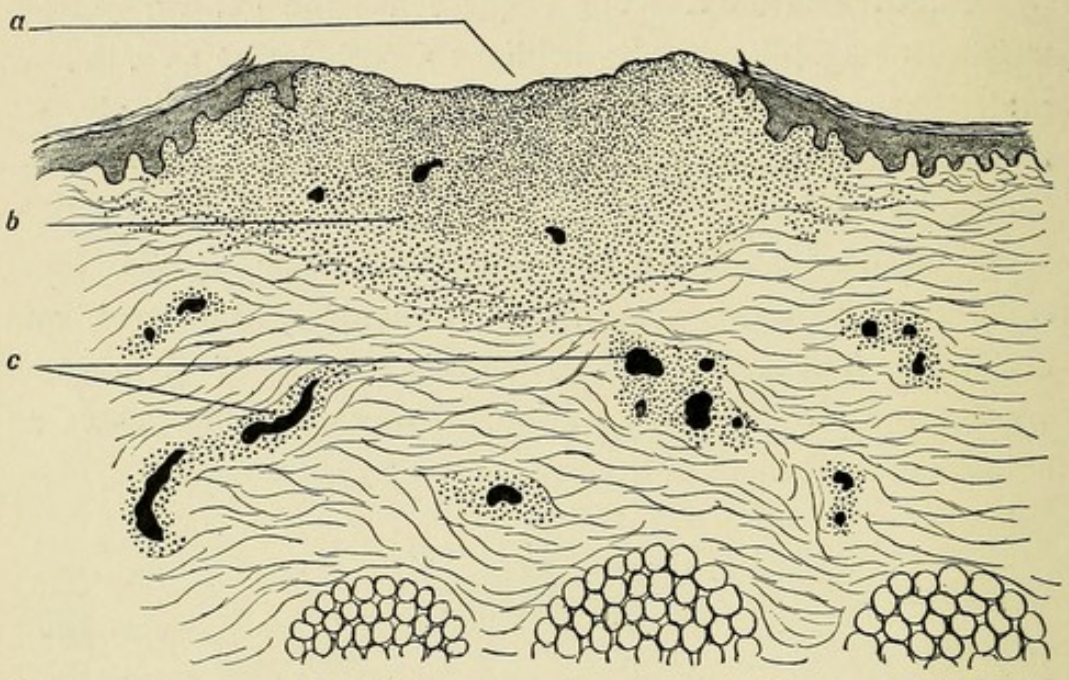

FIG. 10.—PATHOLOGY OF ULCERATED CHANCRE OR ULCERATED SYPHILITIC TUBERCLE (diagrammatic). The induration (*a*, *b*) is deep set. The ulcer (*a*) has destroyed the skin and will leave a scar. The infiltration follows the vessels (*c*) beyond the lesion.

Multiple Chancres.—The most striking characteristic of chancre is its insignificance; next in order of importance is its uniqueness. Yet too much stress may be laid upon this. The chancre is usually single, to be sure, yet Papagaey,[1] who collected 14,004 reported cases, found that in from twenty-five per cent to thirty-three per cent the chancres were multiple. This confirms other Continental statistics; yet multiple chancres are certainly much fewer in my practice. My records show only 56 among 549 cases examined, i. e., one in ten.[2]

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[1] *La syphilis*, 1906, vol. iv, p. 64.

[2] Other American authors agree that multiple chancres are relatively infrequent with us.

But whether one in ten or one in three, the multiple chancre must be counted with, and *it is a grave clinical error to insist on the uniqueness of chancre as a diagnostic factor.*

The *number* of multiple chancres is 2 in seventy-eight per cent of cases (Papagaey). My father has recorded 1 case of 11 and 1 of 12 chancres (4 on the left breast, 8 on the right). Fournier has seen a patient with 23 (7 on the left breast, 16 on the right).

The *location* of multiple chancres is almost exclusively genital. Only two per cent of extragenital chancres are multiple (Fournier). Chancres of the breast are quite frequently multiple. I have seen one patient with a chancre on each lip, another with one on the tongue and one on the lower lip.

The existence of multiple chancres brings up the question, When does syphilitic immunity begin? Is reinoculation possible?

Although in many instances the several inoculations are indubitably simultaneous, in others they doubtless succeed one another, perhaps after an interval of several days. Indeed, Queyrat [1] has apparently proven that *it is sometimes possible to autoinoculate chancre* if the inoculation is performed before the lesion is ten days old, and experimental inoculation bears this out.

Yet, whether this be proven or not, clinically speaking chancre is not autoinoculable. When the sore appears the whole man is poisoned and cannot be reinfected.

Types of Chancre.—The three chief types of chancre are:

1. The eroded chancre (Fig. 9).
2. The ulcerated chancre (Figs. 10, 11).
3. The indurated papule (Fig. 12).

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[1] *Bull. de la soc. Franc., de derm. et de syph.*, 1906, vol. xvii, p. 66.

THE ERODED CHANCRE.—From sixty to eighty per cent of chancres assume this form. It is most characteristically exemplified by chancres within the preputial cavity.

The *induration* is rounded, *circumscribed*, and *thin*, sometimes so thin as to be scarcely perceptible except to the most delicate touch (parchment chancre).

Its *color* is usually a dark, vinous, or "raw-meat" red. Rarely it is of a dusty gray color (the color of lard). It may be covered with little petechiæ.

Its *surface* is usually flat. It may be a little elevated above the surrounding integument, or a trifle sunken below it, or surrounded by a slightly elevated ring of induration.

The eroded surface is smooth and polished. It emits a *slight sero-purulent discharge.* It may be covered by a crust or a false membrane (from infection by skin cocci).

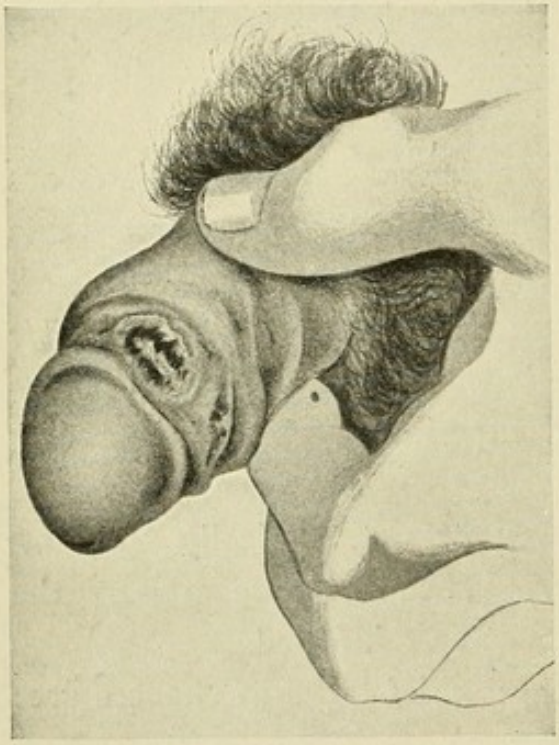

FIG. 11.—LARGE ULCERATED HUNTERIAN CHANCRE. (Kaposi.)

THE ULCERATED CHANCRE.—This is the type of chancre described by Hunter, and to it the title "Hunterian chancre" is, therefore, peculiarly applicable. It is far less common than the eroded chancre.

It has a relatively large indurated base topped by a distinct ulcer. The ulcer is due to extensive necrosis, and the necrosis is proportional to the interference with circulation; thus the thinner induration forms an eroded chancre, while the more nodular mass ulcerates. Exceptionally, and doubtless on ac-

count of some unusual surface infection, the parchment chancre ulcerates deeply.

The ulcer extends into the true derma. Its edges are sloping (not undermined) and give the sore a sort of funnel shape; the base is granulating and may be covered by a false membrane, the discharge is slight and sero-sanguinolent.

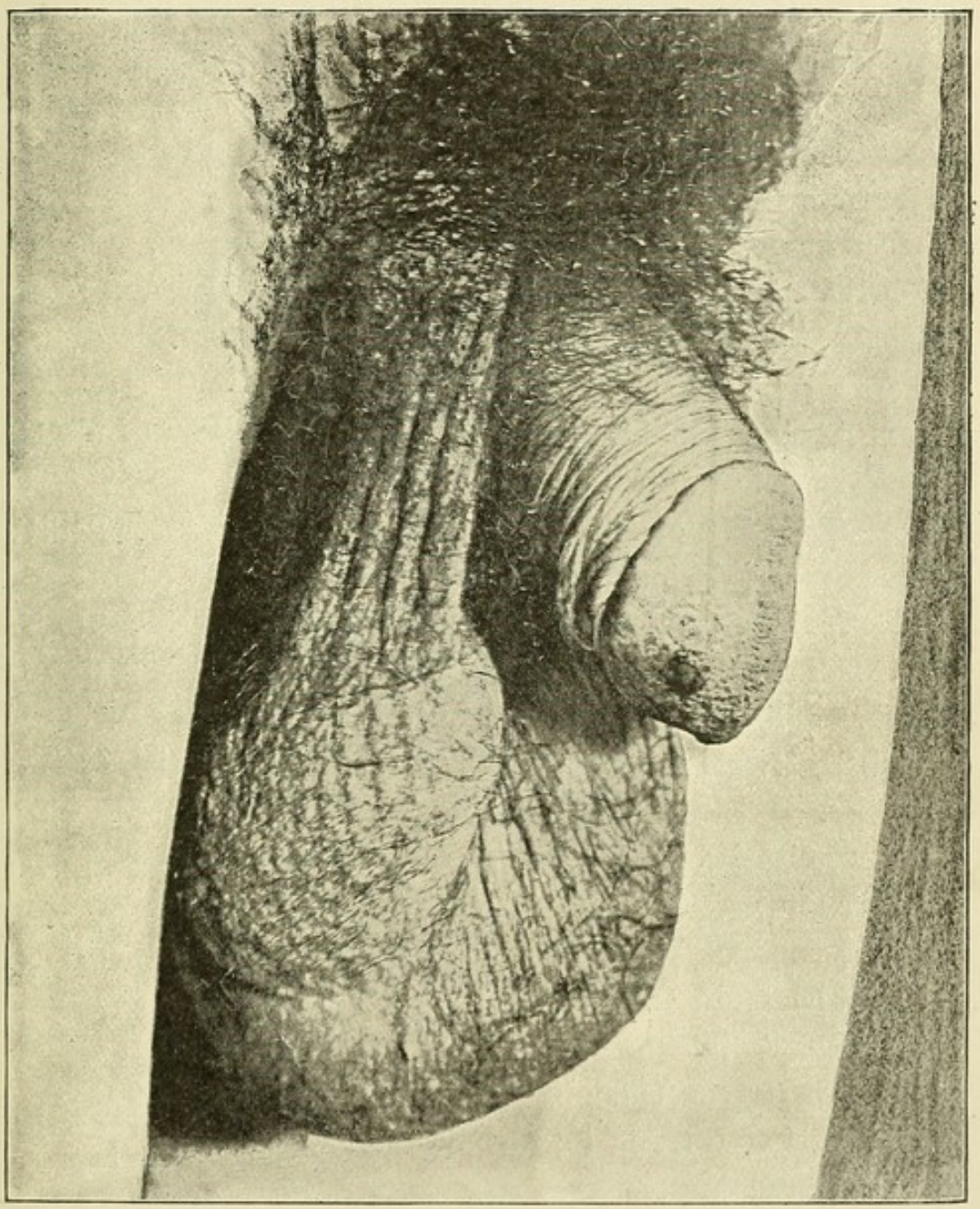

FIG. 12.—SCABIES ON THE GLANS PENIS RESEMBLING CHANCRE OF THE "INDURATED PAPULE" TYPE. (Piffard.)

The clinical picture of ulcerated chancre is that of a neoplasm eaten out by an ulcer, not that of an ulcer surrounded by an inflammatory ring. The neoplasm may be embedded within the skin; but pick it up, and you will realize that it is a distinct lump with an ulcer in its center.

THE INDURATED PAPULE.—This is the rarest type of chancre. It occurs usually in situations where the integument is so dense and thick as to prevent very extensive development of the neoplasm. The induration consequently remains a small, dark-red, flat papule. As it begins to heal the surface becomes scaly.

EXCEPTIONAL VARIETIES.—The induration may be so slight as to be clinically imperceptible. Fournier noted this *absence of induration* seven times in 300 cases.

As a result the lesion appears to be either:

1. A superficial herpetiform ulceration or group of ulcerations (herpetiform chancre) or

2. A grayish or silver-white spot of thickened epithelium. This is seen only on the glans penis.

Both these types are extremely rare.

On the other hand, the *induration* may be very *extensive*, and extend far beyond the ulceration. Its characteristics remain the same as those of the usual smaller varieties.

Complications of Chancre.—The chief complications of chancre are:

1. Lymphangitis and edema.
2. Chancroid (mixed sore).
3. Simple inflammation.
4. Phagedena (gangrene).
5. Transformation into a mucous papule.
6. Vegetations.

LYMPHANGITIS.—The lymphangitis of chancre is by no means constant. Corded lymphatics, running from the chan-

cre to the adjacent glands (e. g., along the dorsum of the penis), are not often seen.

But in certain localities, such as the prepuce and the labia majora, a great mass of lymphatic induration may surround the chancre, or small similar masses may lie adjacent. Such a complication obstructs the lymphatic flow and causes considerable edema. It is sometimes spoken of as indurative edema (Figs. 13, 14).

"Mixed" Sore.—As chancroid itself is rare among the upper classes, so is the mixed sore, the combination of chancroid

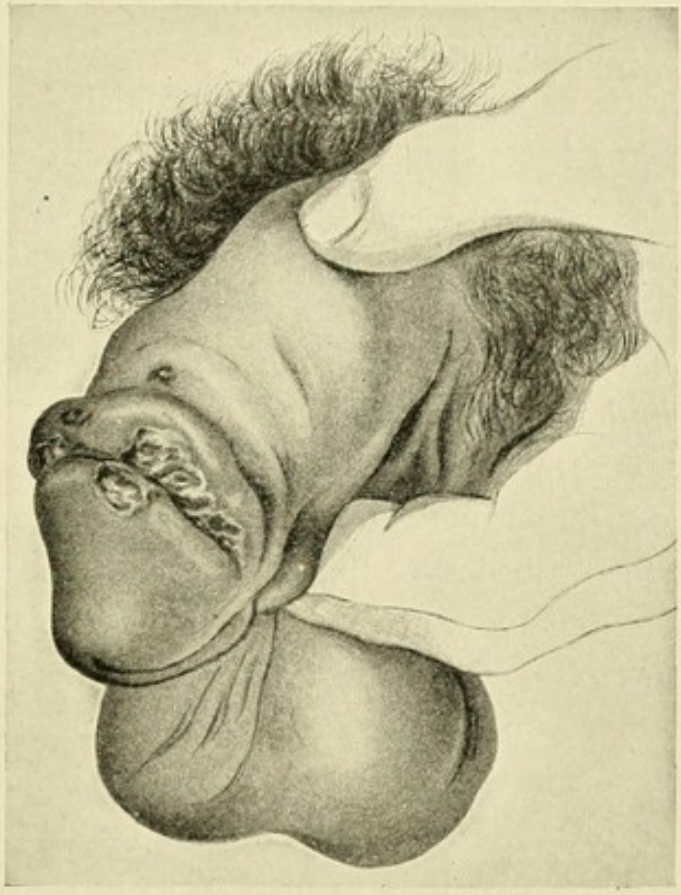

Fig. 13.—Multiple Chancroids Concealing a Chancre ("Mixed" Sore) of which the only Evidence is the Ridge of Induration Back of the Corona. (Kaposi.)

and chancre. Among the chancres seen in the dispensary, however, fully one third are "mixed sores." A person may be infected with syphilis and with chancroid at the same time.

In such a case the chancroid develops first, and a few weeks later becomes chancrous (Fig. 13).

On the other hand, infection with chancroid may occur in the true chancre by subsequent inoculation.

Hence the possible combinations of chancre and chancroid are three:

1. The chancroid may appear, flourish, and be cured, and from its remains the chancre may arise.

2. The chancroid may overlap and overshadow the chancre, so that the latter is suspected only from the induration remaining after the sore heals, or proven by the appearance of secondary syphilitic lesions.

3. A true chancre may become chancroidal.

Of the three types, the second is the one commonly observed. The presence of chancre is not even suspected until the chancroid in healing begins to take on a suspicious hardness, or until a roseola breaks out all over the patient. *While the patient has an active chancroid, therefore, one can never assure him he has not true chancre.*

Inflamed Chancre.—The friction of clothes, or any other form of trauma may so irritate the chancre that it becomes acutely inflamed; yet this is unusual. As a rule, the pyogenic microbes have no effect upon chancre beyond encouraging ulceration.

Gangrenous and Phagedenic Chancre.—The obstruction to circulation in the indurated base of a chancre is habitually sufficient to excite desquamation and exudation from its surface. Exceptionally, it is so marked as to cause *gangrene* of the dermis. Such a complication is of no great importance.

Phagedena is far rarer and far more important. The ancients used to consider phagedenic chancre relatively common, yet we almost never see it. This is not because syphilis is less severe, but because diagnosis is more accurate. We recognize nowadays the frequency of gumma of the penis (chancre

redux), and the imminent prospect of phagedena in this lesion as well as its occurrence (much less than of yore) as a complication of chancroid.

Indeed, *the occurrence of phagedena is presumptive evidence that the sore is not chancre.*

I have seen but two cases of true phagedenic chancre, one in clinic, one in private. The history of the latter is as follows:

Case XXVII.—May 4, 1906.—Male, aged twenty-six, exhibits a large necrotic ulcer which has destroyed almost the whole right half of the upper lip and is advancing rapidly. The ulcer began two months ago as a "pimple" (cause unknown), which refused to heal for about six weeks, and then suddenly began to spread and destroy the lip. He consulted a physician, who saw a macular eruption, which has since faded and has never been observed by the patient. Mixed treatment was accordingly administered, but it ruined the digestion without controlling the lesion, and the patient was therefore brought to me.

Except some tender submaxillary glands and a superficial ulcer on the left tonsil, I find no lesions but the lip ulcer, which is very foul and manifestly progressing. It extends from the premaxillary fold to the angle of the mouth, and has destroyed the lip internally up to the alveolar reflexion of the mucous membrane, externally to within a quarter of an inch of the nose. The patient is clean looking, has no bad habits, and no knowledge of how he acquired the sore. He has lost twenty pounds, suffers such excessive pain in the lip that he neither sleeps nor eats, and is extremely weak and anemic.

A smear[1] reveals *Spirocheta pallida* and various cocci. I stopped internal treatment to give the digestion a rest, advised local applications of potassium permanganate, and administered two hypodermic injections (0.3 c.c., fifty per cent gray oil = 0.15 c.c. metallic mercury in each dose) on successive days. Both injections were excruciatingly painful, but did not salivate. A week later I gave him double the dose (0.6 c.c.). In another week the digestion was much improved, so iodid of potassium

[1] By Dr. Henry Brooks.

was prescribed; no injection. At the end of three weeks both tonsils had ulcerated, but were improving rapidly. The lip was worse; iodid increased; injection of lipiodol, 4 c.c., and gray oil, 1.3 c.c.; local applications of argyrol (which proved more soothing than anything else). After this the patient was mildly salivated, and no further injections were given. Iodid was run up rapidly. On June 7th he reached 255 drops a day, and the digestion gave out so completely that I bade him stop, though the ulcer was not under control. Meanwhile he had lost a pound a day in the past month. His normal weight was 150 pounds; now 102. The ulcer had advanced about an eighth of an inch all around.

Salivated, his power of digestion utterly gone, unable to sleep for pain and weakness, and with the ulcer still slowly progressing, it took all the poor lad's fortitude to accept my assurance that the end was at hand. But in a week healing had begun, and he felt so much better that the iodid was resumed at 120 drops a day, and increased in two weeks to the former maximum, then stopped. On July 5th he had a ravenous appetite, weighed 126 pounds, and the lip was halfway healed, leaving a terrible deformity. Yet the patient is so delighted to be rid of the pain, the disgusting fetor, the everlasting slough, that he could scarcely be happier were he entirely well.

(Within two months he had a large gumma of the shin, which was ten weeks in healing. Since then—to July, 1907—he has remained well.)

Such is the true phagedenic chancre—one of the most virulent types of the so-called "malignant precocious syphilis," and eminently deserving of the name.

Whether this phagedena is due to mixed infection (and comparable to noma and to fulminating gangrene of the genitals) or to the same conditions that produce phagedena in tertiary lesions I do not know. But, like tertiary phagedena, it is virulent in onset, usually self-limited under almost any treatment, and to be combated by hygiene [1] and mixed treatment.

[1] The hygienic possibilities in the cited case were almost nil, for the patient had spent all his money and could scarcely masticate or swallow his food.

Transformation into a Mucous Papule.—Chancre upon the mucous membrane or between moist folds of skin may, at the time of the first, general, secondary outbreak, become a typical mucous papule. The fact requires no further comment.

Vegetations.—Soft warts may surround the chancre. Their presence is accidental, and can scarcely be called a complication.

Duration.—The chancre usually lasts four to six weeks, though some trace of induration may remain many months.

Reinduration of the chancre, which simply means recurrence of syphilitic inflammation (secondary or tertiary) in a chancre partially or wholly cicatrized, may prolong its duration indefinitely.

Fournier relates that he has seen a chancre run its whole course in two weeks. This must be about the minimum. Yet patients will often say that their chancres only lasted a few days, for they are careless observers. Their testimony merely bears witness to the clinical insignificance and painlessness of this lesion so fraught with grave consequences.

Diagnosis.—The diagnosis of chancre depends so largely upon the character of the adjacent adenitis that it is deferred to a subsequent chapter.

Prognosis.—The prognosis is excellent. The chancre will get well spontaneously, and little can be done to hasten its healing.

As to general prognosis, as the chancre is syphilitic, so is the patient.

Treatment.—If the chancre is not ulcerated a daily wash with warm water, protection from friction of the clothes, and the application of any simple dusting powder is all the treatment it needs.

If ulcerated, black wash (lotion nigra) is the customary application, and I know none better.

If it is a mixed sore, treat the chancroid.

If inflamed, wet dressings of carbolic-acid solution, 1 : 200.

If phagedenic, twenty per cent argyrol, and vigorous mixed treatment.

The general treatment of syphilis is not often begun until the chancre is almost healed. Begun earlier it encourages healing, but this is a matter of no moment.

CHARACTERISTICS OF CHANCRE IN VARIOUS LOCATIONS

Chancre of the Male Genitals.—Chancre of the *glans penis* is small, thin, and erosive. When multiple, they may (rarely) simulate a group of herpetic papules (herpetiform chancre).

Chancre of the *coronary sulcus* is large and thick. It is usually associated with plaques of lymphangitis; indeed, it is not rare for this lymphangitis to surround the penis like a collar (Fig. 13).

Chancre of the *internal surface of the prepuce* follows the usual erosive type. At the *orifice* its lymphangitis may cause inflammatory phimosis.

Chancre at the *meatus* is characterized by a dark red tumefaction of one or both lips. The erosion may be entirely within the urethra or it may extend over the surface of the glans. The diagnosis of meatus chancre is made by palpation. Grasp the tip of the glans above and below and you immediately appreciate the characteristic elastic induration.

Chancre of the *skin of the penis* is large, superficial, and crusted. On removal of the crust it may look as though the edges of the ulcer were sharp and undermined, like those of chancroid, but close inspection will prove that they are not.

Chancre of the *pubes* is usually large and deeply ulcerated.

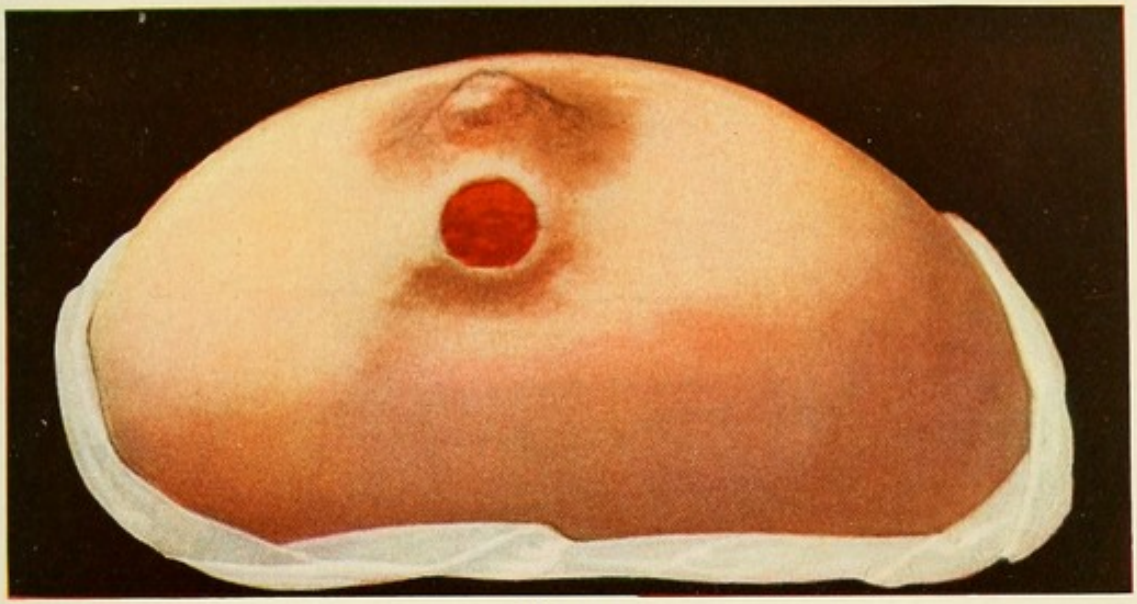

Fig. 1.

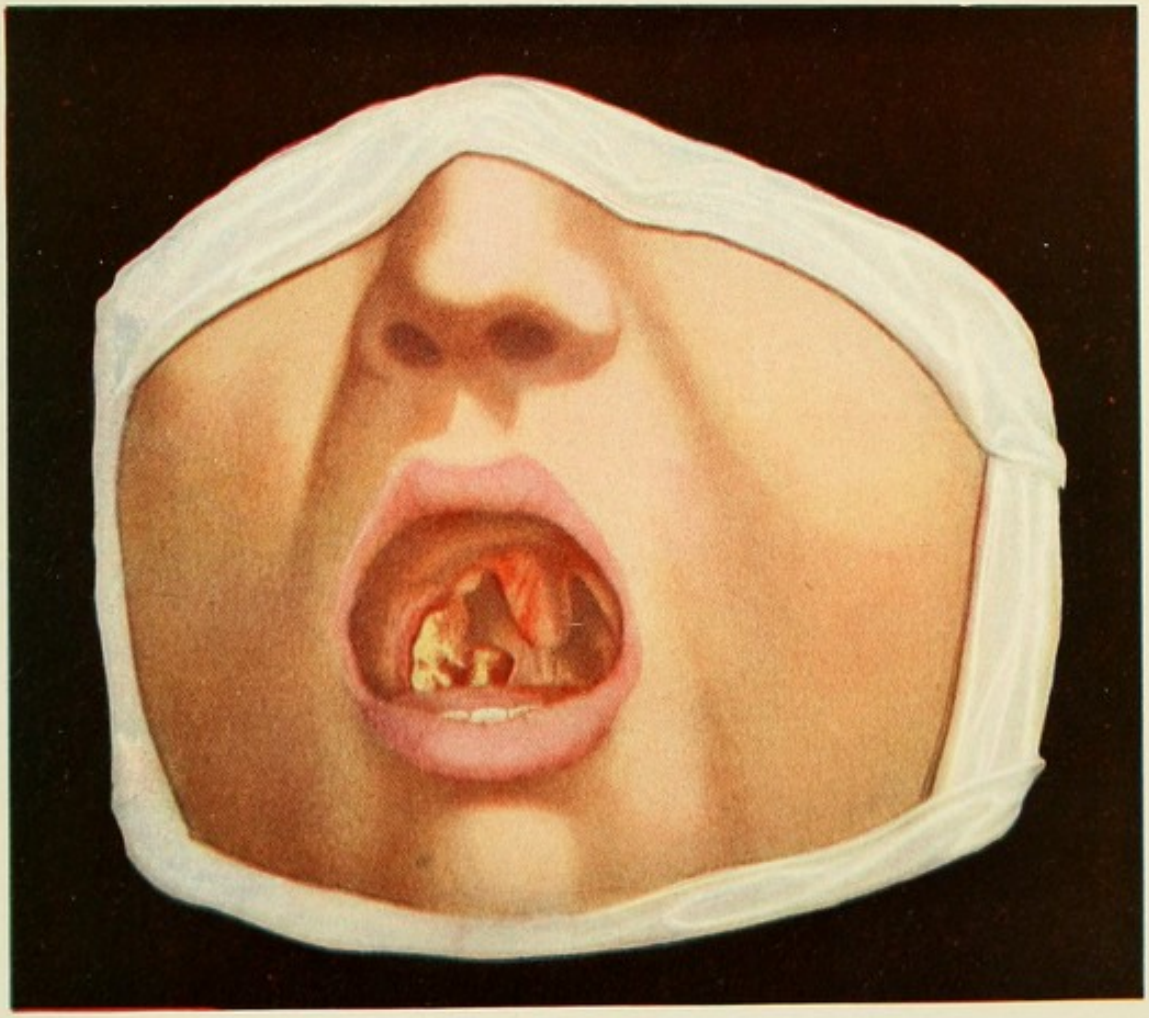

Fig. 2.

PLATE IV.—Extragenital Chancre.

Fig. 1.—Chancre of breast. (Musée, St. Louis.)
Fig. 2.—Chancre of tonsil. (Musée, St. Louis.)

Chancre of the *scrotum* is usually eroded, with marked induration.

URETHRAL CHANCRE.—Chancre of the urethra is far from rare. Among 549 cases of penile chancre examined I have found 62 urethral chancres.[1] They were all in the terminal inch of the urethra, except three. One of these was in the scrotal portion of the urethra, the other two in the pendulous portion. Each of the three was first seen by me at the time of the secondary outbreak. In two of them the mass of induration could be very distinctly felt externally. The third, a chancre of the penile portion, could be plainly seen through the urethroscope as a dark-red neoplasm, but it was too near to healing to be felt externally.

One of these chancres was so large as to cause chordee and great dysuria.

As a rule, however, the only symptom noticed by the patient is a very slight urethral discharge. This he mistakes for a simple urethritis (or he may overlook it entirely). Pain on urination at the inflamed point is usually slight.

Hence it is *prudent to suspect of urethral chancre* (1) *every patient with simple urethritis, and* (2) *every patient in early secondary syphilis who denies the existence of chancre.*

Palpation of the glans from before backward reveals chancres in that region. Lateral palpation of the urethra reveals deeper ones. The urethroscope shows a livid mass covered with false membrane.

Chancre of the Female Genitals.—The regions involved in order of frequency are the labia majora, the labia minora and fourchette, the cervix uteri, the region about the vestibule, urethra, and introitus, the vaginal wall (extremely rare).

Chancre of the *labium majus* is usually large and ulcer-

[1] Yet I feel that this (eleven per cent) is far above the general average of frequency.

ated, often surrounded by a mass of matted lymphangitis (Fig. 14).

Chancre of the *cervix* may surround the cervical canal or may invade one lip only. It is large, and covered with false

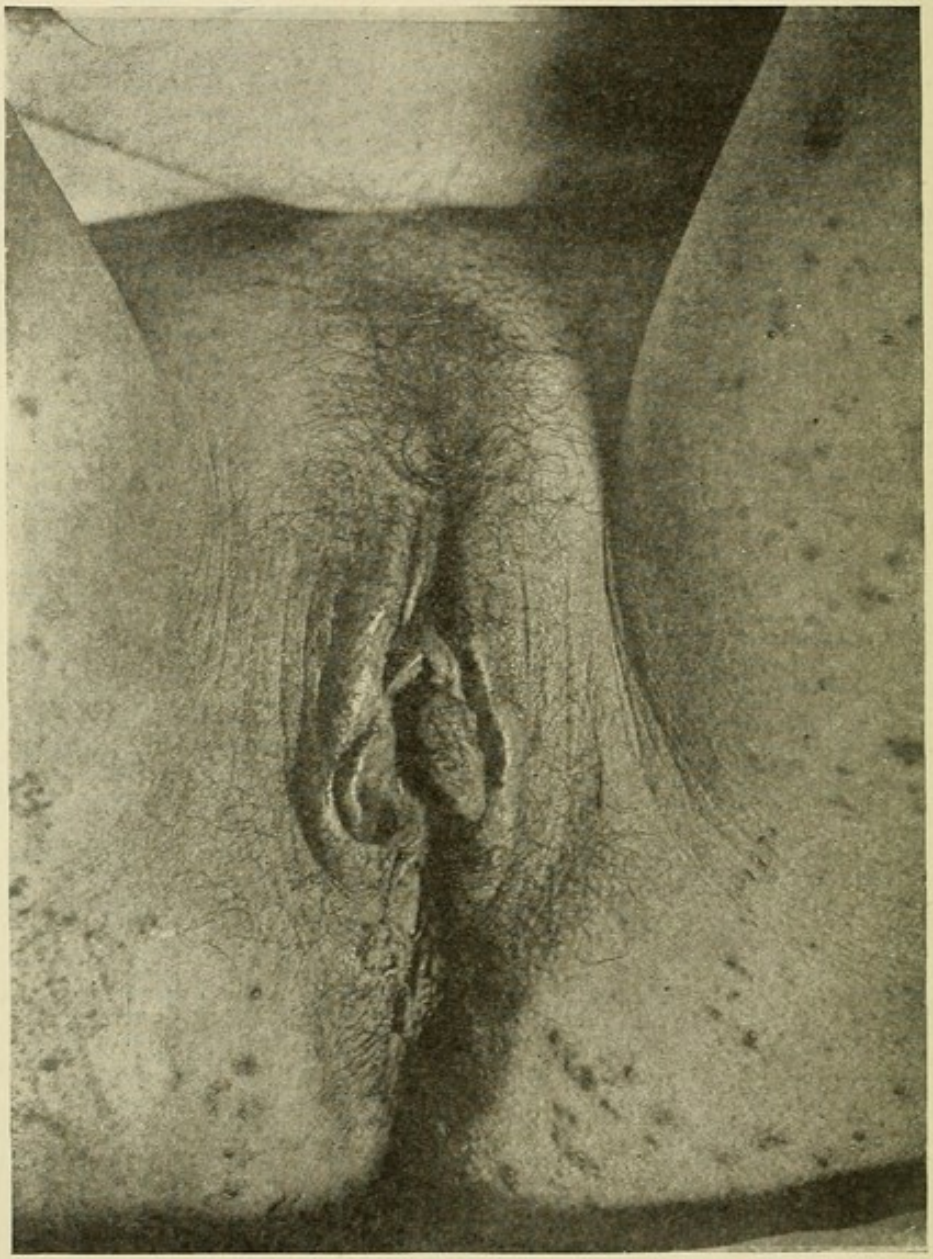

FIG. 14.—CHANCRE OF LABIUM NEAR FOURCHETTE: INDURATIVE EDEMA OF VULVA: PAPULO-MACULAR SYPHILID. (Piffard.)

membrane around which the chancre extends in a thick, purplish rim. Its secretion is slight, the pain nil. Hence the

patient never complains of it; one must search in order to discover it. The induration is scarcely palpable on account of the density of the cervical tissue.

Chancre *elsewhere* within the female genitals is usually small and parchmentlike. In the vestibule and about the urethra it may excite lymphangitis, but in the vagina its induration dwindles down to almost nothing. Indeed, the clinical rarity of vaginal chancres is believed to be due to their being overlooked.

Extragenital Chancre.—As is shown in the tables on page 56 extragenital chancre is much more common in and about the mouth than anywhere else. Chancre of the lips is commonest of all. The lower lip is most frequently affected.

Buccal Chancre.—Chancre of the *lip* (Plate V, Figs. 2, 3, also Fig. 17) varies in size from the smallest papule to a great ulcerated mass. Usually, however, it is large, prominent, crusted, and extremely distressing to its possessor.

Chancre of the *tongue* may appear on any part of that organ, but is commonest on the dorsum toward the tip. It is usually quite large, and the lymphangitis may infiltrate the surrounding tissue widely.

Chancre of the *tonsil* (Plate IV, Fig. 1) produces the general and local symptoms of a mild tonsilitis. There is lassitude, often quite a sharp fever, and considerable local pain on deglutition.

Inspection reveals a general enlargement of the tonsil. It is covered with a thick, adherent, false membrane under which the surface is flat and eroded or exulcerated. All the surrounding tissues are swollen. Palpation reveals marked induration.

Exceptionally the tonsillar chancre is a small eroded papule.

Chancre of the *gum* is extremely rare. It forms an eroded induration surrounding the roots of several teeth.

TEGUMENTARY CHANCRE.—Chancre of the *skin* is, generally speaking, quite large and often ulcerated.

Chancres of the female *breast* (Plate IV, Fig. 2), often multiple, usually affects the areola.

Chancre of the *finger* is rarely seen except upon doctors and midwives; but among them it is sadly common. Its location, four times in five, is about the nail of the index finger. Such types as the fungating chancre (Taylor), the periungual, and the hypertrophic chancre (Fournier) are sufficiently described by their titles.

The sore is usually large, purple, fungating, and scabbed. The induration is marked, but cannot, on account of their density, be isolated from the surrounding tissues.

Vaccinal chancre need only be mentioned.

Anal chancre is fairly common among women. It is often hidden within the anus, and lying in a fold may assume a duplex shape, like the leaves of a book (*chancre en feuillets de livre*). Anal chancre causes some pain on defecation.

Rectal chancre is rare, symptomless, almost always overlooked. It has no special characteristics.

Chancre of the *eyelid* is rare.

Chancre of the *conjunctiva* is rarer still. Occurring near one of the commissures it causes dense induration of both lids, and by pulling these apart we find the ulcer between. Elsewhere on the conjunctiva (usually the palpebral) it is small and erosive. There is always an associated acute conjunctivitis.

THE ADENITIS OF CHANCRE

Syphilitic inflammation of the group of lymph nodes adjacent to the chancre is part of the initial lesion. It is as constant and typical as the chancre itself. Indeed, Fournier failed to find it only thrice in 5,000 cases; he suggests that congen-

Fig. 1. Fig. 2.

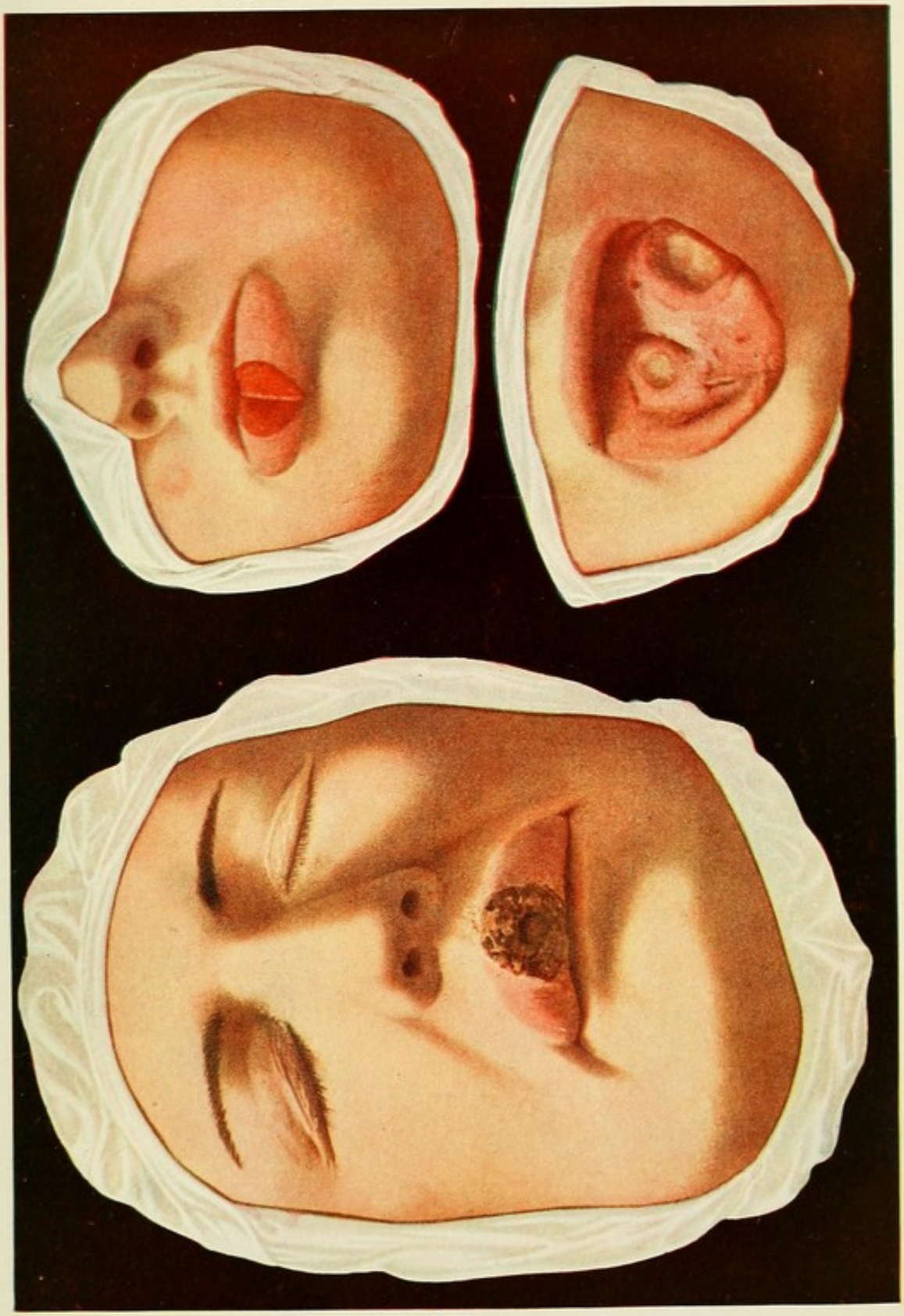

Fig. 3.

PLATE V.—Extragenital Chancre.

Fig. 1.—Chancre of the lip: erosive type, deeply split. (Musée, St. Louis.)
Fig. 2.—Two chancres of the tongue. (Musée, St. Louis.)
Fig. 3.—Chancre of the lip: crusted. (Musée, St. Louis.)

ital absence of the inguinal glands may explain the absence of adenitis in these cases.

The nodes affected are those fed from the site of the chancre, thus:

LOCATION OF CHANCRE	ADJACENT ADENITIS
Genitals, anus, buttocks, thigh, leg, foot.	Inguinal.
Lip or chin.	Submaxillary.
Tongue.	Subhyoid.
Tonsil.	Lateral cervical.
Eye—near outer canthus.	Preauricular.
Eye—near inner canthus.	Submaxillary.
Finger, hand, forearm.	Epitrochlear and axillary.
Arm, breast.	Axillary.

Chancre of the cervix uteri gives no external adenitis, though doubtless the pelvic nodes are affected.

Inguinal adenitis may be bilateral or unilateral; if the latter, it is usually on the side corresponding to the chancre. Exceptionally the lymphatics so anastomose that the adenitis is on the opposite side (*crossed bubo*). As a rule, however, both sides are affected.

Symptoms.—The adenitis (bubo) appears in the second week after the appearance of the chancre, usually on or about the tenth day. It reaches maturity in two or three days, and presents the following characteristics: multiplicity, moderate size, absence of periadenitis and of all acute inflammation, hardness, slow resolution.

MULTIPLICITY.—There is always a group of nodes involved; indeed, inguinal adenitis usually shows involvement of a group in each groin, but the one rather more enlarged than the other.

This group, or pleiad, as Ricord appropriately termed it,

is made up of one (rarely more) large node surrounded by a group of lesser ones (clinically the large node often predominates the scene, the lesser ones being scarcely discernible).

Size.—The larger node scarcely attains the size of a cherry and may be much smaller; the lesser ones are the size of peas.

Absence of Inflammation.—Unless there is mixed infection the nodes are neither painful nor tender. They are freely movable beneath the skin, upon the subjacent parts, and upon one another.[1] The skin over them is not discolored; they do not suppurate. This *complete absence of periadenitis* is one of their most striking characteristics.

Hardness.—"The hardness of the nodes is the hardness of the chancre"; such is the routine statement. The clinical facts do not quite bear it out. Though the nodes may be as hard as the chancre, and when so are typical, in the larger number of cases they are distinctly more elastic. Examination of a series of patients will impress this characteristic.

Slow Resolution.—The great virtue of syphilitic bubo is that it persists many weeks after the chancre has disappeared and may lead to the discovery of the scar of a healed chancre. It usually persists three or four months.

Unusual Varieties.—The bubo may be abnormal, inflamed, or "mixed."

Abnormal Bubo.—Exceptionally the bubo consists of a single very large gland, or the large gland is altogether lacking.

Inflamed Bubo.—Inflamed bubo is much more common than inflamed chancre. The pyogenic bacteria multiply upon the chancre and from it enter the lymph current, yet may not cause much local irritation. The common clinical causes of inflamed bubo are genital filth and cohabitation.

The bubo of labial or buccal chancre is habitually a large, tender, inflamed mass.

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[1] Unless they are greatly inflamed, in which case they adhere tightly to one another.

As a result periadenitis occurs; the nodes become matted together and are painful and tender. Suppuration is extremely rare,[1] and—singular fact—when it does occur, it appears to spare the nodes themselves; evacuate the pus, and the syphilitic nodes remain. The condition is a suppurating periadenitis.

"MIXED" BUBO.—Syphilitic adenitis may be complicated by chancroid or by tuberculosis.

Chancroid and chancre combine to make a "mixed" sore and a "mixed" bubo. In both instances the characteristics of the chancroidal lesion overshadow the other.

Tuberculosyphilitic nodes I have never seen. They are said to assume the tubercular type.

Diagnosis.—The typical group of one large uninflamed gland surrounded by a lot of little ones—all of them hard, insensitive, and not adherent—is so characteristic that a discussion of its differentiating characteristics is all but superfluous.

Certain varieties of herpes or balanitis excite a bubo quite similar to that of syphilis; but the exciting lesion is so dissimilar that a mistake is scarcely possible.

The insensitive, hard, movable nodes of syphilis can scarcely be confused with the inflamed, tender, adherent nodes of chancroid; though, as we have already said, the latter may conceal the former.

The bubo is readily diagnosed and forms, as we shall see, one of the most important means of diagnosing chancre.

Treatment.—Neither demanded nor available.

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[1] Fournier records 1.6 per cent of suppurations in the clinic, 0.6 per cent in private practice.

CHAPTER XVII

DIAGNOSIS OF THE INITIAL LESION

In order to diagnose chancre, study its bubo. The clinical characteristics of chancre are doubtful; how often only the syphilologist knows, while the nodes are usually pathognomonic.

Moreover, the chancre may be insignificant, or may have healed before the patient comes under observation: examine the nodes carefully and you will usually light upon a group that leads to the site of chancre.

In order of importance, therefore, the diagnostic features of primary syphilis may be classified thus:

1. Character of the sore.
2. Character of the bubo.
3. Autoinoculation.
4. Examination for spirocheta.
5. History and confrontation.
6. The test of time. Development of secondary syphilis.

Character of the Sore.—It is usually single, small, uninflamed, indurated, eroded rather than ulcerated, smooth and glistening of surface, dark red in color, not excavated.

Moreover, *any sore lasting more than a week may be chancre.*

Character of the Bubo.—Need not be repeated.

Autoinoculation.—Syphilitic autoinoculation is barely possible, and produces only an insignificant little papule, while chancroid produces a typical chancroidal ulcer.

EXAMINATION FOR SPIROCHETA.—No one but a specially trained bacteriologist should be trusted for an opinion. A positive report should be given not too much weight; a negative report means nothing.

HISTORY AND CONFRONTATION.—An accurate history of a single sexual exposure three to six weeks before the appearance of the sore is a presumption in favor of its being chancre, and if the examination of the suspected partner reveals infectious secondary lesions upon her, this confrontation, as it is termed, adds great weight to the presumption. But the most perfect history cannot constitute objective certainty.

Negative history or negative confrontation, on the other hand, are scarcely to be considered in evidence. Just as syphilis frequently is *not* acquired after exposure to a manifestly infected source, so is it sometimes acquired by exposure to an apparently irreproachable source. The minutest examination of the infectress may fail to reveal the slightest infectious lesion.

Requirements for Diagnosis.—To suspect a chancre is easy; to prove it extremely difficult. Though always assuming a most guarded attitude, I have been deceived often enough to realize that *the diagnosis of syphilis from chancre must not rest upon anyone's opinion; but upon objective certainty.*

Such certainty can be attained only by the concurrence of four different factors, viz.:

1. A typical sore.
2. A typical adenitis.
3. Negative autoinoculation (and search for the Ducrey bacillus).
4. Positive spirocheta finding by an expert.

Many chancres fail to come up to these requirements; better leave the doubt open until the advent of secondary lesions proves the case. This is peculiarly necessary if no real expert

on spirocheta is available. Better no diagnosis at all than one founded on inaccurate microscopic evidence.

One other thing is necessary before treatment is begun; *be sure that the patient is convinced he has syphilis.* Until he is absolutely persuaded of this, sentence should be suspended; for it requires a firm belief to carry him through the long months of treatment; and the worst thing that could happen to him is to take treatment just long enough to control the first outbreak, and then to quit.

The Test of Time.—Most patients must await the advent of secondary lesions before their syphilis can be absolutely proven.

DIFFERENTIAL DIAGNOSIS OF GENITAL CHANCRE

Herpes.—A beginning chancre may be mistaken for simple herpes progenitalis (Fig. 15).

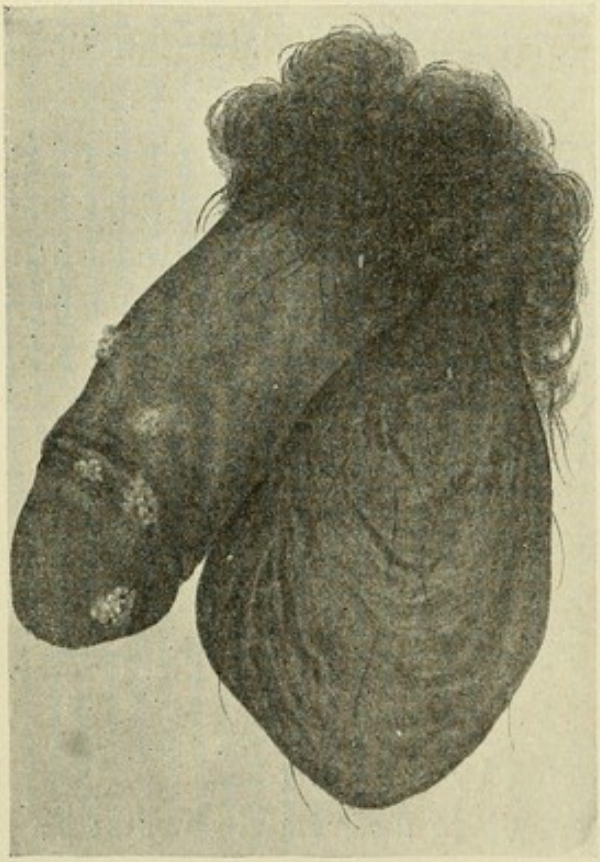

FIG. 15.—HERPES PROGENITALIS (Baerensprung and Hebra).

The typical herpetic eruption, consisting of scattered superficial vesicles and ulcerations surrounded (and preceded) by an erythematous blush, burning and itching, can scarcely be confused with chancre; but the single herpetic ulcer, set on a base of inflammatory induration and associated with mild inguinal adenitis, may closely mimic chancre.

There are three distinguishing characteristics:

Induration is extremely rare with herpes; it is constant with chancre. .

Adenitis is extremely rare with herpes; constant with chancre.

The *contour* of the lesions differs. The herpetic ulcer is made up of many minute lesions, and its edge is, therefore, a series of many little intersecting segments of circles; it is polycyclic and microcyclic (as Fournier has it). The contour of chancre, however irregular, is never thus scalloped.

But all these signs may fail us; herpes and chancre may even coexist. The final test is *time*. Watch the evolution of the lesion. If it gets well within ten days, leaves no indurated scar, no chronic adenitis, it is not chancre. Otherwise one must await the secondaries.

Scabies.—An ulcerated or papular lesion of scabies (Fig. 12) on the genitals may most precisely mimic chancre. As with herpes, there may be slight inflammatory infiltration of its base and enlargement of the adjacent glands.

The diagnosis rests on the discovery of *scabies elsewhere* in the body and the rapid cure of the lesion under sulphur ointment.

Chancroid.—The essential difference between chancre and chancroid is that the former is a neoplasm the surface of which may be more or less ulcerated, while the latter is an ulcer pure and simple. The chancroid may conceal a coexisting chancre.

Gumma.—The three disorders considered above imitate the small ulcerative chancre. Gumma of the penis—chancre redux (page 481), it is very appropriately termed—imitates the large, thick type of chancre (Hunterian chancre).

With gumma there is history of syphilis; with chancre history of exposure. With gumma the concurrence of inguinal adenitis is rare and accidental; with chancre, constant

and characteristic. Gumma is an induration which ulcerates; chancre is an erosion which becomes indurated.

Summary.—The following table sums up the differential diagnosis:

SYPHILITIC CHANCRE	CHANCROID	HERPES	SCABIES	GUMMA
1.— *History.*—Sexual contact, kissing, mediate infection, vaccination, etc.	1. Sexual contact.	1. Relapsing herpes.	1. The family scratches.	1. Syphilitic.
2. *Incubation.*—Two to six weeks.	2. Three to seven days.	2. None.	2. None.	2. None.
3. *Microscope shows.*—Treponema often.	3. Streptobacillus, not always.	3. Nothing characteristic.	3. Nothing characteristic.	3. Nothing characteristic.
4. *Autoinoculability.*—Uncertain and atypical.	4. Produces typical chancroid on third day. From this streptobacillus may be readily obtained.	4. No.	4. Produces a cuniculus, perhaps.	4. No.
5. *Commencement.*—Begins as an erosion or a papule, and remains an erosion or ulcerates.	5. Begins as a pustule or ulcer, and invariably remains as an ulcer.	5. Begins as a group of vesicles, rarely as a single vesicle, and becomes an ulcer.	5. Begins as a papule; becomes ecthymatous.	5. Begins as a tumor which ulcerates later.
6. *Number.*—Usually unique or simultaneously multiple; rarely multiple by successive autoinoculation; never confluent.	6. Usually multiple, both simultaneously and by successive autoinoculation; often confluent.	6. Usually multiple, simultaneously and by successive crops of vesicles; sometimes confluent.	6. Usually multiple and scattered over the body. Not confluent.	6. Usually single. Confluent if multiple.

SYPHILITIC CHANCRE	CHANCROID	HERPES	SCABIES	GUMMA
7. *Physiognomy*. — (*a*) Shape: round, oval, or symmetrically irregular.	7. (*a*) Shape: round, oval, or unsymmetrically irregular, with border described by segments of large circles.	7. (*a*) Shape: irregularly rounded, with borders described by segments of small circles left by the confluent vesicles.	7. (*a*) Shape: round or oval.	7. (*a*) Like chancre.
(*b*) Lesion is habitually flat, capped by erosion or superficial ulceration; or scooped out; or a deep, funnel-shaped ulcer with sloping edges. Sometimes the papule is dry and scaly.	(*b*) Always a true ulcer, excavated, hollowed out.	(*b*) Ulcer usually superficial; sometimes in solitary herpes there is but one absolutely circular vesicle. There are usually neighboring groups of vesicles to clear up the diagnosis.	(*b*) Ulceration slight. Ecthymatous.	(*b*) Like chancre.
(*c*) Edges: sloping and adherent, sometimes prominently elevated.	(*c*) Edges: sharply cut, abrupt, often undermined.	(*c*) Edges: sharp, not undermined.	(*c*) Edges: like herpes.	(*c*) Edges: like chancre.
(*d*) Bottom: smooth, shining.	(*d*) Bottom: uneven, warty, irregular, without luster.	(*d*) Bottom: even, inflammatory.	(*d*) Like herpes.	(*d*) Like chancre.
(*e*) Color: somber, darkish red, gray, or black; sometimes livid and scaly, occasionally scabbed.	(*e*) Color: yellow, tawny, false-membranous-looking; sometimes bright.	(*e*) Like chancre.	(*e*) Like ecthyma.	(*e*) Like chancre.
(*f*) Secretion: slight, serosanguinolent, unless irritation provokes suppuration.	(*f*) Secretion: abundant and purulent.	(*f*) Secretion: slight, seropurulent.	(*f*) Secretion: very slight.	(*f*) Like chancre.

SYPHILITIC CHANCRE	CHANCROID	HERPES	SCABIES	GUMMA
8. *Induration.*—Constant, parchmentlike and very faint, or cartilaginous and extensive, terminating abruptly, not shading off into parts around, movable upon parts beneath the skin, and not adherent to the latter: may disappear in a few days, usually outlasts the sore and may remain for months.	8. Absent in typical cases. An induration may be caused by irritants or by inflammation. It is boggy, not elastic, shades off into surrounding tissues, is adherent to parts around, disappears promptly on healing of the sore, or before that time.	8. Inflammatory induration, capable of being produced by the same causes as in chancroid, and behaving in a precisely similar manner.	8. Same as herpes.	8. Same as chancre. Induration always extensive.
9. *Sensitiveness.*—Absent.	9. Painful and sensitive.	9. Beginning heat.	9. Itch.	9. Absent.
10. *Duration.*—At least a fortnight.	10. Varies from a few days to many weeks.	10. Rarely more than ten days.	10. Indefinite.	10. At least a fortnight. Usually much longer.
11. *Phagedena.*—Extremely rare.	11. Rare.	11. No.	11. No.	11. Uncommon.
12. *Lymphangitis.*—Syphilitic.	12. Inflammatory.	12. Same.	12. None.	12. None.
13. *Lymphadenitis.*—Syphilitic, constant.	13. Chancroidal; or inflammatory in one third of all cases.	13. Rare, inflammatory.	13. None.	13. None.

The final test is the advent of secondary symptoms.

DIFFERENTIAL DIAGNOSIS OF CHANCRE OF THE MOUTH

Chancre of the Lip.—The following table gives the salient points of differentiation between chancre and epithelioma:

CHANCRE	EPITHELIOMA
1. *Incidence.*—Usually occurs in youth. About one third of the patients are women.	1. Rare below 40. Extremely rare in women. Usually a history of excessive smoking.
2. *Physiognomy.*—Surface flat and shining; edges rounded: does not bleed readily.	2. Surface irregular, vegetating: edges thick, raised, everted; bleeds at the least touch.
3. *Adenitis.*—Nodes enlarged within a week or ten days. May be tender from mixed infection. Often very large. Begin to subside in a few weeks.	3. Nodes enlarge only after several months. They enlarge slowly and progressively. Mixed infection is uncommon.
4. *Course.*—The sores and nodes reach their height in a few weeks, and as they subside the secondary lesions appear.	4. Advance slow and progressive for months.
5. *Secretion* contains spirochetæ.	5. Secretion contains vulgar spirilla. Biopsy shows epithelial pearls.

Chancre within the Mouth.—Chancre within the mouth has been erroneously diagnosed as often, probably, as any other lesion of syphilis. This because of the prevalence of Vincent's angina, a fairly common infection, absolutely unknown to the general practitioner, and which causes lesions quite indistinguishable by the ordinary clinical tests from chancre.

Vincent's angina is an acute infection of the mouth caused by a long spirillum and a short, thick bacterium, pointed at the ends. It is characterized by a generalized inflammation of the mucous membrane, or by ulceration, or both. It is very obstinate to treatment, though ultimately curable by cauterization, preferably with nitrate of silver.

Four out of the six last cases of "chancre" of the mouth that I have seen were Vincent's angina, I believe. I can

in no wise better express my sense of the importance of the condition than by briefly reciting these cases:

Case XXVIII.—Eighteen months ago a gentleman consulted me about an ulceration on the tip of his tongue. It had been there three months in spite of various local treatments applied by sundry physicians in the various capitals of Europe. Some had said it was syphilis; others, not.

There were no enlarged nodes. The ulcer itself was so irritated by recent frequent cauterization that its character was that of a severe burn—a ragged ulcer with raised, thick edges, on the tip of the tongue. It was very painful.

The duration, the absence of enlarged nodes and of any secondaries, assured me that the condition was not syphilitic. He promptly recovered under local applications at the hands of another physician. He has had no further symptoms.

Case XXIX.—About six months later, a handsome young fellow, about to be married, consulted me concerning a sore on his left tonsil. An eminent laryngologist had just told him it was chancre. He vowed he had kissed no one except his betrothed for three months.

Yet the sore was a typical chancre. It had existed a week, covered the whole tonsil with a dark red induration, upon which was a slight ulceration recently cauterized by the laryngologist. The nodes under the angle of the jaw were large and tender, the sore itself exquisitely sensitive.

The poor lad was in despair when I told him his wedding must be postponed, and my unwillingness to make a diagnosis on the chancre alone was small consolation to him. But he returned to the specialist for local applications, under which the sore healed in six weeks. He has not yet developed any further symptoms of syphilis.

Case XXX.—As yet I knew nothing of Vincent and his angina. But in the spring of 1906 I was summoned to examine the throats of a young man and his wife. He showed on his left tonsil a superficial ulcer covered with false membrane and set in a slightly indurated border. Surrounding it was an area of inflammation extending forward toward the lower jaw. Indeed, the patient insisted that the sore had begun on the lower jaw and had moved back to the tonsil; but the absence of scar

discredited this story. The nodes under the jaw were enlarged, and both sore and nodes were exquisitely tender.

The wife showed a similar lesion on the right side of the mouth at the angle of the jaws. It was a week old.

I made the diagnosis of syphilis in the belief that two probabilities make a certainty, and for proof had a smear examined. The smear was returned with a diagnosis of Vincent's angina. Neither developed syphilis.

These cases sum up the differentiating points,[1] which are:

1. The tenderness is much more marked in Vincent's angina than in chancre.

2. The anginous sore (unless cauterized) is more superficial than chancre, and is likely to be surrounded by more or less general inflammation.

3. The glands may be large and tender in either case.

4. Chancre gets well spontaneously; the angina may or may not.

5. Microscopic examination of a smear establishes the diagnosis.

Chancre of the Tongue.—This must be differentiated from traumatic ulcer, due to a ragged tooth, from tubercle, from gumma, from neoplasm, and from Vincent's angina (see above).

Inspection of the teeth reveals the cause of *traumatic ulceration.*

The chief points to distinguish chancre from *neoplasm* are those already detailed for chancre of the lip.

Gumma, apart from other evidences of syphilis, is distinguished by its slowly progressive course and the late involvement of the glands.

Tuberculosis is rare. The ulceration is irregular, ragged, eaten, and exquisitely sensitive, while chancre is rounded,

[1] Malherbe (*Gaz. med. de Nantes*, 1905, vol. xxiii, p. 977) reports a case of buccal chancre complicated by Vincent's angina.

smooth, superficial, insensitive. Moreover, tubercular ulcerations may be multiple, upon the under surface of the tongue (where chancre has not been seen), and accompanied by other tubercular lesions.

The secondary explosion and the examination of a section from the growth form the final tests.

CHAPTER XVIII

SYPHILITIC TOXEMIA

In displaying the many-sided image of syphilitic toxemia, we must remember the late toxemia of chronic syphilis as well as acute, early syphilitic toxemia.

Accordingly, the subject may be divided under two main heads, as follows:

Acute Syphilitic Toxemia:

Hematology and pathological anatomy.
The fever of early syphilis.
General debility.
Symptoms of local congestion.

Chronic Syphilitic Toxemia:

The fever of late syphilis.
Arterial and visceral sclerosis.
Amyloid disease of the viscera.

ACUTE SYPHILITIC TOXEMIA

Apart from the primary lesion the symptoms of the first few months of syphilis resemble those of the acute exanthemata. There are (1) changes in the blood, (2) certain visceral congestions, (3) fever, and (4) the exanthem itself.

As we see the disease in the United States at the present day, the early local lesions of skin and mucous membranes so overshadow the mild evidences of general intoxication that we almost forget they exist until reminded by the occasional case of grave debility or severe fever. Under other condi-

tions, however, grave toxemia is much more common. Thus Fournier, for instance, states that while this condition is uncommon in his private practice, he has found syphilitic fever (only one of the manifestations of toxemia) in one third of the women who visit his clinics.

Indeed, the toxemia of syphilis is much more frequently observed in women than in men. Yet a mild toxemia, a toxemia readily overlooked by the physician, may be noted in the great majority of syphilitics. This toxemia shows itself, not by any striking symptoms of pain or fever, but by a definite reduction of the vital forces and a marked loss of weight persisting during the first six to twelve months after the onset of the secondary symptoms.

HEMATOLOGY

The pathology of the blood in syphilis varies widely, and generally in direct proportion to the severity of the lesions. Grave lesions, whether secondary or tertiary, are associated with marked anemia, while patients who suffer little from the disease show no marked blood changes at any time. Women, inasmuch as they suffer more severely than men from the toxemia of syphilis, usually show a more intense anemia, which is likely to be of the chlorotic type.

Syphilis scorns law, and, accordingly, we find it assuming almost every form of blood change, simulating simple anemia, chloroanemia, leukemia, and pernicious anemia; while leukocytosis occurs and disappears with no absolute rule. The usual changes in the blood found in early syphilis, however, are the following:

The Red Cells.—*Before the outbreak of secondary symptoms*, the first sign of systemic infection is a fall in the hemoglobin. This loss may amount to fifteen per cent or thirty per cent before the eruption appears. Meanwhile there is little or

no change in the red cell count. Thus the preliminary condition is a syphilitic *chloroanemia.*

With the secondary outbreak of local symptoms or of fever, and to a less degree with each subsequent outbreak, there is a rapid fall in the number of red blood cells, while the fall in hemoglobin continues. The cells usually fall to between 3,500,000 and 4,500,000, the hemoglobin to sixty or seventy per cent. The two go hand in hand, and thus constitute a *simple anemia*, though the preliminary chloroanemia, with relative hemoglobin deficiency, may persist.

Administration of mercury rapidly renews the red cells, though the return of hemoglobin to normal may be somewhat slower, so that the patient may pass through another stage of chloroanemia on the way to recovery.

All foreign authors are agreed that prolonged administration of mercury has a poisonous effect, causes a rediminution of cells and loss of hemoglobin. This toxic effect they look for in three to five weeks. Hence the interrupted method of treatment (page 153). We in America, however, continue to get good results from continued treatment, and find that it does not impoverish the blood. An example is given below of an excellent blood condition after nine months of continuous and effective treatment by mercury, partly by injections of salicylate, partly by protiodid granules. Indeed, thirty years ago my father showed that, under proper dosage, this excellent condition would persist for five years [1]—long enough for all practical purposes.

The White Cells.—*Before the secondary eruption* the leukocytes are normal or increased in number, but there is no marked change until—

With the secondary outbreak there is usually, though not necessarily, a distinct leukocytosis—10,000 to 15,000, or even

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[1] *Am. J. Med. Sci.*, 1876, January.

higher. Differential count shows increased lymphocytes and marked falling off of the polymorphonuclear neutrophiles. An increase in the eosinophiles and mononuclears [1] is so often seen that it was at one time deemed part of the morbid process; but further investigation has proven it by no means constant.

The effect of mercury is to dissipate leukocytosis (if this exists) and to bring back to normal the disturbed relation between lymphocytes and polymorphonuclears. This is quite the opposite to the effect of mercury upon non-syphilitic blood, which is to increase the lymphocytes.

The following example is typical. The first column of figures shows the condition a month after the secondary outbreak and at the time treatment was begun. The second column shows the conditions after nine months of continuous treatment. (The patient is now in the third year of his disease, and has had no relapse since the initial outbreak.) The tonic effect of mercury is well shown.

Hemoglobin	75 per cent.	98 per cent.
Red blood cells	4,084,000	5,440,000
Leukocytes	15,600	8,200

Poikilocytosis and slight variations in the size of the red cells were noted in the first specimen, not in the second. No degenerated or nucleated red cells, no pigmented leukocytes or malarial plasmodia in either.

The differential count showed:

	Per cent.	Per cent.
Polynuclear neutrophiles	57	73
Small lymphocytes	32	16
Large lymphocytes	4	5
Mononuclears	1	1
Transitionals	2	1
Eosinophiles	3·5	2
Basophiles	0·5	2

[1] Hanck, *Archiv für Derm. u. Syph.*, 1905, vol. lxxviii, pp. 45, 289.

Course.—The secondary anemia of syphilis begins before the outbreak of secondary symptoms and grows more intense up to this outbreak. Then, as the eruption subsides, the anemia decreases only to relapse with each recurrence of local syphilitic lesions.

Thus a mild case may show but insignificant blood changes, which soon disappear, even without treatment, while the gravest cases continue anemic for months in spite of all one can do. The average syphilitic ceases to be anemic after the first year of the disease, this cessation being due both to time and to treatment.

When the anemia persists in spite of intelligent administration of mercury, it must be attacked by hygiene, aided by iron, arsenic, and cod-liver oil.

PATHOLOGICAL ANATOMY

The generalized congestive and exudative processes that result from manifold deposits of spirochetæ in the capillary walls produce upon the skin the familiar, early, macular, papular, and tubercular general syphilids. Similar deposits doubtless occur throughout the body. The resulting areas of congestion or exudation, if minute and scattered, evoke no symptoms and pass unrecognized. But if more pronounced in certain organs they evoke the symptoms enumerated below.

THE FEVER OF EARLY SYPHILIS

The fever of early syphilis is usually dependent upon the general infection, exceptionally upon the local lesions. Hence there are two types, the essential and the eruptive.

In private practice syphilitic fever is most exceptional. Fournier from his clinical experience states that young anemic prostitutes very commonly (every third case) show some

fever during the first six months of the disease, but recognizes its rarity in men and in the better classes of women.

Though it is quite possible for a patient to run a slight temperature for several days without noting it, any fever severe enough to arouse complaint it has not been my fortune to note, except only the prodromal essential fever.

The Essential Type.—As I have seen it, the fever of large pox (syphilis), like that of small-pox, precedes the eruption.

This fever occurs during the two weeks immediately preceding the eruption.[1] It is usually mild, lasts from three to ten days, and is accompanied by the pains and malaise associated in the public mind with influenza. The temperature rarely goes above 102° F., with a drop of a degree or so each morning.

The fever is commonly mistaken for typhoid or influenza until the appearance of the rash. Immediately the fever drops to normal, and the diagnosis is clear (though once I have known it to be mistaken for measles). This preëruptive drop may be rapid or gradual.

The essential fever recognized by Fournier in ill-nourished young women occurs in the first six months of syphilis, and generally follows the type described above. Less commonly, it imitates (1) malaria, (2) typhoid, or is (3) utterly irregular. The malarial type is quotidian, occurs late in the day, and is not a complete paroxysm of chill, fever, sweat, but rather irregular and abortive. The typhoidal type is more confusing, but history and Widal test should make the diagnosis.

Bulimia (increase of appetite) sometimes accompanies the fever. This symptom may suffice to suggest syphilis, for in no other fever is it so marked. The patient with a temperature of 101° and a voracious appetite is a living paradox.

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[1] In several patients whose fever was severe the eruption was quite late in appearing, as though the fever assumed the chronological position of the eruption and so postponed it.

GENERAL DEBILITY

Prodromes.—During the two weeks immediately preceding the general eruption, the patient shows more or less marked symptoms of systemic intoxication.

His *appearance* is that of distress and depression. He is paler than is warranted by the loss of hemoglobin; he begins to lose weight; he eats little and sleeps badly (though exceptionally there is bulimia, as noted above); his physical and mental strength seems to desert him; he is feeble and listless; he wears a cowed, distressed expression; his head feels full and sore or aches distinctly; his bones may be painful and tender.

All these miseries increase toward nightfall. If there is fever this intensifies them; if the patient is neurotic, and suspects the nature of his malady, an admixture of subjective woes confuses the picture. Moreover, the local lesions of brain, bones, joints, liver, etc. (described below), may add their traits.

Finger has observed a prodromal accentuation of the cutaneous and tendon reflexes, while Bulkley has recorded various changes in cutaneous sensibility—paresthesia, anesthesia, analgesia—occurring at the onset of secondary syphilis in women; one cannot but suspect in such cases a latent hysterical tendency awakened by the fear as much as by the toxemia of syphilis.

Under Treatment.—After the outbreak of the secondary eruptions, these symptoms disappear, not immediately like the fever, but gradually and under the influence of treatment. The more acute pains and aches are gone in a few weeks, but the depression of strength and spirits lasts longer and the loss of weight continues longest of all.

After the first few months all may be well; the patient, cleared of his eruption and freed of his toxemia, is, to all

appearances, as strong and clean as ever he was. But some patients do not escape so soon. They require six months or a year to regain their weight and strength.

Exceptional cases remain under weight for years, even permanently (Case III, page 19). Such permanent debility occurs almost exclusively in women.

Nervous Symptoms.—Say to yourself calmly, "I have syphilis," and you will realize in some faint measure the horror that strikes a man ignorant of all save the wildest and most frightful romances concerning the disease, when you say to him, "You have syphilis."

In our neurotic age one is, therefore, astonished rather at the rarity than at the frequency of neurotic manifestations in early syphilis. But man becomes accustomed to anything; and the few weeks that intervene between the primary and the secondary outbreak are usually enough to calm the patient's worst fears. The impressions that persist in most cases concern the falling out of hair and the falling in of nose.

Indeed, if it has been impossible to make an accurate diagnosis from the initial lesion, the advent of secondary symptoms is usually hailed with something like joy; for the certainty of an evil fate is infinitely less distressing than blank doubt. Hence the horrors of syphilophobia when the patient has not syphilis.

Thus it is not remarkable to find that syphilis excites old neurasthenic and hysterical taints, or even develops them in persons in whom they had never shown before—all this in the first months of the disease, as a rule.

More definite nervous symptoms, such as neuralgia and neuritis, need not concern us here.

In what proportion certain symptoms, such as asthenia, cardiac palpitations, arrhythmia, tachycardia, dyspnea, vomiting, gastralgia, hyperidrosis, cold feet, insomnia, paresthesia,

analgesia, and even epilepsy or general asthenia, are due to toxemia and to neurosis must be decided for each individual case.

Fournier gives the following characteristics of the analgesiæ of secondary syphilis: They are (1) much more frequent than anesthesia, (2) superficial—the deeper tissues retain their sensitiveness, (3) irregularly distributed, (4) usually found in the mammary region and on the back of the hand.

SYMPTOMS OF LOCAL CONGESTION

Under this heading it were proper to discuss almost every lesion of secondary syphilis. Such is not my purpose; but there are certain forms of visceral and bone congestion that occur in a characteristic manner during the first few months of syphilis; these it is convenient to group together, inasmuch as they form part of the secondary outbreak, and are due either to the wide distribution of spirochetæ throughout the body or to the poisons elaborated by them.

These lesions are in order of importance:

1. Lesions of skin and mucous membranes.
2. Lesions of the lymph nodes.
3. Lesions of the bones, joints, and muscles.
4. Lesions of the brain and its envelopes.
5. Lesions of the abdominal viscera.
6. Alopecia.

Lesions of the Skin and Mucous Membranes.—These constitute the body of the secondary outbreak. They are too important for brief consideration here.

Lesions of the Lymph Nodes.—Though the adenitis which forms part of the initial lesion is apparently limited to the adjacent lymph nodes and attacks viciously but one of these, a less active reaction occurs in the whole chain of nodes beyond. Thus with chancre of the mouth a slight enlargement of the

anterior chain of nodes down to the clavicle may sometimes be noted.

Generally speaking, however, there is a pause between the primary and the secondary adenitis. A single group of nodes enlarges with the chancre; after that nothing until the secondary outbreak. Then a general adenitis occurs; not the general adenitis one would expect affecting all the nodes;[1] but *an adenitis confined especially to certain groups—notably to the posterior cervical and epitrochlear.*

The *appearance* of this adenitis may immediately precede or immediately follow the outbreak of skin lesions. Hence it is scarcely possible that the adenitis is secondary to lesions of the skin, as Sigmund and others have held, or to lesions of the deeper structures, as suggested by Lang. Probably it is simply due to diffusion of the spirochetæ through the body. Why the posterior cervical and epitrochlear glands should be most commonly affected we do not know.

The *characteristics* of the adenitis are those of the primary adenitis repeated. The nodes form hard, elastic swellings in single glands or in a little group, with no pain, tenderness, or periadenitis.

Pathologically there is congestion of the parenchyma, enlargement of its cells, exudation of small round cells; later fatty degeneration and atrophy. When actively inflamed the nodes contain a few spirochetæ.

The *epitrochlear nodes* may be felt as small, hard, movable kernels, one or two in number, in the groove in front of the internal epicondylar ridge of the humerus and behind the inner edge of the biceps. They usually lie at least an inch above the condyle, sometimes much higher. Slight enlargements can only be palpated with the elbow well flexed. The inexpert may confuse the brachial artery with a node.

[1] Though such generalized syphilitic adenopathy does occur very exceptionally.

The *posterior cervical nodes* most commonly enlarged are those running along the edge of the trapezius, low down, two little nodes lying high up on the nucha, just over the occipital bone, and a node lying over the mastoid process of the temporal bone.

Other nodes may become enlarged, but this is not characteristic.

Inasmuch as this early syphilitic adenitis forms an important element in the diagnosis of syphilis, its occurrence must be carefully noted. *Not all cases of early syphilis have palpably enlarged nodes.* Estimates as to their frequency vary from seventy-five per cent to ninety-nine per cent: I should accept the lowest estimate. Hence if the nodes are enlarged it is very suggestive, almost convincing;[1] but if they are not enlarged, the evidence has no value.

The secondary adenitis lasts usually from one to three months, very rarely into the second or third year. Hence it is absurd to depend upon it for a diagnostic aid after the first few months of the disease. To "diagnose syphilis from the state of the nodes" is a perilous venture.

Lesions of the Bones, Joints, and Muscles.—The *pains* of early syphilis, so much feared by those who know little about the disease, occurred in twenty-one per cent of the men among my cases and in thirty-three per cent of the women (page 86). Mild pains, of which the patient did not complain, are not included in this estimate.

These pains have been called *osteocopic* (bone-breaking) because of their severity and their predilection for the osseous system. They precede or accompany the secondary outbreak. Whether continuous or intermittent, they are typically *noctur-*

[1] The posterior cervical and epitrochlear nodes may, like any others, be enlarged from local absorption. But when the two sets are enlarged simultaneously and are rather hard and entirely insensitive, syphilis may almost be diagnosed from this fact alone.

nal, i. e., they increase markedly at about sundown, and do not let up till morning. They may last for several weeks if untreated, but under light doses of *potassium iodid* they promptly disappear.

These pains may be vague and diffuse, but are more often definitely localized in a given bone, joint, or muscle. Palpation usually reveals some point of sensitiveness.

Manifestly, therefore, these pains are due to *localized* syphilitic manifestations, and may be put down to acute periostitis, myositis, or arthritis, as the case may be. Indeed, in severe or neglected cases, the condition readily passes from mere pain to manifest syphilitic inflammation of bone, joint, or muscle.

A description of such characteristic types as pleurodynia, myosalgia, and arthralgia may suffice to elucidate the subject. Headache is described below.

Pleurodynia is a sharp pain in the side, precisely similar in character and intensity to that of acute pleurisy, but occurring essentially at night. By day it is absent or slight, but throughout the night it rages. A tender point may be found on one or more of the ribs or cartilages.

Myosalgia may cause torticollis or lumbago. It often affects the legs. It is a typical nocturnal pain, but is excited by movement of the affected muscle, and, consequently, it excites instinctive rigidity. The muscle may or may not be sensitive to pressure.

Arthralgia is pain in a joint unaccompanied by effusion. It occurs chiefly at night, and is improved by exercise (during the day). Knee, shoulder, elbow, wrist, and ankle are the joints most often involved.

Lesions of the Brain and Its Envelopes.—*Headache* is the most frequent as well as the most important form of pain at the onset of syphilis. It may be slight or severe, throbbing, constricting, or weighing. It is felt chiefly in the frontal or

occipital regions. *Like all the pains of syphilis it is much intensified at night.*

The exact lesion may be difficult to trace. Lang concludes that many headaches are due to meningeal irritation, since the ophthalmoscope often shows retinal congestion (though sight is not impaired). In other cases tenderness over some portion of the skull suggests a periosteal lesion.

But the great importance of early syphilitic headache lies in the fact that *we can never tell whether or not it is a warning of some grave intracranial lesion.* The severest headache may lead to nothing, yet a mild pain may be the first sign of a hemiplegia or meningitis. Ricord has said: "Suspect every rebellious headache of being syphilitic"; let us add: Attack every syphilitic headache with vigor and determination. Attack it as though you knew it portended cerebral syphilis; thus you will abort many a grave lesion.

Lesions of the Abdominal Viscera.—*Jaundice,* though very rare, is the most important lesion under this head. The symptoms are those of ordinary catarrhal jaundice. The liver is enlarged and slightly sensitive. Bile is absent from the stools, present in the urine and the skin. The cause is not known. It has been attributed to pressure of enlarged glands on the ducts, to hepatitis, even to "roseola" of the ducts. Suffice it to say that it usually disappears in two or three weeks.

Enlargement of the spleen was noted by Woelfert in 16 out of 490 cases of early syphilis. It has no clinical significance and subsides spontaneously.

The *digestive* and *renal lesions* of syphilis do not usually form part of the secondary explosion.

Alopecia.—Alopecia is one of the latest, the least important, and the most feared lesions of the secondary outbreak. It occurs between the third and the eighth month of the disease.

Alopecia in syphilis may be *essential* or *symptomatic*. The latter, falling of the hair over ulcerative lesions on account of destruction of the papillæ, has no peculiar features and requires no comment.

The *essential alopecia* affects the hair of the scalp almost exclusively. Three degrees may be distinguished.

1. General thinning of the hair occurs to some degree in almost every case of syphilis. The hair falls freely for a few months, and then returns to its original thickness. But, as in the other exanthemata, the texture of the hair may be permanently altered. Moreover, since syphilis is usually acquired at the age when senile baldness begins, the hair of the crown or over the temples may never return; but this is no fault of the disease.

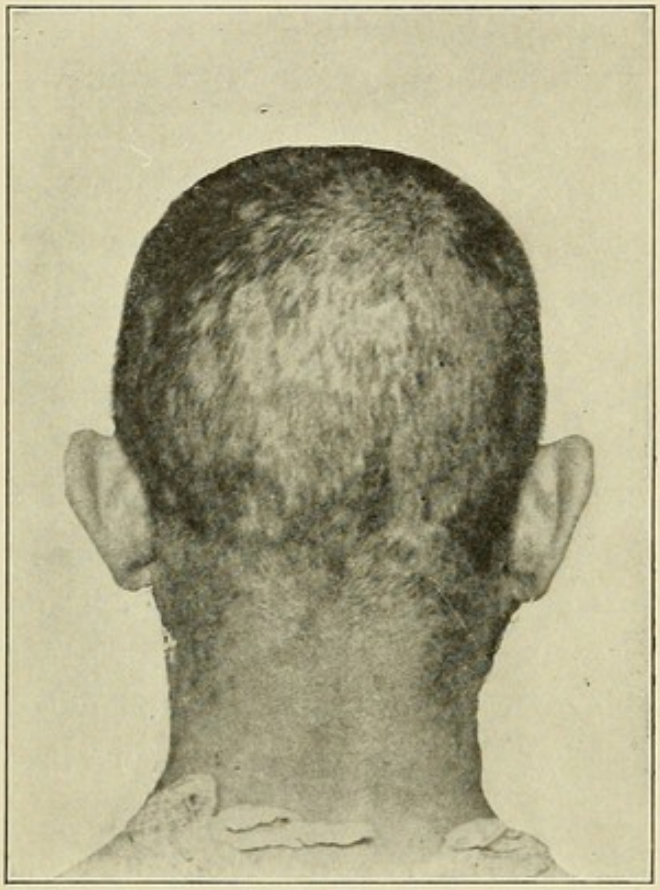

FIG. 16.—SYPHILITIC ALOPECIA. (Pusey.)

2. Besides the general thinning, the hair may fall more markedly in certain parts than in others. This produces a characteristic, mottled, moth-eaten appearance, which is almost pathognomonic (Fig. 16).

3. Finally, this mottling may be so intense as to produce spots of baldness quite similar to those of alopecia areata, but distinguishable by three characteristics.

Syphilitic alopecia	*Alopecia areata*
Spots irregular in shape.	Spots absolutely rounded in contour.
Spots not absolutely bald, a few stray hairs are spared.	Spots absolutely bald.
Skin of the spots normal.	Skin glossy, white, and atrophic.

The two more intense degrees of syphilitic alopecia are quite rare. I have seen but three cases of the third.

Alopecia areata occurs quite frequently in syphilitics; but it is generally believed that there is no essential connection between the two maladies.

The *pathology* of syphilitic alopecia is obscure. The fall of hair seems to be due to the general toxemia.

Any stimulating lotion suffices for *treatment*. The following are excellent:

℞ Tr. capsici ..... ʒij–v (8–20 c.c.);
Glycerinæ ..... ʒss (2 c.c.);
Aqua colognensis .....ad ʒi (ad 30 c.c.).

or

℞ Tr. cantharidis ..... ♏xv (1 c.c.);
Ol. ricini ..... ♏ij (0.12 c.c.);
Aqua colognensis .....ad ʒi (ad 30 c.c.).

or

℞ Resorcin ..... gr. xv (1 gm.);
Alcohol, } .....āā ad ʒi (āā ad 30 c.c.).
Aquæ, }

CHRONIC SYPHILITIC TOXEMIA

Chronic syphilitic toxemia is rare, complex in cause, and clinically atypical. Thus it differs markedly from the clean-

cut type of acute syphilitic toxemia described in the preceding paragraphs.

It is rare, so rare that nine out of ten syphilitics (in the United States) may be said to escape it altogether.

It is complex in cause; indeed, exclusive of the "fever of late syphilis," it is scarcely ever caused by syphilis alone. It is due, habitually, to some combination of syphilis with alcoholism, neglect, privation, etc., amidst which it may be impossible to distinguish essential or preponderant cause.

It is clinically atypical, for its type varies somewhat with every case. For example, Case III (page 19) is an instance of chronic debility (doubtless tubercular), prolonged for years after the first acute toxemia. The fever of late syphilis (see below) is a second type. As a third instance I may cite the the following:

Case XXXI.—A stout, middle-aged man, having suffered for years from ulcerative paronychia of the great toe, was suddenly overwhelmed last winter by a general outbreak of an ulcerative syphilid. A dozen lesions broke out upon him almost simultaneously, varying in diameter from 2 to 6 cm. He could not sleep because of the sores on his trunk, nor walk because of those on his foot. He had a slight afternoon temperature; he could not eat, in two weeks he lost twenty pounds. When I first saw him he had been overtreated with iodids, and four injections of gray oil sufficed to relieve him. Six weeks later another acute relapse was equally readily cured. (He has remained well one year.)

Case VI showed a similar toxemia, purely syphilitic in nature until superseded by the iodid toxemia.

A fourth type is that of the sufferer from arteriosclerosis or visceral sclerosis (under the clinical type of hepatic cirrhosis or chronic interstitial nephritis); a fifth, that of amyloid disease of the viscera.

Of these five more or less empyric types of chronic toxemia,

only the second (syphilitic fever) and the two last require any special comment.

The Fever of Late Syphilis.—The fever of late syphilis is one of the most impenetrable mysteries of the disease. When we have said that its cause is unknown, its type chronic and irregular, its cure, mixed treatment, we have summed up almost all our knowledge on the subject.

The following conclusions, drawn chiefly from a *résumé* of Dr. Arthur Birt,[1] summarize the consensus of opinion upon this subject:

1. Late visceral syphilis is attended with persistent fever much more commonly than is generally recognized.

2. Though variable in type and sometimes accompanied by chills and night-sweats (simulating tuberculosis) or by chills and fever (simulating malaria or sepsis), it is usually a prolonged, low-grade fever which may last quite indefinitely.

3. Though usually accompanied by evidences of liver syphilis, these are rarely marked, sometimes absent (Janeway, D'Amato), and sometimes replaced by lung syphilis (Sobernheim).

4. Moreover, the fever, like the visceral lesions, always occurs years (sometimes fifteen or twenty) after the chancre.

5. Hence its nature may often be determined only by exclusion and by the "test course" of mixed treatment, though a careful history and physical examination reveal the nature of many cases.

6. Every atypical fever of unknown origin should, however, be considered suspect.

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[1] *Montreal Med. J.*, 1905, vol. xxxiv, p. 748. Other recent contributions are those of D'Amato, *Riforma med.*, 1906, vol. xxii, No. 9; Mannaberg, *Zeitschr. für klein Med.*, 1907, vol. xii; Klemperer, *Zeitschr., für klein Med.*, 1905, vol. iv; Sobernheim, *Therap. der Gegenwart*, 1905, vol. vii, p. 486; and Raubitschek, *Centralbl. f. d. Grenzgeb. der Med. u. Chir.*, 1907, vol. ix, p. 641.

7. Mild mixed treatment suffices for a cure and a severe course may do harm.

Arterial and Visceral Sclerosis.—That syphilis may cause arteriosclerosis and interstitial inflammations of the viscera has long been an accepted doctrine. That it often does cause them is extremely doubtful. Certainly the great majority of cases are not syphilitic, and equally certainly those patients who, having had syphilis, come to have sclerotic arteries or chronic interstitial nephritis or hepatic cirrhosis, are in no wise different from their non-syphilitic brethren. One may be tempted in a given case to believe that a prolonged, neglected syphilis must have had some share in the production of these lesions, while recognizing that such a theory has no clinical or therapeutic bearing upon the case except to spur one's efforts toward healing the syphilis if any trace of it persists.

Amyloid Degeneration of the Viscera.—Almost the same may be said of amyloid degeneration of the viscera. It may be caused by inveterate, neglected syphilis;[1] indeed, it may complicate syphilitic disease of the viscera. But amyloid degeneration of syphilitic origin is no different from amyloid disease of other provenance. Antisyphilitic treatment can only do it harm.

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[1] It is much more commonly due to tuberculosis or to sepsis.

CHAPTER XIX

SYPHILIS OF THE SKIN: GENERAL CHARACTERISTICS

DIVERS and dissimilar as are the skin lesions of syphilis (the syphilids, as they are more concisely termed), they have, nevertheless, certain general characteristics to which they adhere quite faithfully and which serve as diagnostic criteria. Indeed, a thorough familiarity with these general characteristics and with the way in which the various syphilids conform to them is of more service in diagnosis than any knowledge of individual types of eruption.

These general characteristics are clinical and pathological. The general clinical characteristics are the following:

GENERAL CLINICAL CHARACTERISTICS

1. Slow and progressive development.
2. Polymorphism.
3. Absence of local, general inflammatory reaction.
4. Absence of pain and itching.
5. Peculiar raw-ham color.
6. Rounded form.
7. Scales white, superficial, non-adherent.
8. Crusts greenish or black, thick, irregular, adherent.
9. Ulcerations rounded or circinate, with abrupt edges, sanious secretion and sluggish base.
10. Scars round, depressed, thin, non-adherent, smooth, often pigmented at first.

11. Early eruptions disseminated and profuse, later ones asymmetrical and grouped.

Slow and Progressive Development.—The earlier and more generalized the eruption the more rapidly does it develop. A general macular or papular syphilid covers the man in a few days; but a late tuberculo-ulcerative lesion may take weeks to develop its true character.

Much more striking is the progressive development of the lesions. With various degrees of speed, but, as a rule, slowly and gradually, the eruption spreads. The superficial general eruption spreads by the appearance of new lesions; the later single lesions by encroaching on the surrounding tissues.

Polymorphism.—The early, general eruption develops progressively in type as well as in multiplicity of lesions. Thus a general, papular eruption a week old may show macules, papules, tubercles, vesicles, pustules, and minute ulcers irregularly intermingled. Such is polymorphism. It is all but pathognomonic of syphilis.

The later the eruption the less polymorphic is it likely to be.

Absence of Inflammation.—Fever very rarely accompanies a syphilitic eruption. Exceptions, chiefly due to absorption from mixed infection of a pustular syphilid, are so few as to be negligible.

Neither is tenderness, heat, nor inflammatory congestion discernible in the lesion itself. It develops, as the French say, *à froid*.

Absence of Pain and Itching.—The syphilitic lesion as such is painless. In those exceptional instances (and they are singularly rare) of mixed infection with the ordinary pyogenic microbes, the pain is due to the adventitious inflammation.

Moreover, the syphilid does not itch. A little tingling may accompany the relatively rapid development of a diffuse exanthem, but even this is rare and does not amount to a real itch.

But confusing complications are many. A *seborrheal scalp* is always rather itchy, and a syphilitic eruption upon it may render this tendency more pronounced. Hence slight itching in an eruption of the scalp must always be discounted.

Parasitic eruptions and *eczema* are not the less pruriginous for being associated with syphilis, and a skin which is normally irritable and itchy is no less so when covered with a syphilid.

Peculiar Color.—The color of the syphilids is not a frank, inflammatory red, but a vinous, empurpled redness, resembling, when well marked, the raw meat of ham. This color is found also in many of the gouty, papular eruptions and in psoriasis, rarely in other eruptions. The color of the syphilids passes by pigmentation from this dusky red into a yellowish copper color, and sometimes by a deep pigmentation to brown or black, the skin around the lesion (areola) being usually also pigmented to a certain extent. This pigmentary coloration sometimes lingers for years, but usually clears off after a few months, disappearing first centrally, then peripherally. Finally, the spot becomes brilliantly white.

Rounded Form.—Every syphilid is composed of circular lesions. If discrete, as in most of the earlier eruptions, the individual lesions are manifestly round. But many of the later lesions are confluent. Either because the lesions begin so close together or because in spreading they invade the same territory, the resultant lesion is a composite one, and this may show a *circinate*, polycyclic edge, which is very characteristic.

Moreover, although the disposition of the first general eruptions is diffuse and irregular (though symmetrical), *the lesions composing later eruptions are often distributed in circles* or in segments of circles.

Finally, certain of the more chronic syphilids as they progress extend in every direction toward the periphery, healing at the same time in the center. Hence result lesions of a cir-

cular or circinate or horseshoe shape quite pathognomonic. Such eruptions are called serpiginous.

The Scale.—The scales on the cicatrices and on the patches of scaly, syphilitic eruptions are thin, white, non-adherent, lamellar; very different from the dense, thick, imbricated, adherent scales of psoriasis.

The Crust.—The scabs formed on syphilitic ulcerative, rupial, and pustular lesions are rough and adherent, dark brown, or greenish black, sometimes loosened by an underlying accumulation of pus, but more often seemingly set into the skin, and tightly adherent. They may be of light color over secondary pustular lesions, but, light or dark, the green hue is rarely totally absent and is often brilliantly marked.

The Ulceration.—With the exception of the chancre and of the ulcerated mucous papule (both of which may vegetate and are usually elevated rather than depressed), the ulcerations of syphilis resemble chronic, indolent ulcers. They are rounded or oval, with abrupt edges cut away like those of a chancroid; the base is covered with yellowish, false membrane, sometimes bluish, like boiled sago. The edges and base of the ulcer are usually hard, and the former generally, but not invariably, firmly adherent and not undermined, as in the ulcerations of tuberculosis. These ulcers do not bleed easily, are generally atonic and sluggish, and usually entirely painless. Apparent exceptions to the rule in regard to pain are often due to the dependent position or other cause calculated to excite inflammation, or to the situation of the ulcer over a bone, the periosteum of which is inflamed and painful.

The Scar.—The cicatrices of such syphilitic lesions as have destroyed tissue (i. e., tertiary lesions), whether there has been surface ulceration or not, are rounded, very thin,[1] depressed, smooth, shining, and non-adherent. They are *usually at first*

[1] Marked only with the slightest irregularities (like cigarette paper).

uniformly pigmented of a dark-brown hue (nearly black in brunettes). This pigment clears off from the center to the circumference until only a dark border is left, which sometimes lasts for years, but finally the whole cicatrix acquires an almost pearly whiteness (though the pigmentation may persist indefinitely).

Cicatrices over bone adhere if they have been connected with bone lesions. The cicatrices left by an ulcer partaking of the nature of both syphilis and tuberculosis are often complex; i. e., the scar is irregular, uneven, bridled on its surface, contracted in parts, adherent at points, not much pigmented—possessing, in a word, the characteristics of a tubercular cicatrix rather than those of syphilis. Such complex cicatrices are seen where syphilitic glands have undergone tubercular caseation.

GENERAL CLINICAL CHARACTERISTICS OF SECONDARY AND TERTIARY SYPHILIDS

Certain general clinical characteristics distinguish the secondary from the tertiary syphilids.

The *secondary lesions* are:

1. Superficial, benign, resolutive.
2. Multiple, profuse, or even generalized.
3. Irregularly disseminated, but usually symmetrical.
4. Polymorphous.
5. Curable by mercury.

The *tertiary lesions* are:

1. Deep, destructive, and malignant.
2. Few in number, often single.
3. Distributed in circles or segments of circles, and usually asymmetrical.
4. Usually monomorphous.
5. Usually, though not always, require mixed treatment for a cure.

1. The secondary lesions are superficial, benign, and resolutive; the tertiary lesions, deep, destructive, and malignant. These characteristics are the common distinguishing ones between all secondary and all tertiary lesions, whether in the skin or elsewhere. But the distinction is peculiarly marked in syphilis of the skin. No contrast could be greater than that between a general roseola, for instance, which covers the trunk with a blush that lasts only a few days and disappears spontaneously, and an ulcerative lesion driving deep through the skin and into the subcutaneous tissue, advancing slowly and impartially in every direction, destroying the skin before it, and showing no tendency to recover spontaneously.

It must not be forgotten, however, that certain secondary lesions, notably the scaly ones, not only fail to get well spontaneously, but are very rebellious to treatment; while, exceptionally, one encounters a patient whose tertiary lesions, long neglected, have finally healed spontaneously, though, as a rule, this healing of one lesion is accompanied by the outbreak of others.

2. The secondary lesions are multiple, profuse, or even generalized, while the tertiary are few in number and often single. This rule must be accepted broadly. The average tertiary syphilid is made up of fewer lesions than the average secondary lesion. Yet a single papule may be no less secondary for being unique; while multiple gummata of the skin lose none of their tertiary malignancy through being numerous; and an early general tubercular syphilid is tertiary in spite of time and multiplicity.

3. The secondary lesions are irregularly disseminated, but usually symmetrical. The tertiary lesions are distributed in circles or segments of circles and usually are asymmetrical.

The elements of a generalized secondary syphilid, though symmetrically distributed in that they affect similar parts of the body on opposite sides, are not grouped in any geometric form; and the later, less generalized, secondary lesions, though

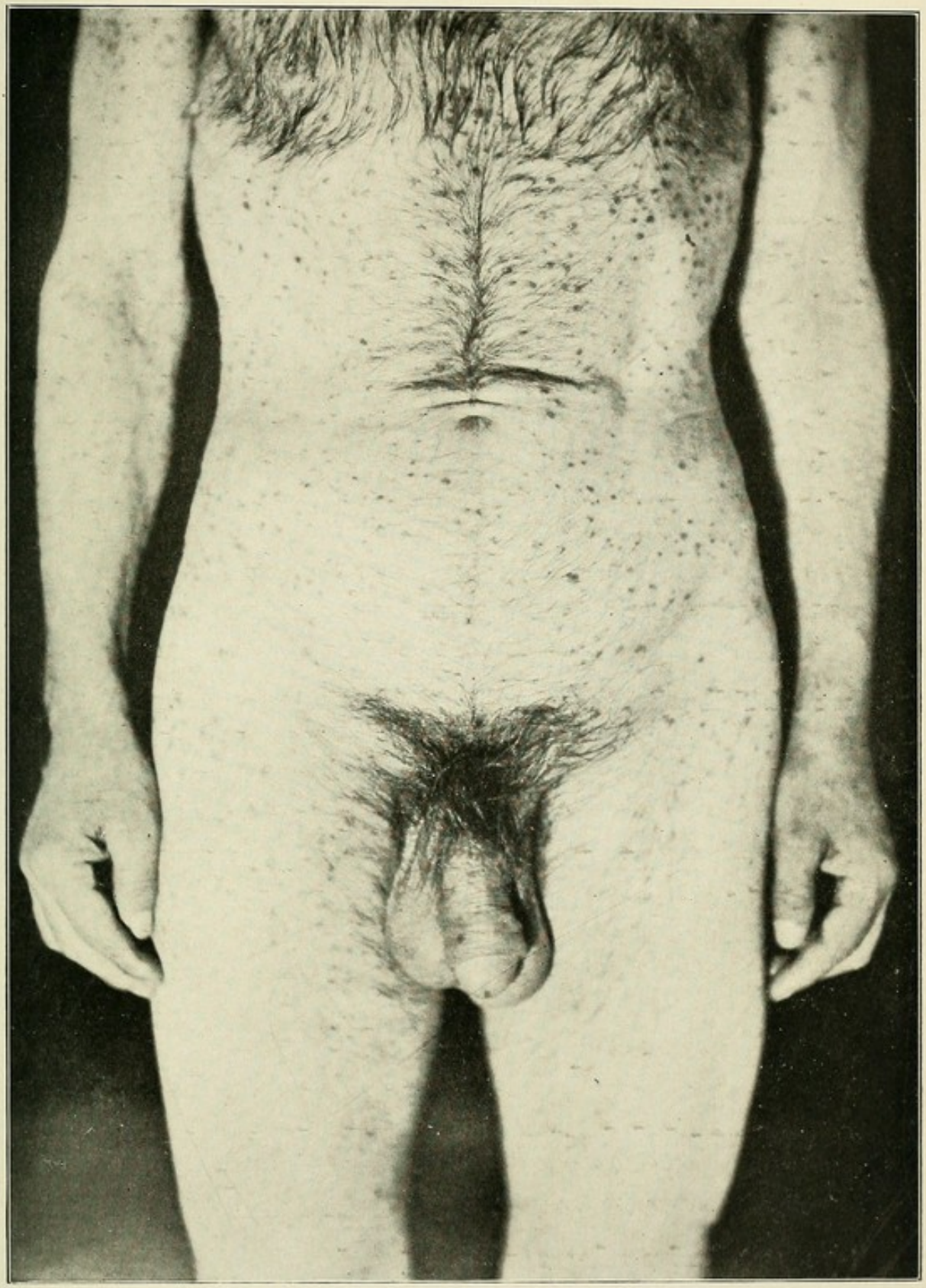

PLATE VI.—The Onset of Syphilis.

Chancre on left side of foreskin, adenitis in left groin, maculo-papular (polymorphous) eruption. (Piffard.)

they tend more and more to group themselves in circles, are usually quite irregularly distributed. Tertiary lesions, on the other hand, whether (as already described) because of the original distribution of the lesions or because of their destructive advance from the center outward, adhere closely to the circular or polycyclic type; and, though tertiary syphilids occurring within the first two or three years of the disease are often symmetrical, the later the lesion the less is this symmetry to be looked for, and, generally speaking, it is not characteristic of the tertiary syphilid.

4. The secondary lesions are polymorphous; the tertiary lesions, monomorphous. This, again, is a relative distinction, for nothing could adhere more closely to a single type than (for example) the typical squamous secondary syphilid of the palm, while, exceptionally, various tertiary lesions appear simultaneously and even coincide with secondary lesions in other parts of the body.

5. The secondary lesions are curable by mercury, while the tertiary lesions usually, though not always, require mixed treatment for a cure. This rule, as far as the secondary syphilids are concerned, is practically universal: one does not see them cured by iodid.

But in the treatment of tertiary syphilids it is often difficult to determine beforehand which of the two drugs, iodid or mercury, will effect a cure. Phagedenic or gummy lesions are usually to be attacked by iodid. This checks their advance, and heals them partially or wholly, but to maintain the cure mercury must be administered. On the other hand, the more superficial ulcerative and tubercular lesions often do better under high doses of mercury, though here the rule is by no means universal. I have seen similar lesions in different patients get well; in the one under iodid after mercury administered for many months had failed; in the other, under mercury after iodid administered for several years had failed.

The best rule is to give mixed treatment. If this, in moderate doses, fails to cure, push the one drug to the point of toleration, and then, dropping that, push the other to the point of toleration. Thus you will find which may be expected to effect the cure.

GENERAL PATHOLOGICAL CHARACTERISTICS

We need not repeat what has been said in a previous chapter concerning the general pathological characteristics of syphilis. Suffice it to say that, in the skin the papule is the type of all secondary syphilids, the tubercle that of tertiary syphilids, and the gumma that of subcutaneous syphilis.

The *syphilitic papule* is an exudation beginning in a papilla of the derma. "The papule is prominent because there is a cellular infiltration, hard because this infiltration is dense, brilliant because the epidermis is tense over the summit, surrounded by a scaly collar because the epidermis breaks under the effect of this tension, red because of extravasated blood."

The mucous layer of the skin is thickened by infiltration, the papillæ are hypertrophied, and the epidermis is desquamated or, in the squamous papule, thickened. The dilatation and obliteration of the vessels, the infiltration of their walls, and the perivascular exudations extend well into the subcutaneous connective tissue. Indeed, the prominence of the papule above the surface is due chiefly to the subdermal congestion and edema. Fig. 9 will serve to illustrate the pathology of the syphilitic papule as well as the chancre.

The *macular syphilid* might be described pathologically as an aborted papular eruption. There is congestion from dilatation of the blood vessels and slight exudation; but, unless the macula is transformed into a papule, no marked exudate occurs.

If the papule becomes *squamous*, the essential change is a

proliferation and desquamation of the epidermis. If it becomes *vesicular*, the epidermis is raised by serum exuded into the lesion. If *pustular*, this vesicle is filled with the necrotic tissue and pus cells.

The *syphilitic tubercle*, like the papule, is an infiltration of the derma, but it is more intense and deeper. The exudate, instead of being confined almost exclusively to the papillæ, involves the whole derma and the subjacent connective tissue. The syphiloma resides in the true skin instead of on it. Hence the healing is accompanied by loss of tissue in the skin, and leaves a permanent defect, a typical syphilitic scar (Fig. 17).

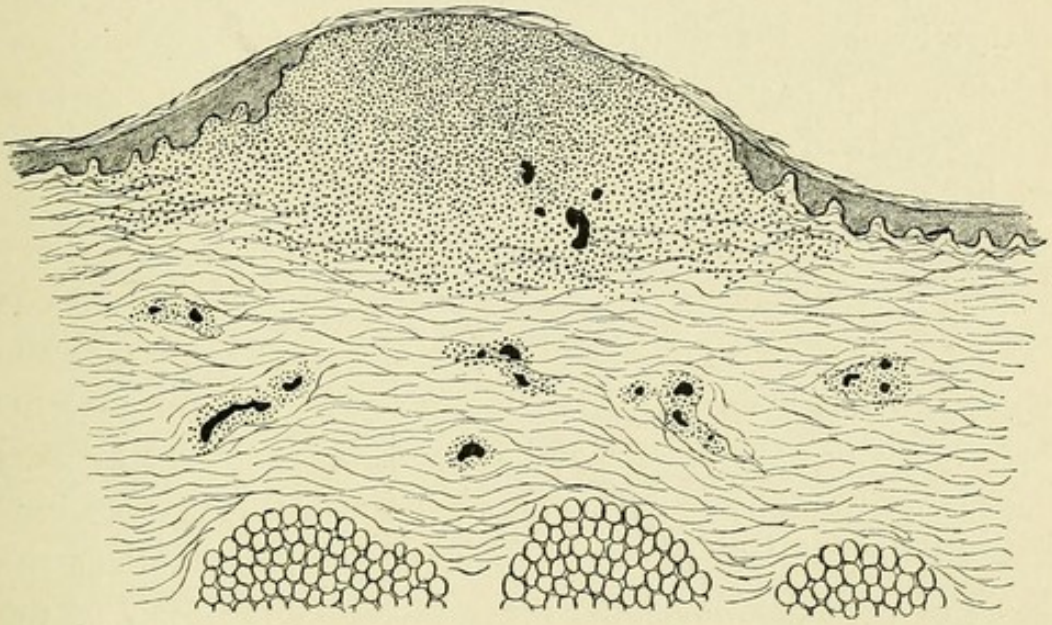

FIG. 17.—PATHOLOGY OF THE SYPHILITIC TUBERCLE (diagrammatic). The lesion, though covered with epithelium and not ulcerated, has destroyed the true skin and will leave a scar. Compare Figs. 10 and 36.

The *subcutaneous gumma* is the same lesion removed to a still deeper plane. Instead of a primary focus in the skin with secondary involvement of the subcutaneous tissue, there is primary involvement of the subcutaneous tissue and secondary extension to the skin. Vascular dilatation and infiltration, interstitial exudation, and, finally, atrophy and caseation are the common lesions of all gummata.

CHAPTER XX

SECONDARY SKIN SYPHILIDS

In describing syphilis of the skin it is more convenient to group together those lesions which affect the general integument and to separate in another chapter those lesions, such as mucous papules, condylomata, eruptions of the scalp and onychia, that are peculiar to special parts of it.

The division of skin syphilis into secondary and tertiary lesions is relatively unsatisfactory, inasmuch as certain lesions belong to an intermediate indeterminate class. They have some characteristics common to the secondary lesions, others common to the tertiary lesions, and are sometimes curable by mercury, sometimes by iodid. Yet, on the whole, it is more convenient to maintain the distinction, taking the papule as the basis of secondary syphilis, the tubercle as the basis of the tertiary lesions.

The secondary syphilids affecting the general integument may therefore be discussed under the following heads:

Macular syphilids.

Papular syphilids.

Vesicular syphilids.

Pustular syphilids (including crusted and ulcerating lesions).

Squamous syphilids.

Pigmentary syphilids.

THE MACULAR SYPHILIDS

The important macular syphilid is that general, macular eruption or roseola which is habitually the first seen of the secondary eruptions of syphilis (Fig. 18).

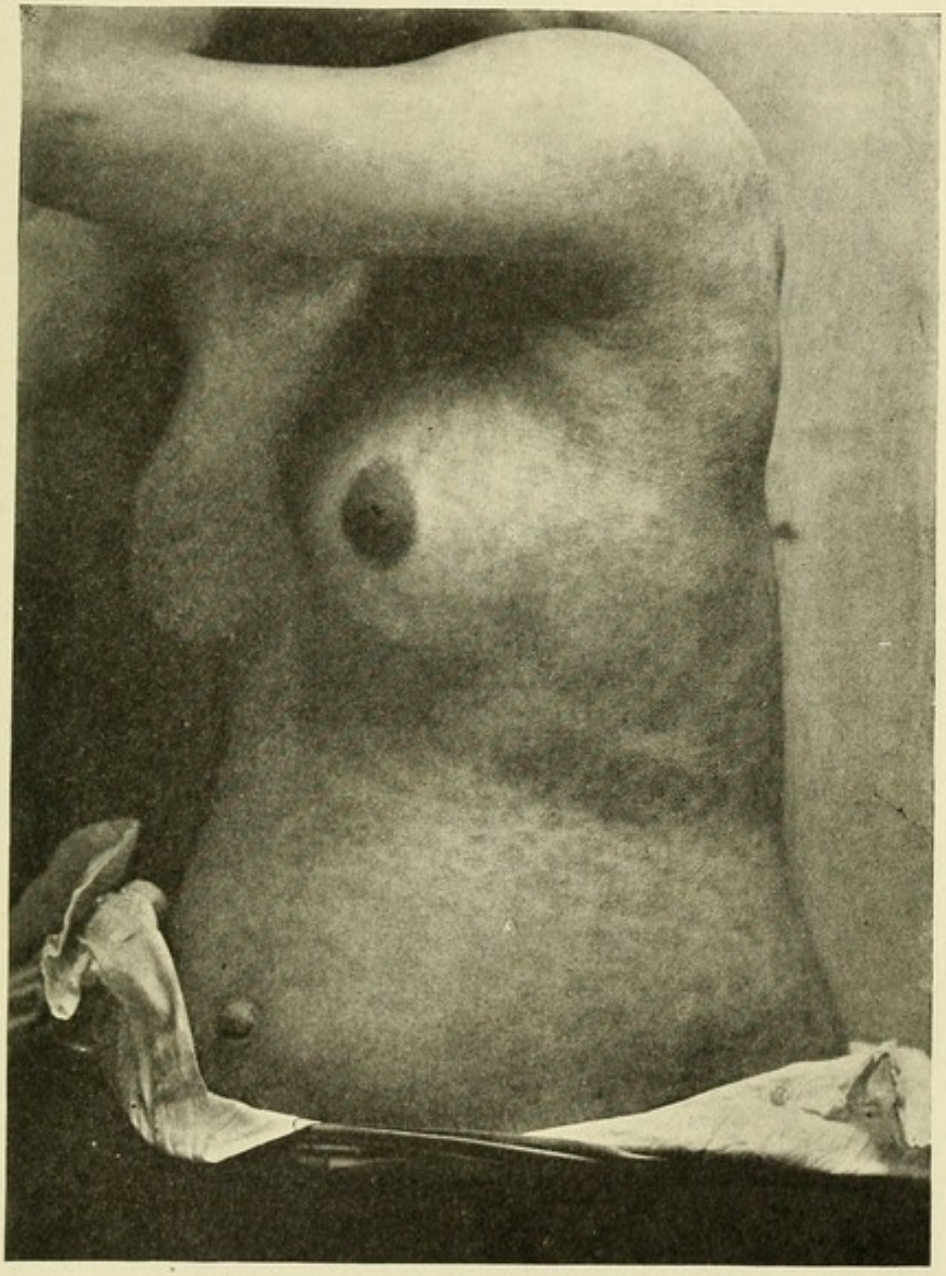

FIG. 18.—MACULAR SYPHILIS. (FOX.)

Regions Involved.—The eruption first appears upon the loin or flanks and the lateral thoracic regions. Thence

it extends all over the trunk, and to a less degree to the extremities affecting chiefly their flexor surfaces. The head and hands and feet, are, happily, almost always spared.

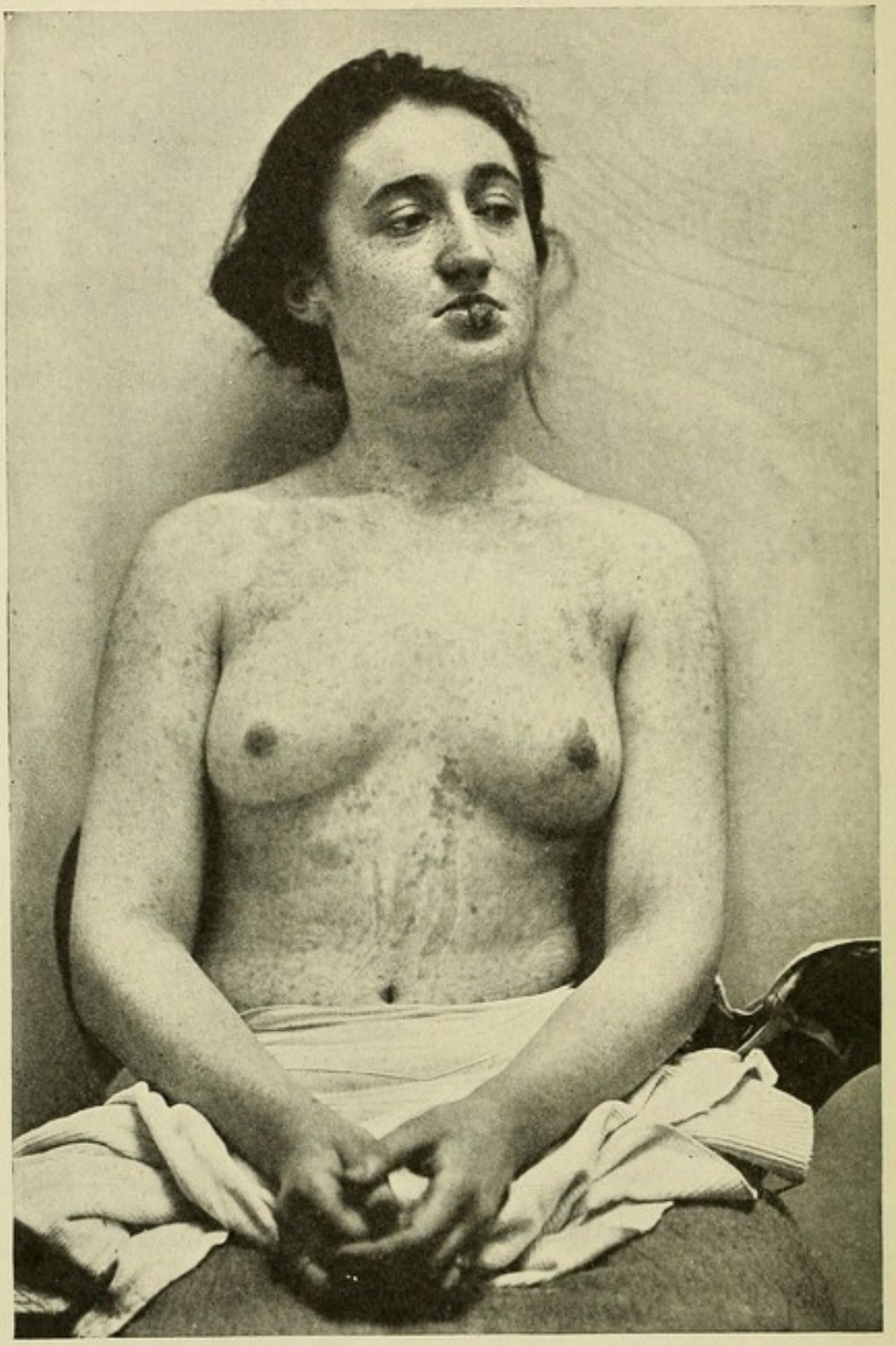

FIG. 19.—LABIAL CHANCRE AND MACULAR SYPHILID (FACE FRECKLED, BUT FREE FROM ERUPTION). (FOX.)

Character of the Eruption.—The roseola is often so faint as to be overlooked by the patient. I have several times had a physician bring for diagnosis a patient covered with macules

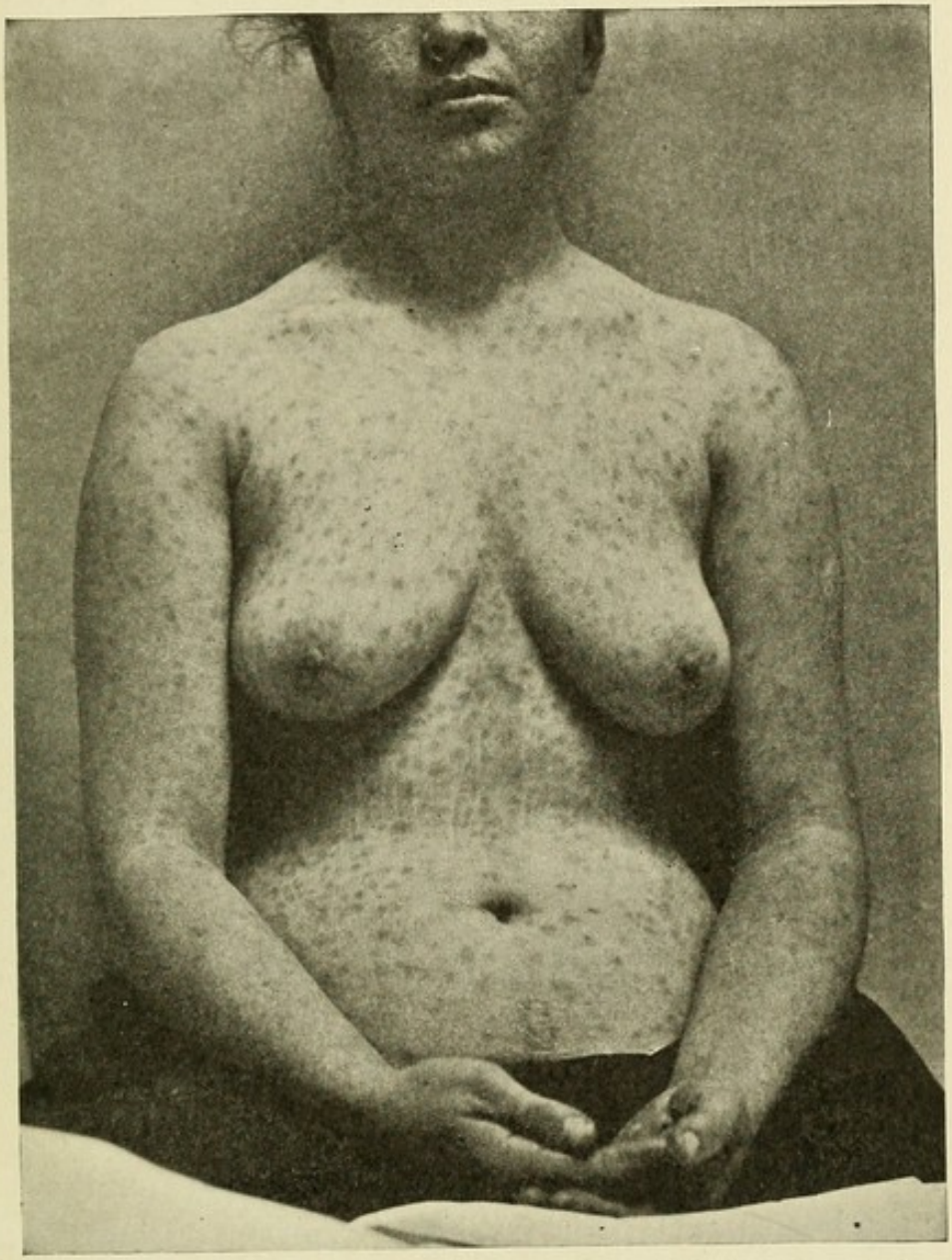

FIG. 20.—MACULO-PAPULAR (POLYMORPHOUS) SYPHILID. (FOX.)

which neither the victim nor his doctor had been able to distinguish until I pointed them out. But usually the lesions persist, become more and more evident, and at last are unmistakable even by the patient.

The eruption is made up of small blotches, varying in size, circular or crescentic in form, with slightly indented margins. The average size is that of a split pea. Exceptionally, the macules are very small or they may be an inch across. Macules of various sizes appear on the same individual. Their color is rosy and not at all a ham color unless they have existed for some time, when they may take on a certain yellowish pigmentation. They disappear on pressure unless this pigmentation has occurred, in which case, after pressure, they appear as light yellow spots. On healing they leave a light grayish mark if they have been pigmented. Desquamation is rare.

Varieties. — Polymorphous Eruption. — This is the most important variation of the simple roseola. Many cases begin with roseola, and before the macules disappear a certain number of them become papular, and gradually the macular gives way to a papular eruption (Figs. 20, 21).

Meanwhile the papules themselves may be undergoing further transformation into tubercle or pustule.

Elevated Macule.—In some cases there is sufficient exudation to cause a distinct elevation of the macules above the surrounding skin. This can be readily discerned by throwing a strong light at an acute angle upon the affected surface.

Late, Circinate, Macular Syphilid.—At various periods during the course of the disease the macular syphilid may relapse; though such relapses are extremely rare, they are none the less characteristic. When they occur during the first year they may be quite diffused, but in later years the macular syphilid assumes a distinctly circinate form; it is, as it were, the shadow of a circinate tubercular lesion. I have four times seen such an eruption in the third year of the disease. In one patient the spots occurred upon the arm, replacing a sunburn.

Duration.—The initial roseola covers the trunk within two or three days of its inception, and, if untreated, lasts from two to six weeks; but under treatment, it rapidly disappears. In many cases the roseola, if untreated, merges into the succeeding papular eruption.

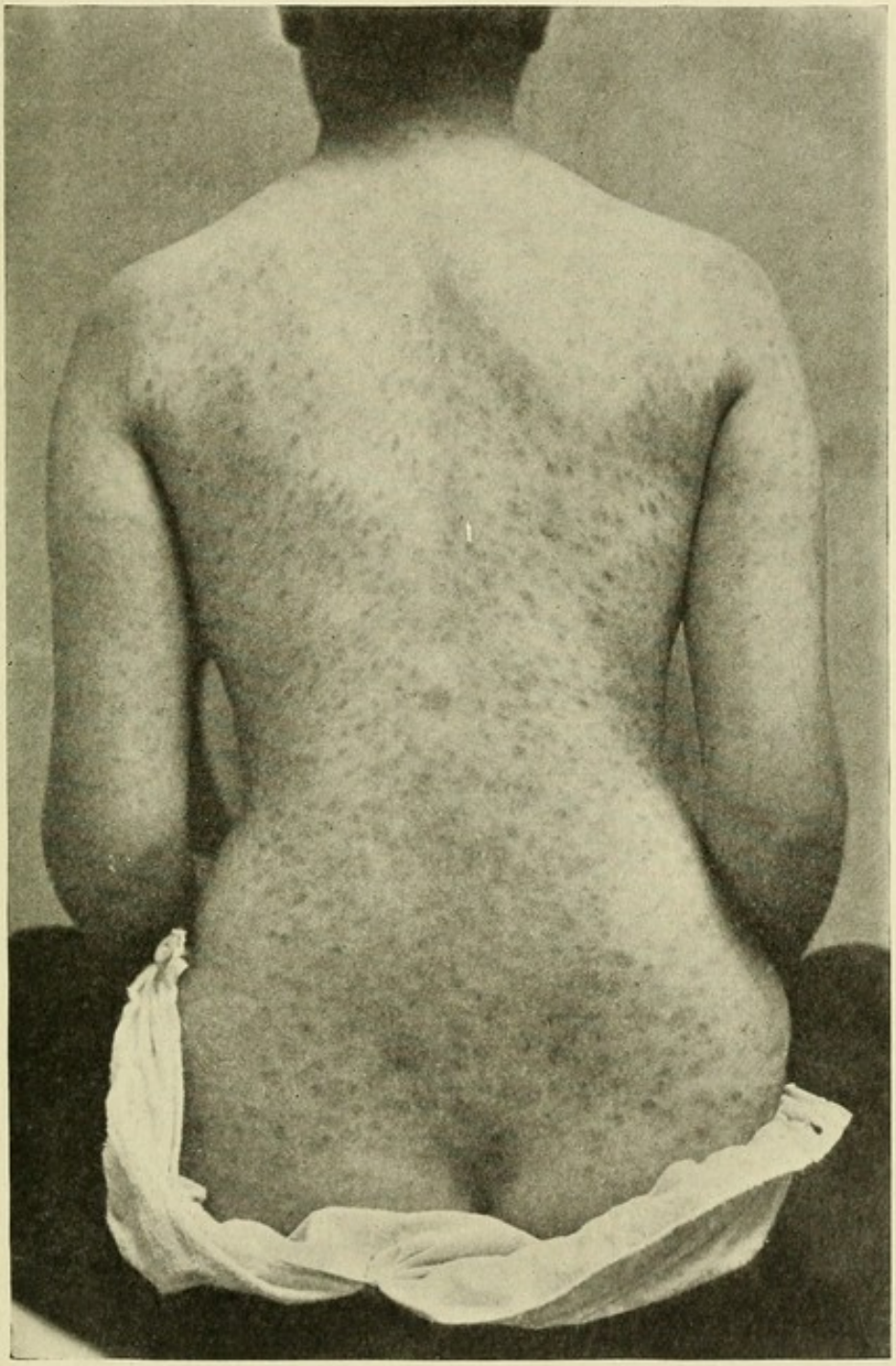

FIG. 21.—BACK OF CASE SHOWN IN FIG. 20. (FOX.)

Though the late circinate macular syphilid is usually much more tenacious than the earlier eruption, it disappeared rapidly in three of my cases, while the fourth lasted a year.

Diagnosis.—The distinguishing characteristics of the general macular syphilid need no further description. If the eruption is faint or has just begun to appear, it may be much better distinguished by looking at it through a cobalt-blue glass. The best light for this examination is a moderate daylight, and the glass should be held close to the eye in order to cut out extraneous rays.

The diagnosis of syphilis should be further enforced by the discovery of the glands and perhaps the induration remaining at the site of the primary lesion, while concomitant lesions inside the mouth, epitrochlear or posterior cervical nodes, or other symptoms of the onset of syphilis may be found (Fig. 19).

The diagnosis of syphilis may not be made on the roseola alone, any more than on the chancre alone.

Differential Diagnosis.—The roseola may be distinguished from that of *measles or scarlatina* by the absence of fever and by the concomitant syphilitic lesion (of the upper air passages, etc.).

Drug eruptions, notably those due to copaiba and antipyrin, are confluent, inflammatory and itching, usually associated with urticarial lesions, variously distributed, and due to the taking of the drug, of which a history may be obtained.

Simple, or springtime roseola itches, usually affects the face, is accompanied by fever and digestive disturbances, and appears and disappears within a short time. Macules due to various *parasites* are irregularly distributed and intensely itchy. The *natural marbling of skin* due to cold or the *flush of shame* can scarcely be mistaken for syphilitic roseola, while the itching wheals of *urticaria* are equally distinctive.

THE PAPULAR SYPHILIDS

The Papule.—The syphilitic papule is very slightly elevated above the surface of the skin. It varies from the size of the head of a pin to that of a split pea. Its summit is flat,

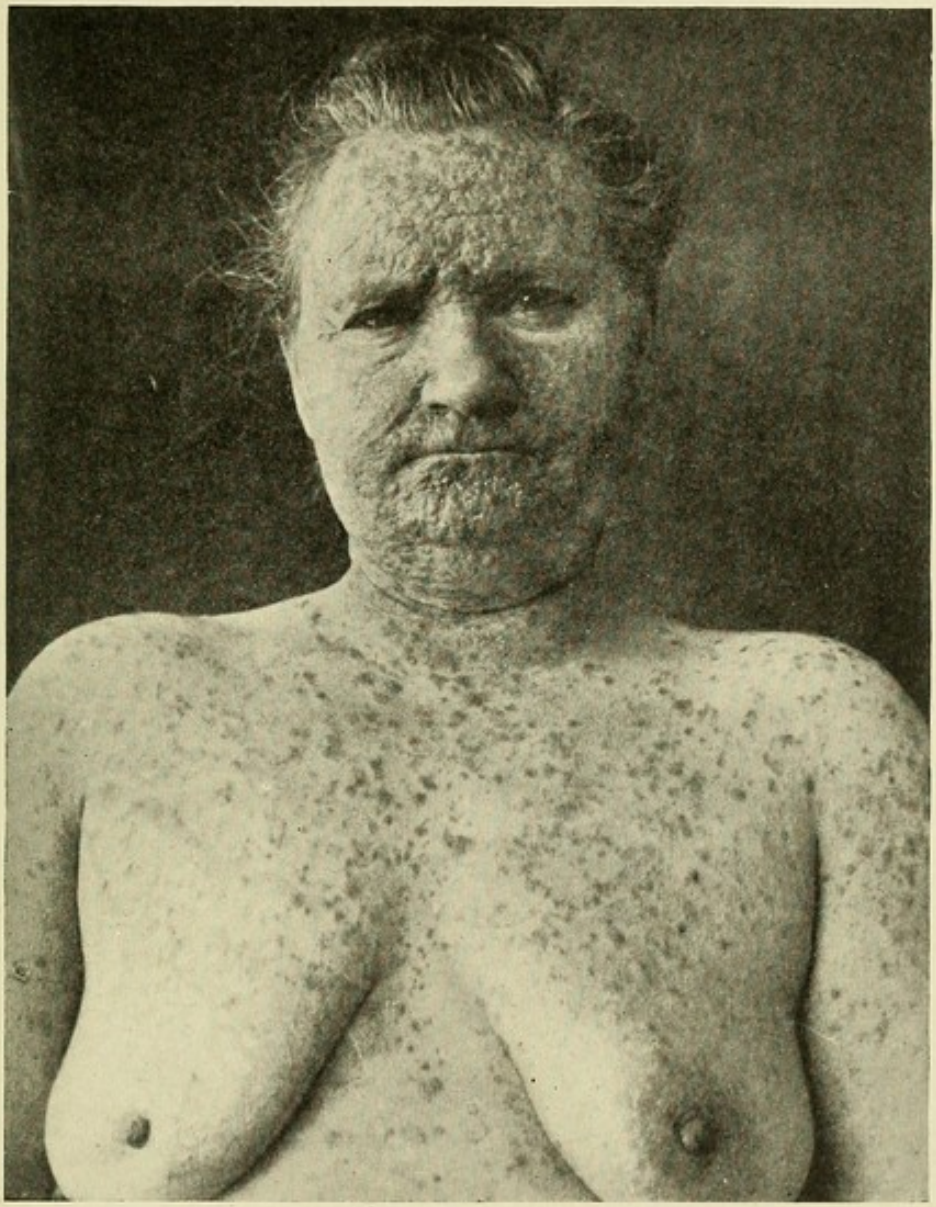

FIG. 22.—PAPULAR SYPHILID: CONFLUENT ON FACE. (Fordyce.)

though, if the papule be very small, this feature may be lacking. Its shape is absolutely and characteristically circular; its color, at first pink, soon takes on a purplish, ham or cop-

per hue although quite frequently this is not marked, but may be intensified by pressure. Its surface is shining and tense, and this may be covered by a slight, thin scale, or, more com-

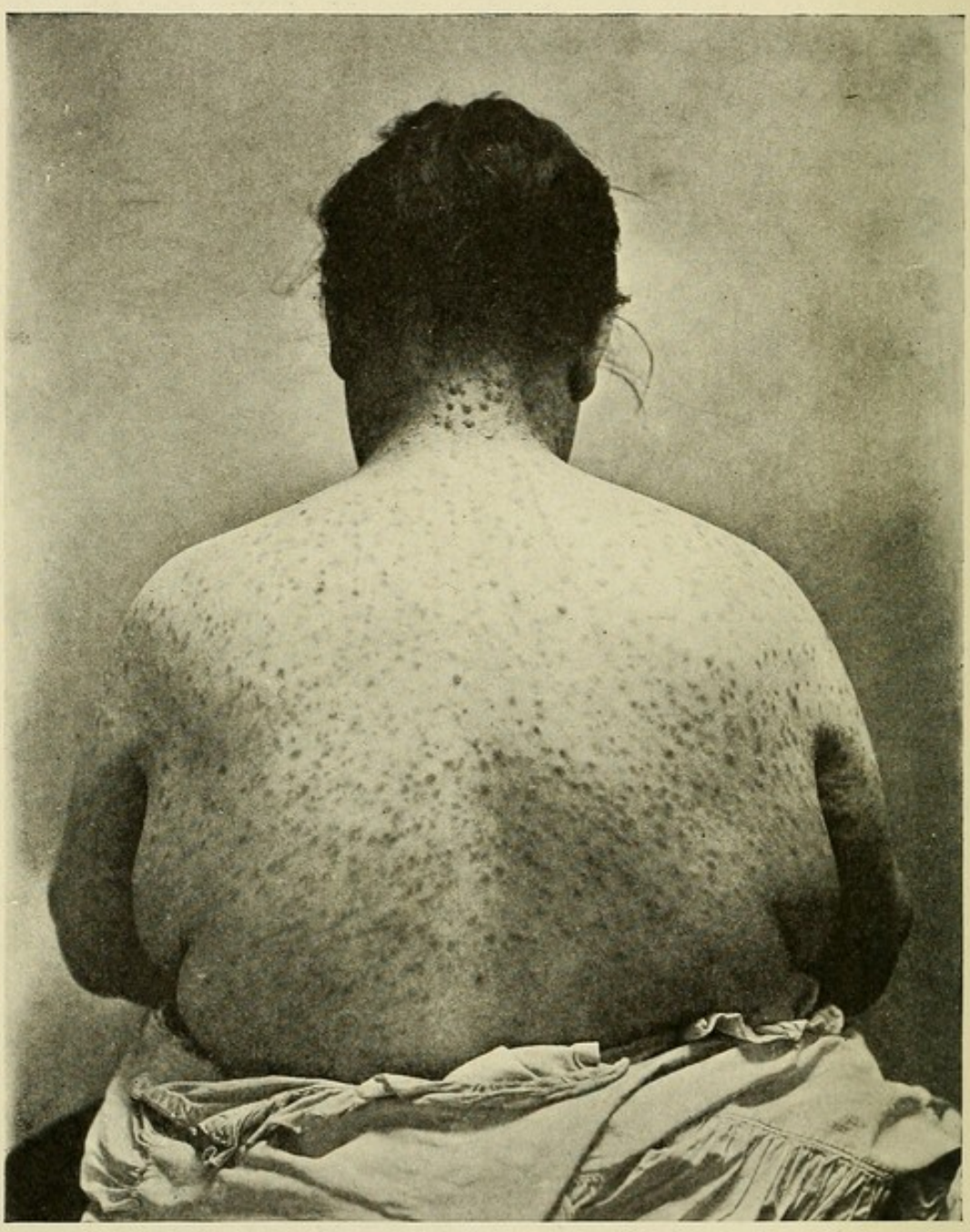

FIG. 23.—PAPULAR SYPHILID, SHOWING MARKED PREDILECTION FOR THE BACK OF THE NECK. (FOX.)

monly, this scale is promptly shed, leaving a faint, scaly collar around its base. The papule is hard, and has in some measure

the induration characteristic of all syphilitic exudates. From the papule all the other elements of secondary skin syphilis arise, and in the earlier, papular eruptions, it is not uncommon to find admixtures of pustules, squamous papules, etc.

Papular Eruptions.—The papular syphilid may be general and disseminated or partial and scattered. In a certain proportion of cases the general macular or papular eruption is preceded by the appearance of a few scattered papules. As a rule, however, the first papular eruption is generalized. It begins with the appearance of scattered papules over the body, and these gradually multiply until, at the end of a week or two, they cover the whole surface of the body, the trunk, the head, the face, and the extremities. The papules show

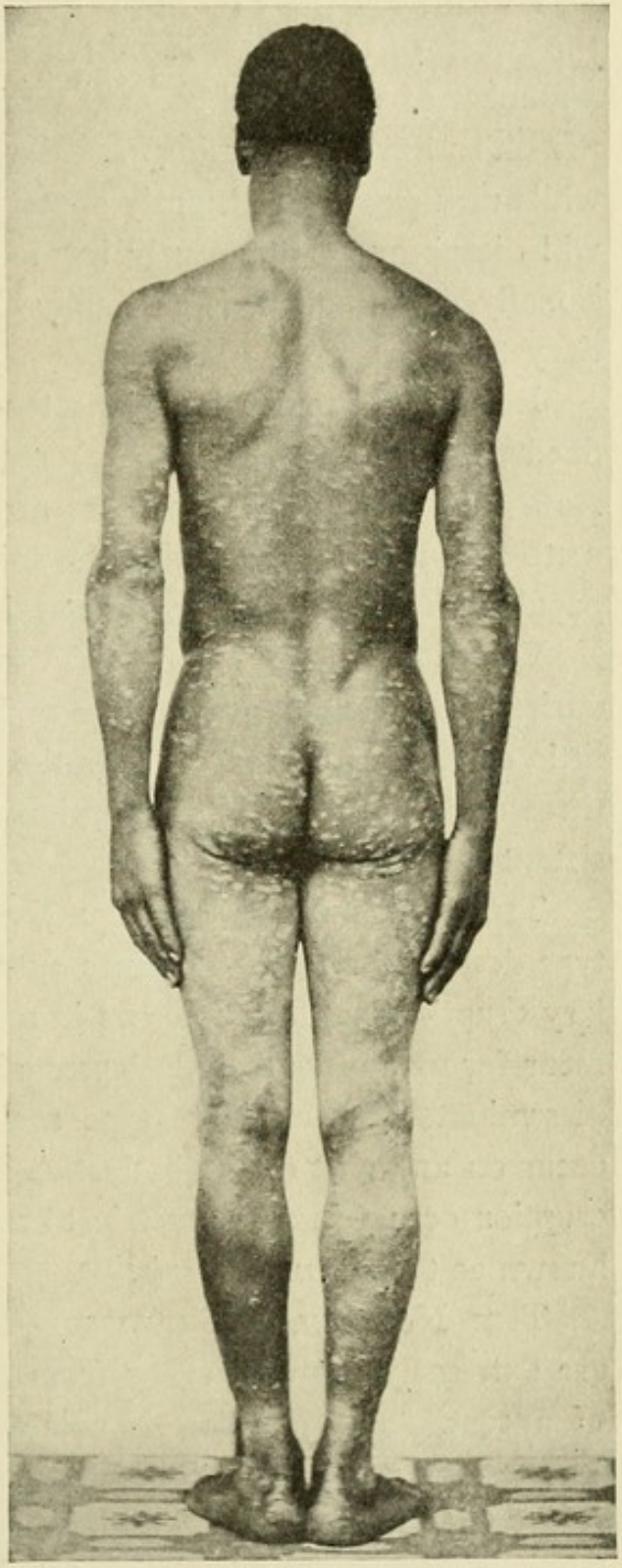

FIG. 24.—PAPULAR SYPHILID ON A NEGRO. (FOX.)

no tendency to grouping, but are distributed broadcast, though in the costal region they are in some measure aligned with the sweep of the ribs. If untreated the eruption remains for a number of weeks and becomes more and more polymorphous, the older lesions becoming pustular, squamous, or tubercular, while new papules constantly appear. Ultimately the eruption will disappear spontaneously in most instances, though, occasionally, if untreated, it gradually changes to a persistent tertiary type.

Relapsing papular eruptions tend to be more closely confined to certain (indeterminate) regions the later they occur. As a rule, however, the papules are not grouped in any geometric figure; they remain disseminated. Even those later papular eruptions are often bilateral and symmetrical.

Varieties.—The Miliary Papular Syphilid.—In this variety the papules are minute and pointed and grouped in little colonies scattered all over the body. It develops within three or four days. This eruption is very rare and quite resistant to treatment.

The Lenticular, Papular Syphilid.—This is the usual type described above. It comes out more slowly than the miliary eruption, and often occurs in successive crops, which may recur for many months. Relapses of this eruption of a partial, disseminated, or circinate type are very common, and may occur on any part of the body. A peculiarly striking papular eruption occurs in a symmetrical band across the brow, and is known as the *corona veneris*.

The Nummular Syphilid.—Exceptionally the papules are extremely large, varying from the size of a dime to that of a cent. The eruption is a late one, and often circinate. The lesions are often umbilicated and may be covered with a scale which adheres in the center and is detached at the edges. These nummular patches may become annular by healing at the center and leaving a persistent indurated ring.

To distinguish between a nummular and a tubercular syphilid may be clinically impossible. The nummular eruption is a (clinically rare) link between the two types. But in healing the tubercle leaves a scar, the papule does not.

THE DIFFUSE INDURATION.—This, like the nummular syphilid, is an "intermediate" lesion. It consists in irregular, circinate areas of infiltration. It is rarely seen except about the mouth and nose (page 312).

THE CRUSTED PAPULE (see page 292).

THE MOIST PAPULE (see page 302).

THE SCALY PAPULE (see page 296).

Diagnosis.—Apart from the history and the other coincident lesions of syphilis (nodes, mouth lesions, crusted papules in the scalp), the distinguishing characteristics of the papular syphilid are:

Its Distribution.—General, relapsing, and polymorphous, or regional (diffused or circinate).

Its Character.—A shotty, dark-red, perfectly round, flat-topped, slightly scaling papule.

Differential Diagnosis.—The general papular syphilid must be distinguished from *lichen planus*. The papules in this disease vary greatly in size (in a given eruption), are not strictly circular in form, are often umbilicated rather than flattened, and may be covered with a striated scale. There is a certain tendency to linear distribution. Yet, on disappearing, the papules of lichen planus leave macules which may give a semblance of polymorphism to the eruption (though in lichen planus the macules are never intermingled with the papules), and the papules itch but little, with an occasional brilliant exception have a dusky hue, and may be grouped in a circinate way. The diagnosis may thus be extremely difficult, and may await the test of treatment or depend upon collateral evidence.

Psoriasis is closely mimicked by the squamous papular syphilid (page 298).

The corona veneris may be mistaken for *acne*, but the dusky color and shotty feel and flat surface of the syphilitic papules distinguish them from the pointed, tender, inflammatory acne lesions.

THE VESICULAR SYPHILIDS

The appearance of a vesicle on top of a syphilitic papule is usually but a step toward pustulation. Varicelloid and herpetiform syphilids are accordingly more appropriately classified as pustular syphilids.

The Bullous Syphilid.—The bullous syphilid or syphilitic pemphigus is the only lesion properly vesicular, and since it is extremely rare in acquired syphilis (some authors even doubting its existence) and quite common in the hereditary form of the disease, its description is more appropriate elsewhere (page 513).

THE PUSTULAR AND CRUSTED SYPHILIDS

The pustular syphilids are all (excepting the crusts in the scalp) rare secondary lesions. Pustulation is, generally speaking, excited by dirt and debility. It is, therefore, properly regarded as an evil omen.

The pustule is, as it were, superadded to a syphilitic papule and sits upon it, destroying but little tissue, and hence leaving little or no scar. Pustular lesions may be scattered among the papules of a polymorphic eruption, but the more notable pustular eruptions are those which follow given types and mimic certain non-syphilitic eruptions. Accordingly, we distinguish:

Herpetiform syphilid.
Varicelloid (or varioloid) syphilid.
Acneiform syphilid.
Impetiginous syphilid.
Ecthymatous syphilid.

Herpetiform Syphilid.—Extremely rare. Constituted by groups of minute vesicles, pustules, or crusted papules. The lesions lie very close together. They come out in successive crops.

Color and slowness of development may distinguish this lesion from true herpes, though the mimicry is sometimes perfect.

Varicelloid Syphilid.—Very rare. Occurs early in the disease. Upon the papules arise pointed, round, or umbilicated vesicles, surrounded at their base by a dark-red areola afterwards becoming brown. The contents of the vesicles quickly become purulent and dry up into a greenish brown, adherent crust. This scab falls in about a fortnight, leaving a purplish discoloration, which slowly disappears. There are usually but few spots of eruption scattered over the face, limbs, and body. Successive crops of vesicles may prolong the eruption for several months, and ordinarily some other syphilid coexists with it.

Diagnosis.—This lesion, occurring soon after a syphilitic fever, and itself perhaps accompanied by some rise of temperature from absorption, may lead to a diagnosis of small-pox. Such mistakes were numerous in other days when the pustular syphilid was more common,[1] for the eruption may precisely imitate that of variola. The mistaken diagnosis, whether with variola or with varicella, cannot long continue before the atypical course of the eruption and fever leads to a fruitful investigation of the patient's history.

Acneiform Syphilid.—This eruption occurs scattered over the scalp, the face, and the extremities, the lower rather than the upper—or it may cover the whole body. Each pustule is distinct, and out of most of them grows a hair. They are not prominent, usually small, often but little larger than a grain

[1] Cf. Morrow, *J. Cut. and Venereal Dis.*, 1886, March.

of millet. Each separate pustule rests on a syphilitic papule, which itself never suppurates. The pustule slowly grows, taking from two to three weeks to develop and harden into a dry

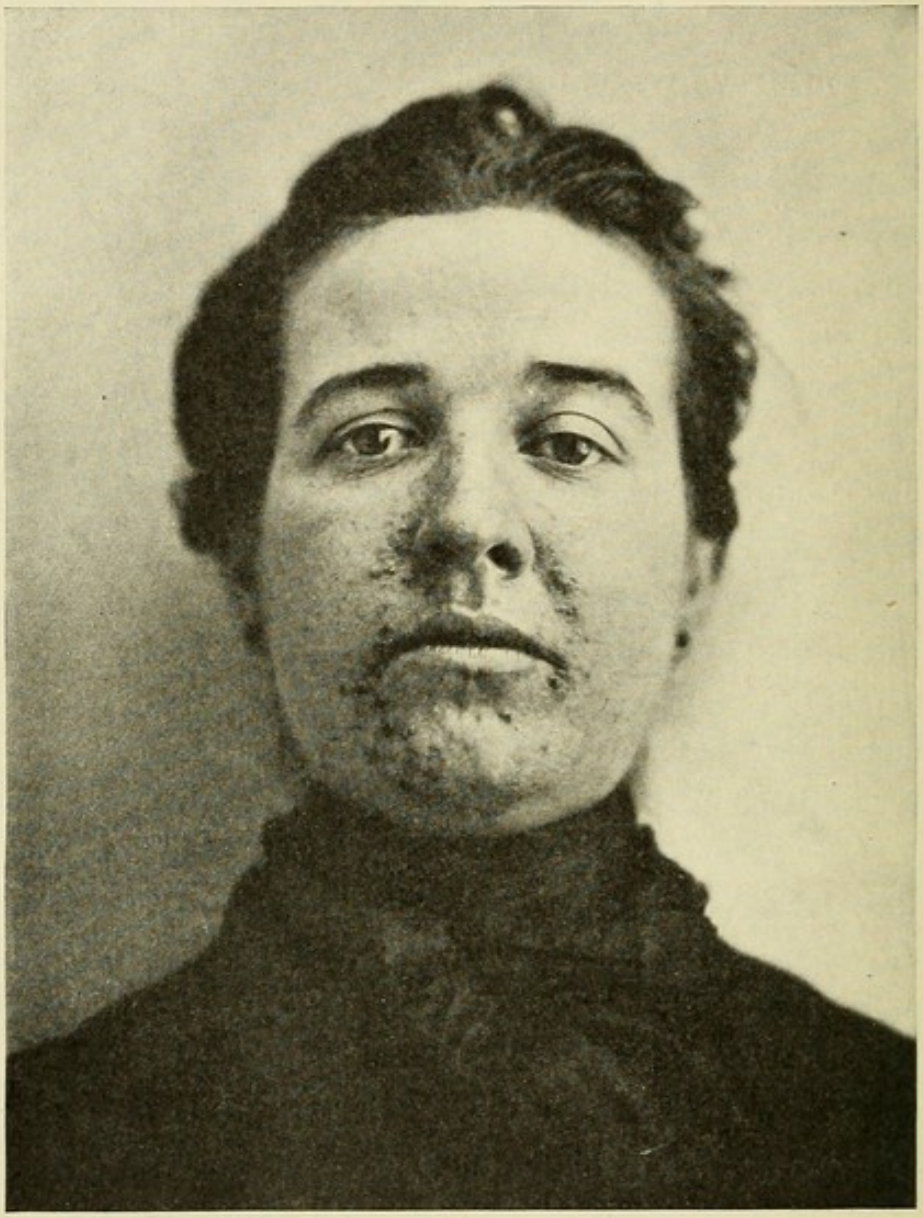

FIG. 25.—PAPULO-PUSTULAR (ACNEIFORM) SYPHILID. (FOX.)

scab (Fig. 25). The underlying papule has meantime been getting brown and becoming surrounded by a copper-colored areola. When the scab falls an umbilicated papule persists. This is gradually absorbed, leaving a purplish, pigmented discoloration, which is very slow to disappear. Sometimes a

slight, superficial ulceration remains. This is followed by a minute, round, white, depressed cicatrix, very different from the puckered scar of ordinary acne.

The acneiform syphilid of the scalp is one of the most constant early eruptions of syphilis. It corresponds with the papular syphilid elsewhere on the body. It has the general characteristics just described, except that the papule is relatively insignificant and the lesion is usually seen in the crusty stage, and is therefore familiarly spoken of as "scabs in the scalp."

Acneiform syphilid is rare elsewhere in the body and does not usually appear until several months after the first eruption. It tends to relapse.

DIAGNOSIS.—"Scabs in the scalp," if syphilitic, are thickest on top of the head, if due to *iodic or simple acne* are found only in the occipital region.

The acneiform syphilid is distinguished from *acne vulgaris* by its acute, transitory course, its irregular, disseminated distribution, its essentially papular character (while acne is essentially pustular), and by the intermixture of simple, syphilitic papules.

Iodic acne may simulate syphilitic acne, though the iodic pustules are usually large, sensitive, and inflammatory. It may be necessary to desist from administering the iodid in order to make sure of the diagnosis.

Impetiginous Syphilid.—Scattered pustules set on rather large syphilitic papules, surrounded by an areola, constitute this eruption. The pustules are superficial and soon dry to a granular, fragile, yellow, or greenish crust. The eruption occurs most frequently about the face and scalp.

DIAGNOSIS.—These lesions may be quite indistinguishable from *impetigo contagiosa* except by their less indolent course. But other syphilitic lesions almost always coexist and establish the diagnosis.

Ecthymatous Syphilid.—Syphilitic ecthyma may be secondary or tertiary in type. In the latter form the skin is destroyed by deep ulceration (page 323). In the superficial variety the lesions resemble those of the impetiginous syphilid, but are larger and more often found upon the lower extremities, the neck, the inguinal and gluteal regions. The crusts are also darker, denser, and more adherent. The ulceration, though superficial, is typically syphilitic.

Diagnosis.—*Ecthyma vulgaris* is a painful, inflammatory lesion, but in other respects is quite like the ecthymatous syphilid.

THE SQUAMOUS SYPHILIDS

Nearly all the eruptions of syphilis go through a desquamative stage; thus a patch of eruption which is essentially papular, tubercular, or pustular may finally become scaly, and, remaining so for a considerable time, pass for a squamous syphilid. So also does desquamation occur in syphilis, as of the scalp with early alopecia; sometimes in little patches along the margin of the scalp with the other syphilids; again, with syphilitic cachexia, furfuraceous desquamation of the scalp, or even of the whole body, may occur. But such desquamation is not an essentially syphilitic lesion; it may be induced by many other causes. There are, however, two varieties of essentially scaly syphilid. These are:

Generalized squamous syphilid.

Palmar and plantar squamous syphilid.

Generalized Squamous Syphilid.—This eruption occurs in two varieties—as a guttate or diffuse psoriasis and as a circinate eruption. The character of the eruption is the same in each. They may be seen together on the same subject. The patches vary from a split pea to a penny in size—or much larger in the circinate form—have, as a rule, the deep syphilitic color, and are but slightly elevated above the surface. The

scales are white, very fine, not adherent, not imbricated (as in true psoriasis) (Figs. 26, 27). After a few weeks the scales fall, to be replaced by others finer than the first, and thus

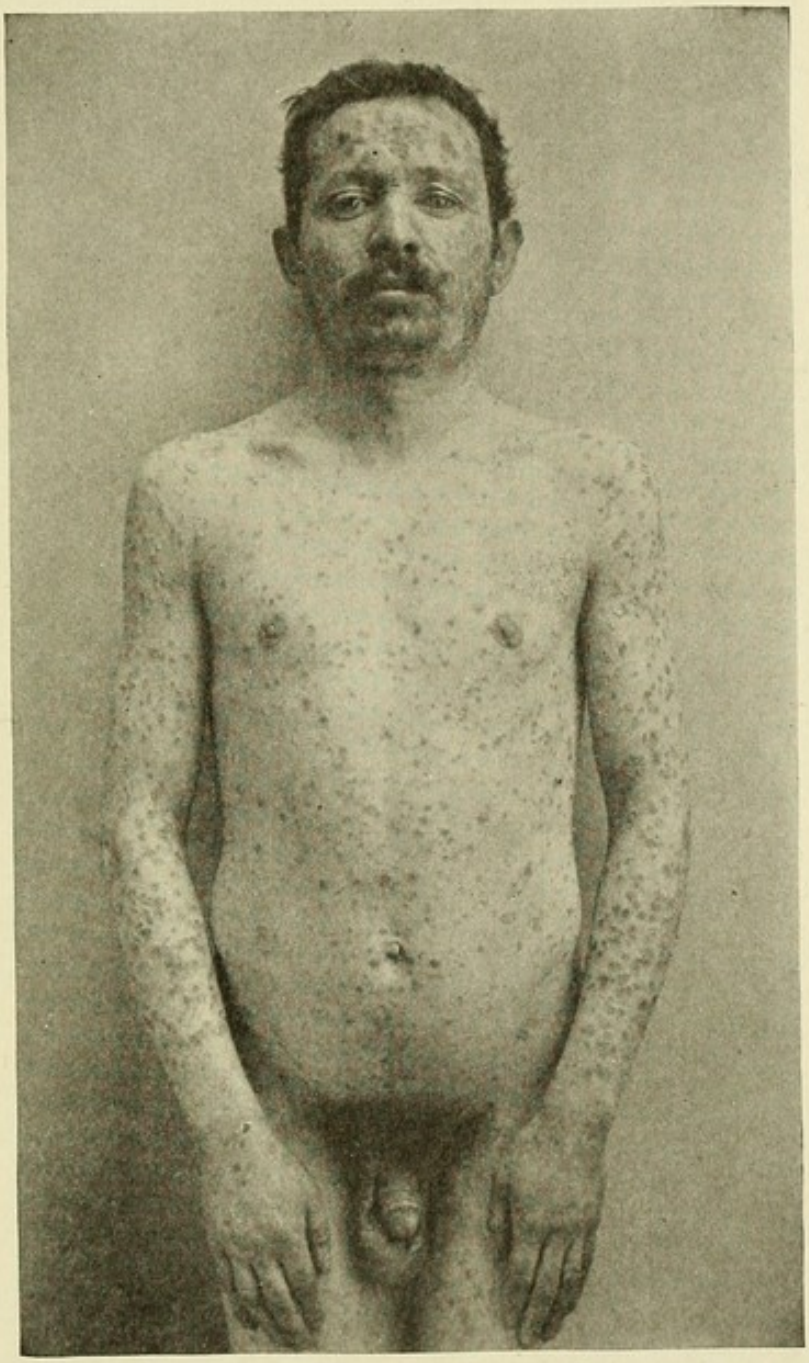

FIG. 26.—GENERAL PAPULO-SQUAMOUS SYPHILID. The lesions are confluent on the face: the scaling is most marked about the shoulders. (Fox.)

several desquamations occur. Finally, the color pales and the lesion disappears, leaving no cicatrix, provided the eruption

has not been a tertiary one (tuberculo-squamous). The circinate form starts as a circle, or segment of a circle, inclosing

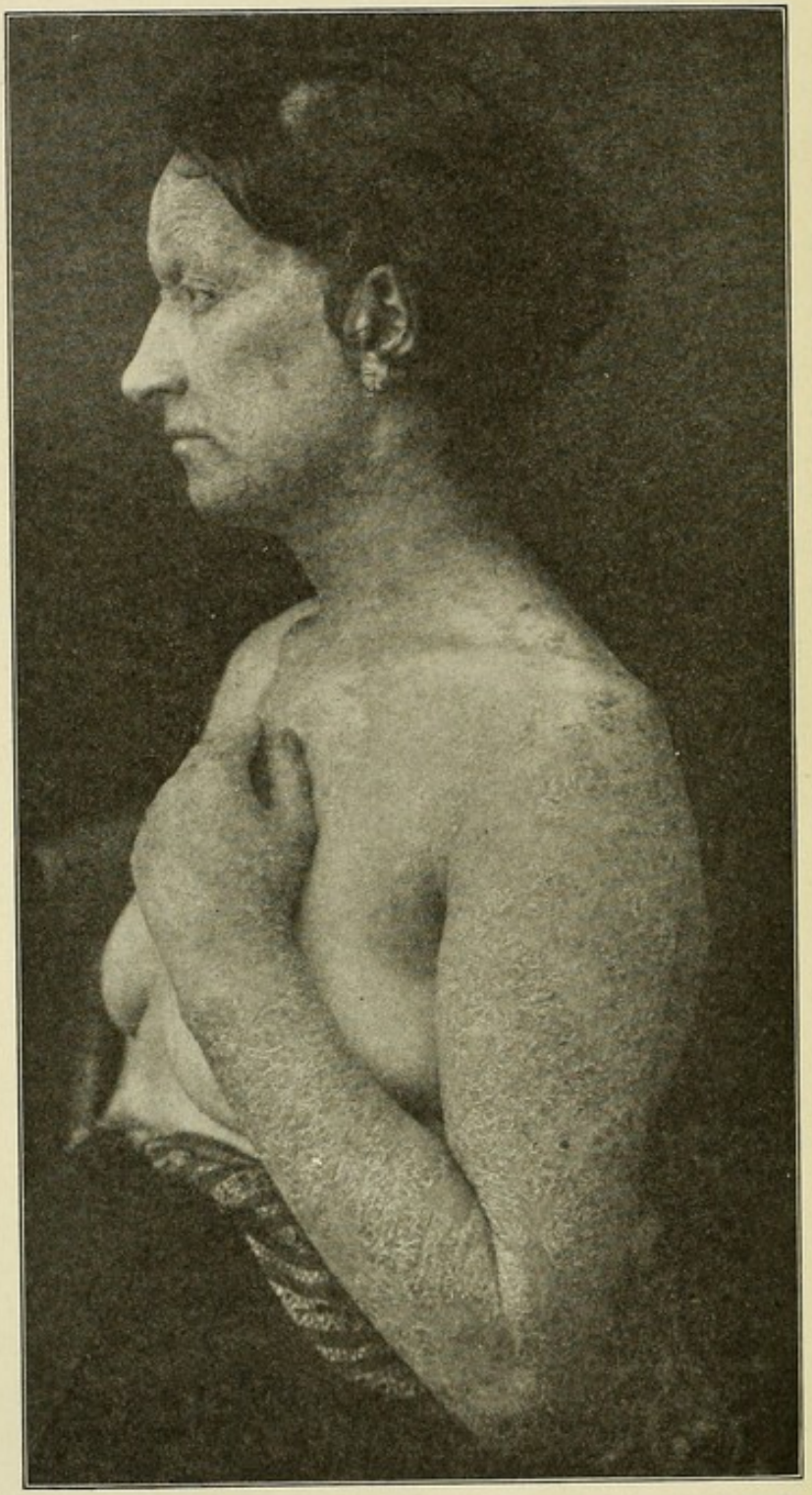

FIG. 27.—CONFLUENT PAPULO-SQUAMOUS SYPHILID, RESEMBLING PSORIASIS. (Fordyce.)

healthy skin, does not generally increase in size, and lasts from a few weeks to some months (Fig. 28). This eruption is a late one, as a rule, and may come on after an interval of many years.

The squamous syphilid appears upon the trunk, the members, the face, and along the forehead at the edge of the hair.

FIG. 28.—CIRCINATE PAPULO-SQUAMOUS SYPHILID OF FOREARM. (Fordyce.)

It shows no tendency to cluster at the elbows and knees and to affect almost exclusively the extensor surfaces of the extremities, as does psoriasis. The later its appearance after chancre the more chronic its course.

DIAGNOSIS. — When not associated with other specific lesions, the squamous syphilid is often difficult to distinguish from *psoriasis*. Much light is thrown upon such cases by a study of the previous history on such points as the well-known inveterate tendency of ordinary psoriasis to relapse and its tendency to outbreak in the spring and fall. Neither eruption itches, and both have the same livid redness of color under the scales, but ordinary psoriasis tends to cluster about the elbows and knees and upon the scalp, and practically *never affects the palms and soles*; its scales are thick, imbricated, tightly at-

tached, and lying in several layers, so that it is difficult to scrape them all away and get down to the livid redness of the patch beneath; and when the scales are all rudely rubbed off the patch is very apt to bleed.

Common lepra, whose scales come off in patches, is usually much more extensive in its distribution than the syphilitic eruption and of indefinite duration. In the syphilitic affection the scales are more lamellar, finer, less adherent, not imbricated, or in thick layers, while the duration of the eruption is not so great. Finally, antisyphilitic treatment has a marked and rapid effect in the one form while it does not modify the other.

The circinate form in some of its stages exactly simulates ordinary *ringworm*, but the diagnosis may be made by the absence of spores and by watching the course of the eruption which, in syphilis, remains stationary, while in ringworm a progressive, centrifugal enlargement is observed.

The Palmar and Plantar Syphilid (see page 303).

THE PIGMENTARY SYPHILID

The pigmentary syphilid is one of the rarest skin lesions of the disease. It appears almost exclusively in women between the fourth and the twelfth month. It seems to be much rarer in the United States than in Europe.

The typical eruption consists of a pigmented area on the side of the neck inclosing a multitude of white islets (Plate VII). The characteristics of the eruption are:

1. It appears spontaneously, and is not apparently due or in any way related to any other syphilid.

2. The islets, though they appear abnormally white, have the same color as the normal skin; the apparent whiteness is an optical illusion. This may be proven by cutting in a piece of paper little holes corresponding to the white patches: the

PLATE VII.—Pigmentary Syphilid of Neck. (Musée, St. Louis.)

paper is placed over the lesion so as to hide the pigmented spots, and one immediately appreciates the normal color of the white islets.

3. The disappearance of the pigmented spots is extremely slow, and is apparently unaffected by antisyphilitic treatment. Though most authorities believe the eruption an essential or independent lesion, G. H. Fox maintains [1] that he has watched it grow from macules upon the neck; macules that became pigmented themselves, and then gradually faded, leaving the surrounding area pigmented.

DIAGNOSIS.—This typical eruption, looking like a brown net spread over the side of the neck, is unmistakable. But the eruption may appear elsewhere on the body and may not have the reticular distribution. Such instances are so rare as to be extremely misleading. Only the history would serve to differentiate them from idiopathic or cachectic vitiligo.

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[1] *Am. J. Med. Sci.*, 1876, p. 356.

CHAPTER XXI

SECONDARY SYPHILIDS OF SPECIAL REGIONS INCLUDING ONYCHIA

CERTAIN syphilids take on peculiar qualities from the physical influences brought to bear upon them in certain special localities.

Taking them in order of importance we shall describe:

Moist papules, erosions, and condylomata.

Squamous eruptions of the palms and soles.

Onychia and paronychia.

Eruptions in the scalp.

Infiltrations about the face.

MOIST PAPULES, EROSIONS, AND CONDYLOMATA

The *moist papule* (erosive papule, mucous papule, patch, or plaque) and its associates, the erosion and the condyloma, are not so common upon the skin as upon the mucous membranes, though extremely frequent about the genitals of women; yet, as they are the most infectious lesions of syphilis, except the chancre, their importance cannot be overestimated.

The moist papule is none other than the syphilitic papule modified by its surroundings. It occurs only upon thin skin, in regions lying in folds, and more or less macerated by perspiration.

Hence these papules are relatively common upon dirty people, fat people, women, and infants. They are relatively rare, in fact, extremely rare upon thin, clean men. Their favorite

location is about the genitals and anus. They are often seen on the lips and within the female genitals. Less common locations are between the toes, under the breasts, in the axillæ, and elsewhere.

The moist papule is the same flat, slightly elevated lesion as occurs on the mucous membranes, though sometimes rather harder in consistence. In fact, it is an intermediate lesion between the dry papule of the skin and the moist papule of the mucous membrane, and bears the characteristics of the one or of the other according as it is more or less macerated.

Accordingly, they are best described with the secondary eruptions of the mucous membranes (Chapter XXIII).

SQUAMOUS ERUPTIONS OF THE PALMS AND SOLES

The palms and soles are very commonly affected by syphilis, and the later syphilitic eruptions in these regions are strikingly persistent and often extremely hard to cure.

Early disseminated macular (Fig. 29) or papular eruptions and later ulcerative and gummatous lesions of the palms and soles have no noteworthy peculiarity. The squamous eruptions alone deserve mention.

Among 137 cases of squamous palmar syphilid, 10 began in the first year, 19 in the second, 38 in the third, 19 in the fourth, 9 in the fifth, 15 in the sixth, 6 in the seventh, 4 in the eighth, and 3 in the ninth year of the disease; 9 between the tenth and the fourteenth year and 1 each in the sixteenth, eighteenth, twentieth, twenty-second, and twenty-third years. Of these lesions, 3 lasted seven years, 1 lasted six, 3 lasted five, 3 lasted four, 4 lasted three, 6 lasted two, and 7 one year. As many of the cases were not cured when last seen, in some instances the duration was surely longer. Two of them were not finally cured until the twenty-fifth year of the disease. Eight cases relapsed, 1 after seven years' interval

(two to nine years), 2 after four years, the others at shorter intervals.

Plantar syphilids are, doubtless, somewhat less frequent than the palmar eruptions, yet we see them very much less frequently, for the patient does not complain of them unless they are quite severe. I find records of 24 cases of plantar syphilids, 14 of them occurring at the same time as palmar

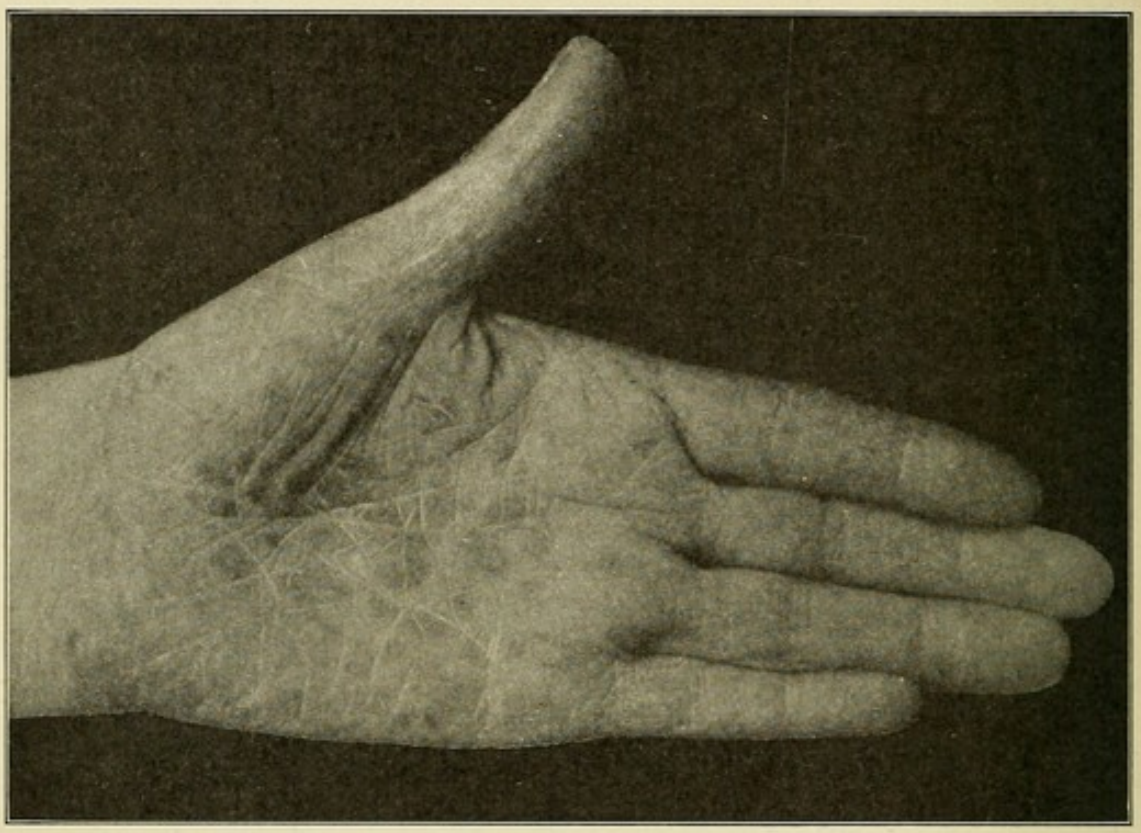

FIG. 29.—MACULAR SYPHILID OF PALM. (Fordyce.)

eruptions. Eighteen of these cases were within five years of the chancre, 2 in the sixth and twelfth years, and 1 in the tenth, eleventh, thirteenth, and twenty-fifth years. One relapsed in the third, fourth, and sixth year; 1 lasted six, 1 five, and 1 two years.

These figures are given in order to impress the importance, the persistence, and the lateness of these lesions. All the late secondary lesions of the palms are in some degree squamous.

With the first general papular eruption there is often involvement of the palms and soles by papules little more scaly than

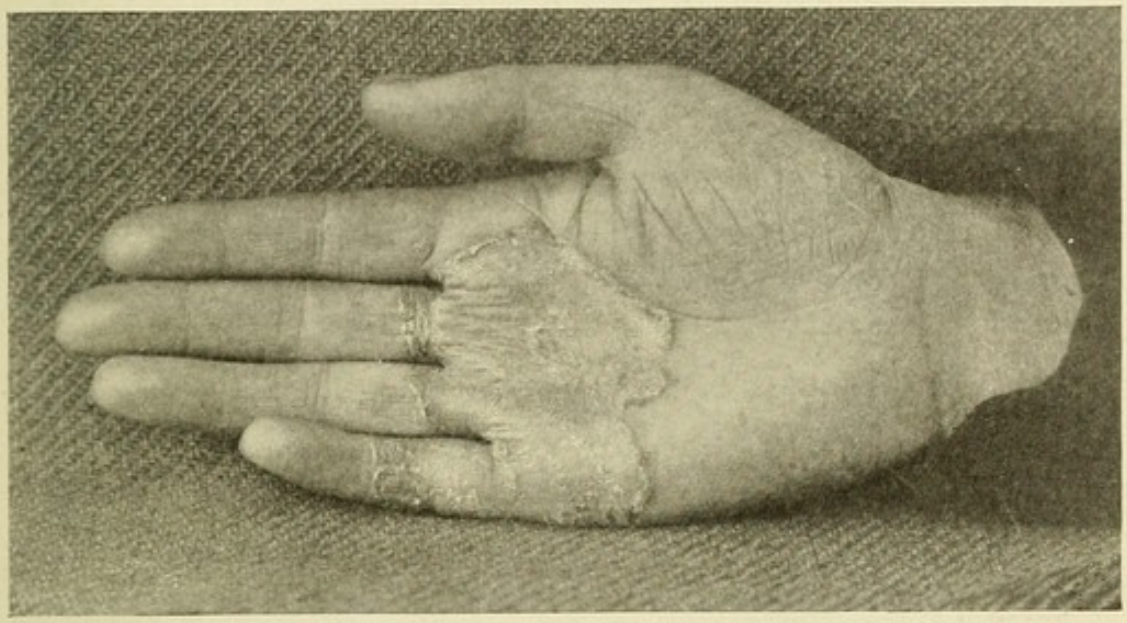

FIG. 30.—SQUAMOUS SYPHILID OF PALM. Note circinate border and absence of definite papules. (Fordyce.)

those elsewhere on the body; but the later papular lesions, exclusively confined to the palms and soles, come out much

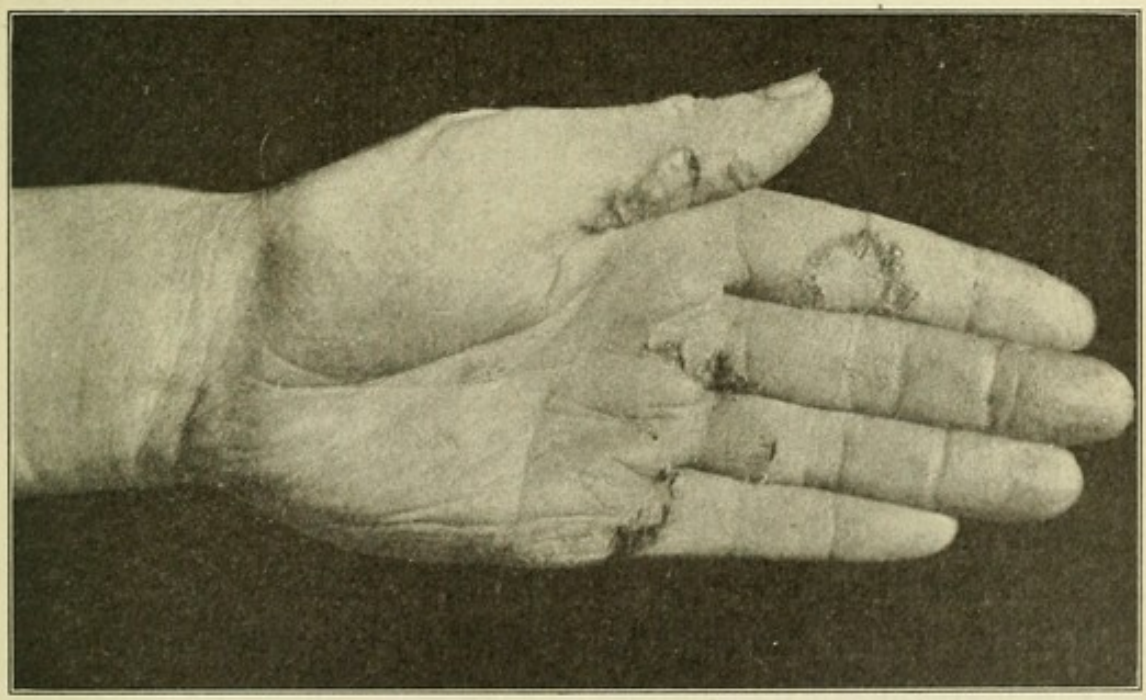

FIG. 31.—FISSURED AND ULCERATED ECZEMA OF PALM, SIMULATING AN ULCERATING SYPHILID. (Piffard.)

more slowly, rise very little above the surface of the surrounding skin, and are either covered with a thick epidermal scale or else surrounded by a meshed fringe of cracked, des-

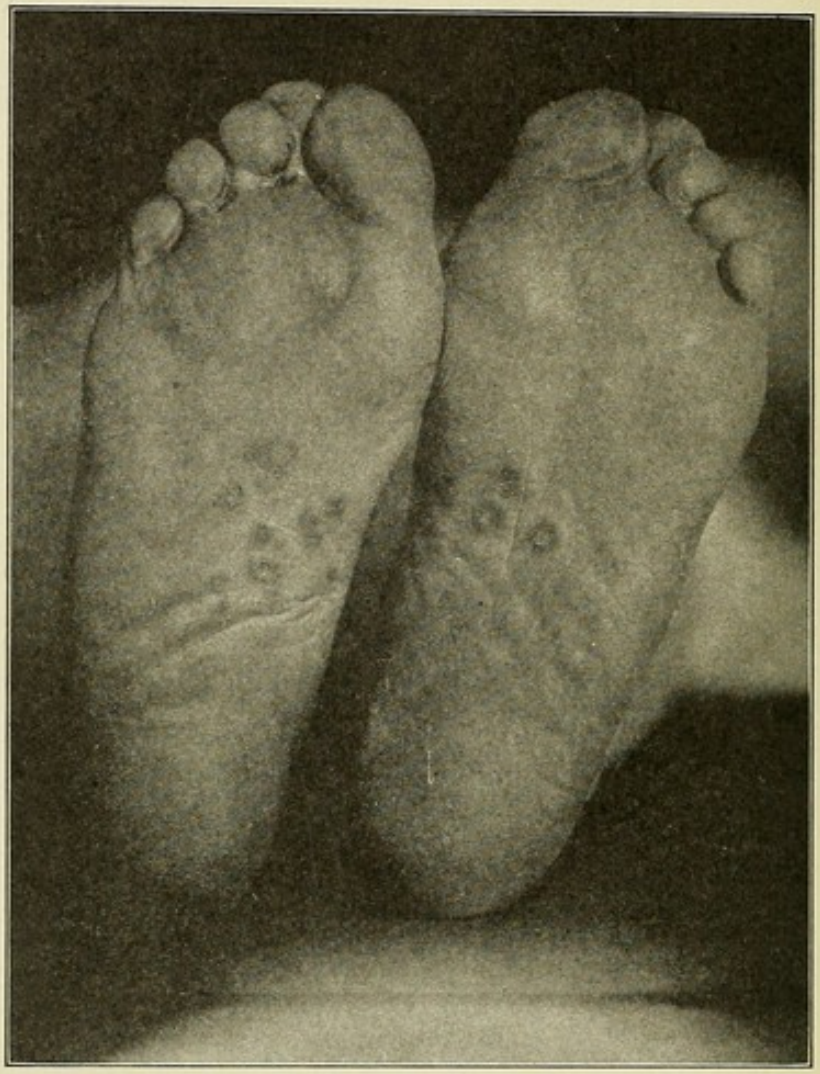

FIG. 32.—ANNULAR PAPULO-SQUAMOUS SYPHILID OF SOLES. (Piffard.)

quamated epithelium. Moreover, the papules tend to agglomerate in masses or to spread over the hand in an irregular circinate way (Figs. 30, 31).

If the eruption is seen when it first comes out, its papular quality may be readily recognized in the manifest, individual papules or in the sinuous line of confluent papules. But in an aged lesion, even if it be only a few months old, the papules

may almost entirely disappear, and all that is left visible is the squamous almost scarlike change.

In this condition the eruption may continue active and spreading in a circinate way for many years, during which time an occasional papule may become visible, or the conflu-

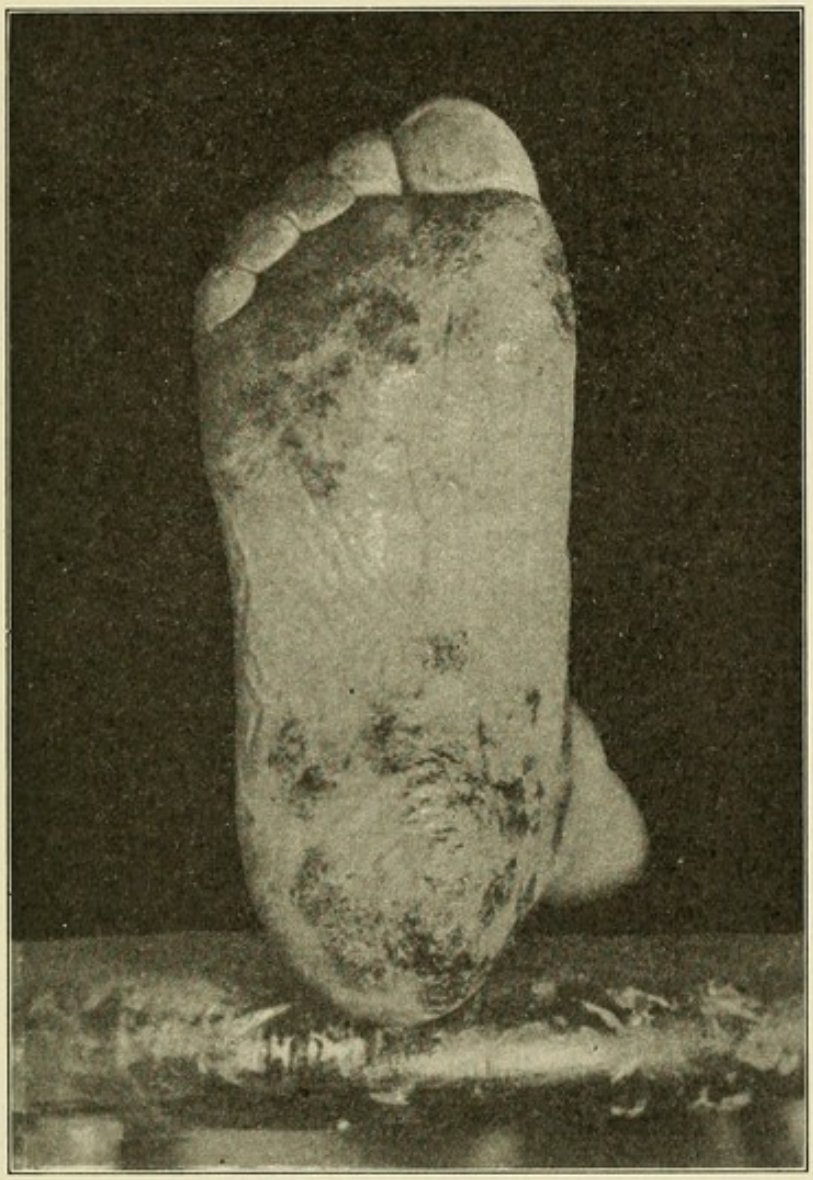

FIG. 33.—PAPULO-SQUAMOUS SYPHILID OF SOLE WITH GREAT THICKENING OF THE EPITHELIUM. (Piffard.)

ent red ribbon may show itself here and there. But the general characteristic of the eruption is a purely scaly one. The scales follow one of two types. In the more common form they pro-

duce glistening, psoriatic-looking spots (papular, nummular, or serpiginous), or else these scales fall, and there remains a glistening, atrophied-looking skin, surrounded by a rough, raised edge of epithelium, precisely like the collarette that surrounded the syphilitic papule, but thicker and more marked. In its mildest type this form of the lesion may simply show little punched-out spots scattered over the palm—little rings of elevated epithelium—while the graver lesions show irregular, circinate spots covering the whole palm. In the earlier cases both palms are usually affected.

Another form of scaly syphilid consists in the heaping up of a great callus. I have seen this more commonly about the heel (Fig. 33) than anywhere else, but most authors agree it is more frequent in the palm. If this callus is dug out it leaves quite a deep pit, which rapidly fills in again. If the hands or feet are much used while in this diseased condition, ragged, intractable fissures result (Fig. 31).

On the fingers the lesions show either on the palmar surface or along the interdigital surfaces. The backs of the hands and of the feet are always spared.

Diagnosis.—So closely do these lesions mimic *eczema* (Fig. 31) that almost all of the rebellious cases that have come to me have had prolonged treatment for that disease.

The diagnosis, though difficult and sometimes impossible without the test of treatment, rests on the following distinctions: Eczema usually itches and oozes and runs over on the back of the hand; syphilis does none of these.

Eczema often exists elsewhere on the body; syphilis often leaves traces elsewhere, or at least a history.

Eczema does not produce the syphilitic papule nor syphilis the diffuse, reddened area of eczema.

The edges of the eczematous patch have rarely the typical rounded or circinate shape of the syphilid.

Psoriasis is so extremely rare upon the hand that it scarcely

enters into the question. Moreover, when it does occur, there is always at least a history of the disease elsewhere in the body, and lesions may be found clustered about the elbows and knees.

Treatment.—I have never seen iodid do this lesion any good. Mercury in small doses cures certain of the milder lesions, but the aged ones often require the most vigorous treatment to cure them. Intramuscular injections of insoluble mercury salts demonstrate their superior virtues in these intractable cases more brilliantly than on any other lesion I know.

The following local applications encourage but do not effect healing:

℞ Ichthyol ..... gr. xv (1 gm.);
Ung. acid boracic ..... ʒj (30 gm.).

or

℞ Ung. zinci oxid, } ..... āā
Ung. picis liquid, }

ONYCHIA AND PARONYCHIA

Secondary syphilis of the nails is fairly common in the early stages of the disease, and I have seen 7 cases of paronychia in late years, 1 each in the fifth, sixth, seventh, twelfth, and twenty-fifth years and 2 in the eighth year. The nails of the fingers are much more commonly affected than those of the toes. The lesions are usually multiple and bilateral.

Syphilitic Onychia.—Onychia occurs in four forms:

1. Atrophic onychia (*onyxis craquelé* of Fournier).
2. Partial detachment of the nail.
3. Total detachment.
4. Hypertrophic onychia.

Atrophic Onychia.—This is quite common in early syphilis. If slight, the nail is simply thin, ragged, brittle, and easily broken; if severe, the corners of the nail thicken, the

nail itself is marked by pearly ridges, and is constantly breaking and tearing away from the nail bed.

This more severe condition is almost always associated with paronychia and due to it. The milder form affects all the nails, and is not a condition peculiar to syphilis. It may be caused by any severe systemic infection.

Partial Separation of the Nail.—This is an uncommon, fairly early lesion associated with palmar syphilids. The whole edge of the nail from top to bottom is gradually elevated from its base, and at the same time usually rather thickened. It is due to lesions immediately under the elevated portion.

Total Detachment.—If the disease is in the matrix itself, it may, without causing paronychia, cause a transverse total detachment of the nail. This is quite unusual.

Hypertrophic Onychia is extremely rare. It consists of an extraordinary thickening of the nail itself, and is comparable to that form of palmar syphilis in which great callous accumulations of epidermis occur.

Other Varieties.—Fournier has described a type of destructive ulceration of the nail. Taylor has noted the occurrence of little pittings on the surface of the nail. Hutchinson has described individual variations of the atrophic onychia.

Diagnosis.—Syphilitic onychia is often mistaken for an eczematous condition from which it may be distinguished by the absence of the cracked, oozing, eczematous lesions.

Paronychia.—Three forms are distinguished: dry paronychia, inflammatory paronychia, and ulcerating paronychia.

In the dry or *squamous type* lesions resembling the active squamous syphilids of the palm surround the ridge of the nail. There is often great thickening of the epidermis and much cracking.

In the *inflammatory type* the bed of the nail is much inflamed, thickened, and painful. At first sight it closely resem-

bles surgical paronychia, but may be readily distinguished by its chronic course, its extremely slight tendency to suppurate, and the absence of cellulitis or fever.

Ulcerating paronychia is extremely rare. It consists of a ragged ulceration about the base of the nail from which there is usually considerable exuberant granulation.

TREATMENT.—Local applications of mercurial ointment or plaster have a slightly beneficial effect upon the graver forms of onychia and paronychia. Inasmuch as the patient has to wear a glove-finger to protect the nail, it is perfectly feasible to make such local applications, though the danger of irritating the skin should be borne in mind. I prefer to use one of the simple soothing ointments referred to above.

The progress of these lesions is extremely slow, and, like the palmar syphilid, they may require hypodermic medication. It is futile, however, to attempt to cure the condition of the nail as such. If the lesions in the matrix or in the bed of the nail have resolved, the nail will ultimately become normal, but may take many months to do so.

ERUPTIONS IN THE SCALP

Pustular or Crusted Syphilid.—As already stated, the general papular syphilid shows itself on the scalp as a pustular lesion, which promptly becomes crusted. Little papules covered with crust can almost always be found disseminated over the scalp in connection with a diffuse papular or pustular syphilid. These so-called scabs in the scalp, though in themselves not very typical, are absolutely characteristic of syphilis when found in connection with a syphilitic eruption of the body (page 120).

Vegetating Syphilid.—If the patient does not keep his scalp clean these crusted lesions, instead of healing, may continue to ulcerate, grow larger, and finally throw out exuberant

vegetations. Such an eruption is not peculiar to syphilis, and quite strikingly resembles the essential lesion of the tropical disease called *frambœsia* or yaws (page 122). In syphilis, however, these vegetations are not widespread and are almost exclusively confined to the hairy portions of the body, and to such folds in the skin as are bathed in acrid secretions.

ERUPTIONS ABOUT THE FACE

Corona Veneris.—This common, late eruption has been already described (page 290). The corona may be either papular or tubercular.

Diffuse Infiltration.—Diffuse infiltrations about the lips and nose are fairly common. They are due to the confluence of numerous papules and are quite rebellious to treatment. They form thick, circinate infiltrations of the skin, dark red, scaly, or ulcerated. This eruption may be curable by mercury alone or may require high doses of iodid. It is impossible, except by the test of treatment, to distinguish in a given case whether the eruption is secondary or tertiary.

Vegetations.—Little condylomata may occur upon the hairy regions of the face and in the creases of the *ala nasi* and of the lips.

PLATE VIII.—Tubercular Syphilid.

The eruption consists of (a) isolated tubercles, (b) masses formed by the confluence of several tubercles, and (c) pathognomonic circular and polycyclic lesions. (Musée, St. Louis.)

CHAPTER XXII

TERTIARY SYPHILIDS OF THE SKIN

THE syphilitic tubercle is to tertiary skin syphilis what the papule is to secondary skin syphilis. Yet it is difficult to draw the line between the two. Some of the nummular and irregular infiltrative and eczematoid lesions described in previous chapters lie upon the border line between secondary and tertiary lesions, and it may be quite impossible to state beforehand that a given case will be curable by mercury rather than iodid, or that it will not leave a scar. Indeed, certain lesions begin with apparently innocent papules, and these more or less rapidly develop into tubercles, and perhaps finally break down as gummata.

The pathological difference between papule and tubercle consists in this: The papule is an exudation rather *upon* than *in* the true skin. It does not destroy any of the tissue of the skin, therefore it does not leave a scar. The tubercle, on the other hand, is an exudation within the derma which, upon healing, leaves a permanent scar (see page 279).

Tertiary syphilids of the skin are made up of various tubercular, tuberculo-pustular, tuberculo-ulcerative lesions, and of gummatous lesions. "The tubercle may be described as a gumma of the skin, and, conversely, the gumma as a deep tubercle of the connective tissue" (Morrow).

Thus tertiary syphilids may be divided as follows:

Tubercular lesions.

Tuberculo-ulcerative lesions, and

Subcutaneous gummata.

THE TUBERCULAR SYPHILID

The non-ulcerative, tubercular syphilid occurs under two forms:

The disseminated tubercular syphilid and

The grouped tubercular syphilid.

Either of these may be scaly upon the surface (tuberculo-squamous).

The Tubercle.—The tubercle is a deep infiltration of the true skin. Its volume varies from that of a small pea to that of a dime, or larger. It has the characteristic dark-red color common to syphilitic skin lesions; it is tense and shining upon the surface, though it may be covered with a scale. Its top is rounded or flattened or even slightly concave. It is hard to the touch, and is embodied in the skin.

The Disseminated Tubercular Syphilid.—A generalized, tubercular eruption may be one of the first, general eruptions to appear; but, as a rule, the tubercles, though disseminated, are confined to a given region of the body.

The tubercles are usually small. Their favorite positions are on the forehead (circle of Venus), the lips, the back, the leg, the outer surface of the forearm, and the back of the shoulders. A single tubercle may constitute the whole eruption or there may be great numbers of them.

The Grouped Tubercular Syphilid. — The favorite locations for the grouped lesions are those just mentioned.

The grouping occurs in three forms:

1. The discrete form.
2. The confluent, circinate form (Figs. 34, 35).
3. The massed form.

If *discrete*, the tubercles appear to be ranged in circles or in arcs of circles, forming a polycyclic or circular design. If *confluent* they form sinuous scalloped figures, or else a solid, rounded, indurated *mass*.

As the tubercles may come out in successive groups, one sees them in different stages of their evolution; some small, dark red and tense; others larger, flatter, shriveling, and a little scaly on the surface. Exceptionally, as the tubercle heals, the center becomes normal, and there remains for a while a

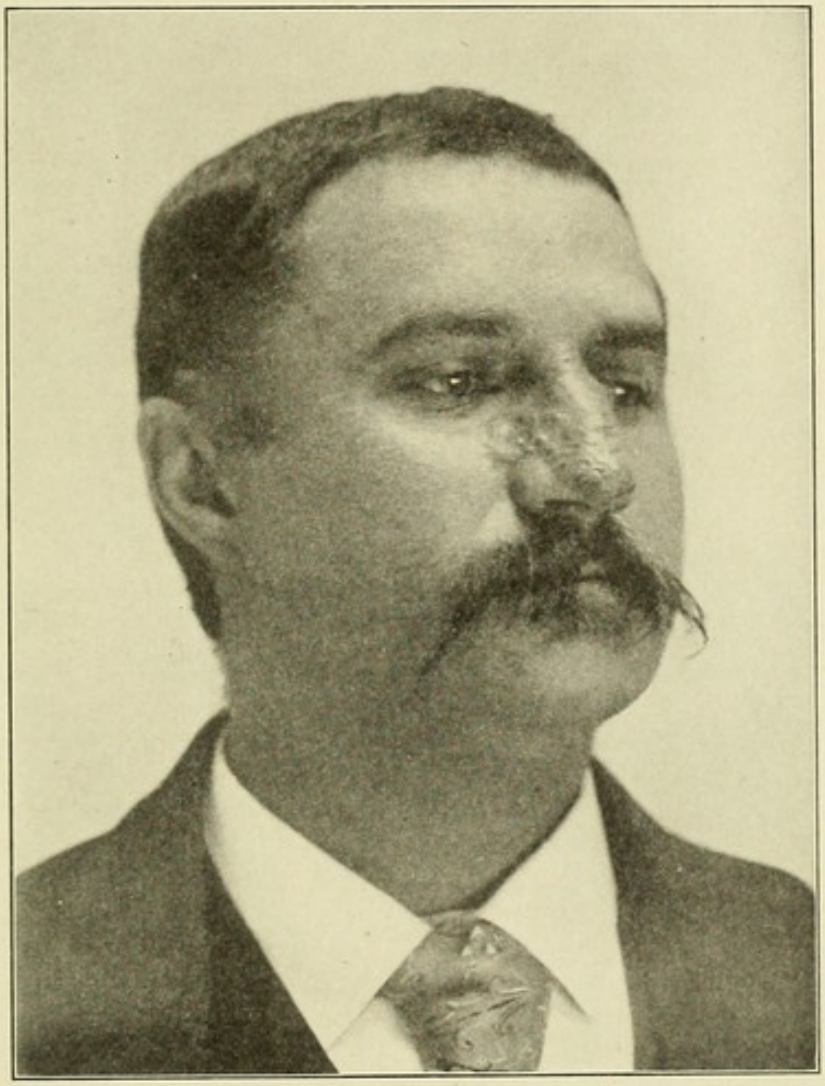

FIG. 34.—CONFLUENT TUBERCULAR SYPHILID OF NOSE. (FOX.)

little indurated circle around a central scar. As complete healing occurs, there is formed a flat, thin, depressed syphilitic scar, which is usually pigmented for some months.

The progress of the lesions is often very slow even under treatment. If untreated, the eruption may be serpiginous, in that the first tubercles gradually disappear and new ones spring

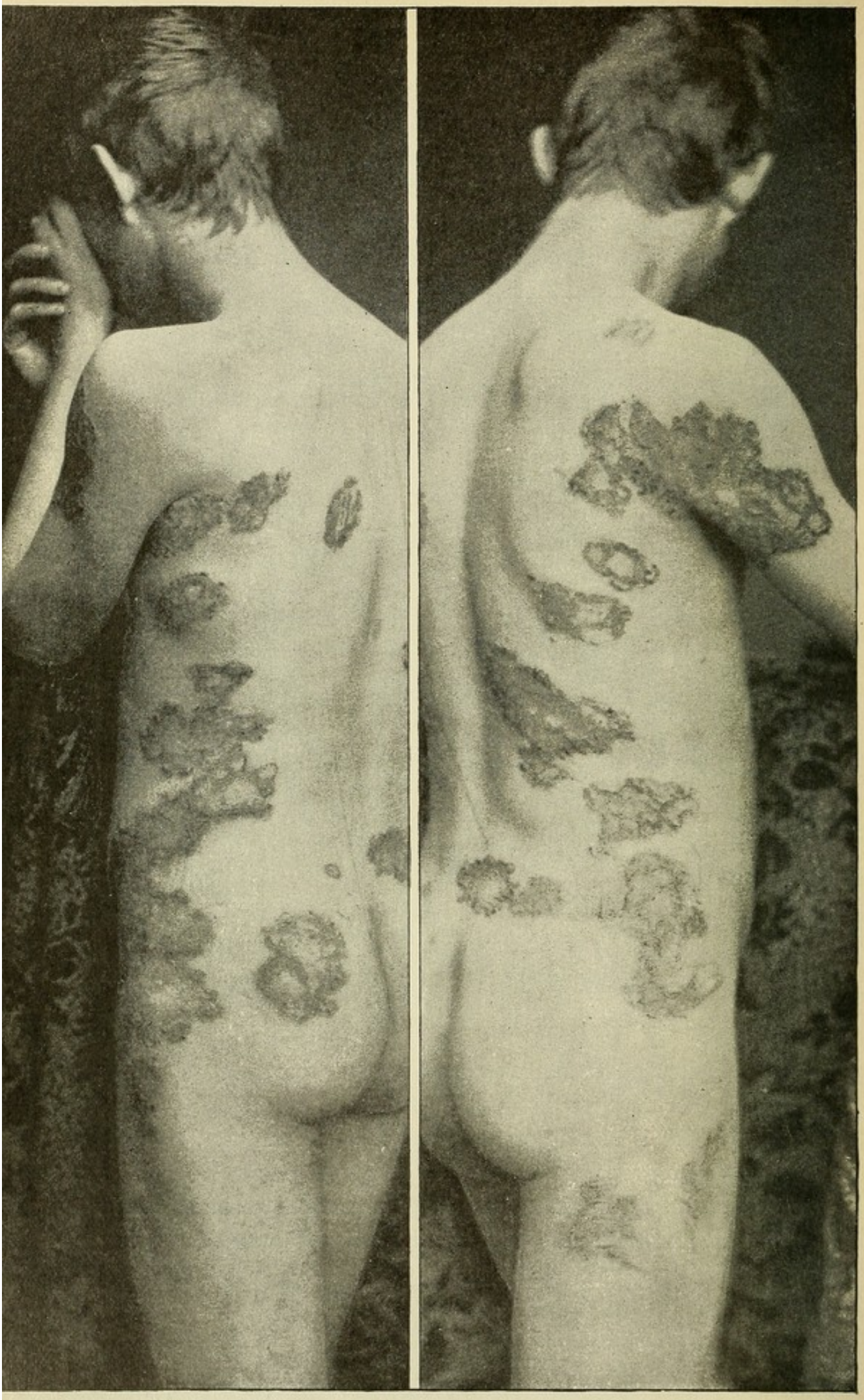

FIG. 35.—SERPIGINOUS TUBERCULAR SYPHILID. (MORROW.)

up about them, so that there is a constantly growing irregular ring of tubercles, either distinct or confluent, in the center of which is an area of smooth, white or pigmented scar tissue (Fig. 35). One can never say when a tubercle will take on ulceration.

Diagnosis.—Although the tubercular syphilid is one of the most strikingly characteristic lesions of the disease, it may be confused with non-ulcerative lupus. The following *characteristics of lupus* serve to differentiate it from the tubercular syphilid: The lupus tubercle is smaller and much more completely sunk into the skin than the syphilitic tubercle. It has a rather more yellowish color and translucent quality. It is softer and more friable. Though not painful, it is distinctly sensitive; though grouped, it is grouped in an irregular way; and its evolution is far more chronic than that of the tubercular syphilid.

Treatment.—The treatment is by iodids and mercury.

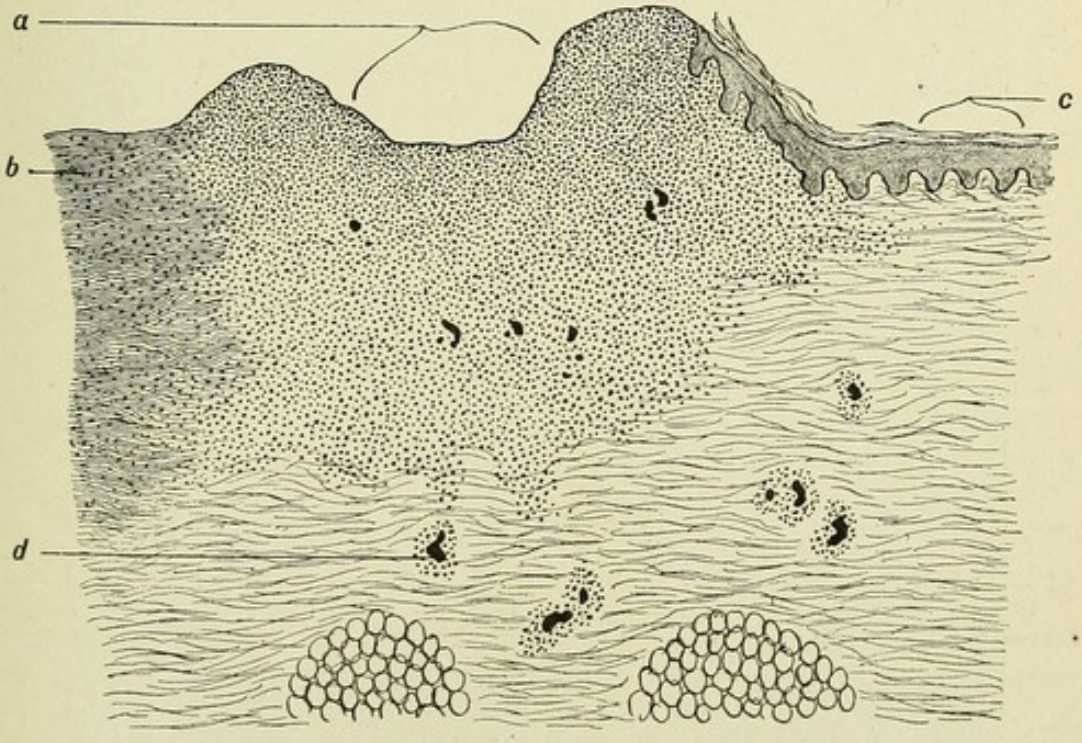

FIG. 36.—PATHOLOGY OF SERPIGINOUS TUBERCULO-ULCERATIVE SYPHILID (diagrammatic). *a*, Ulcer advancing toward normal skin (*c*) and leaving scar (*b*) behind it. *d*, Outlying perivascular infiltration.

THE ULCERATIVE TUBERCULAR SYPHILID

Under this general title may be included all pustular, ulcerative and crusted tertiary lesions of the skin, except the subcutaneous gumma.

This group of lesions has certain common characteristics.

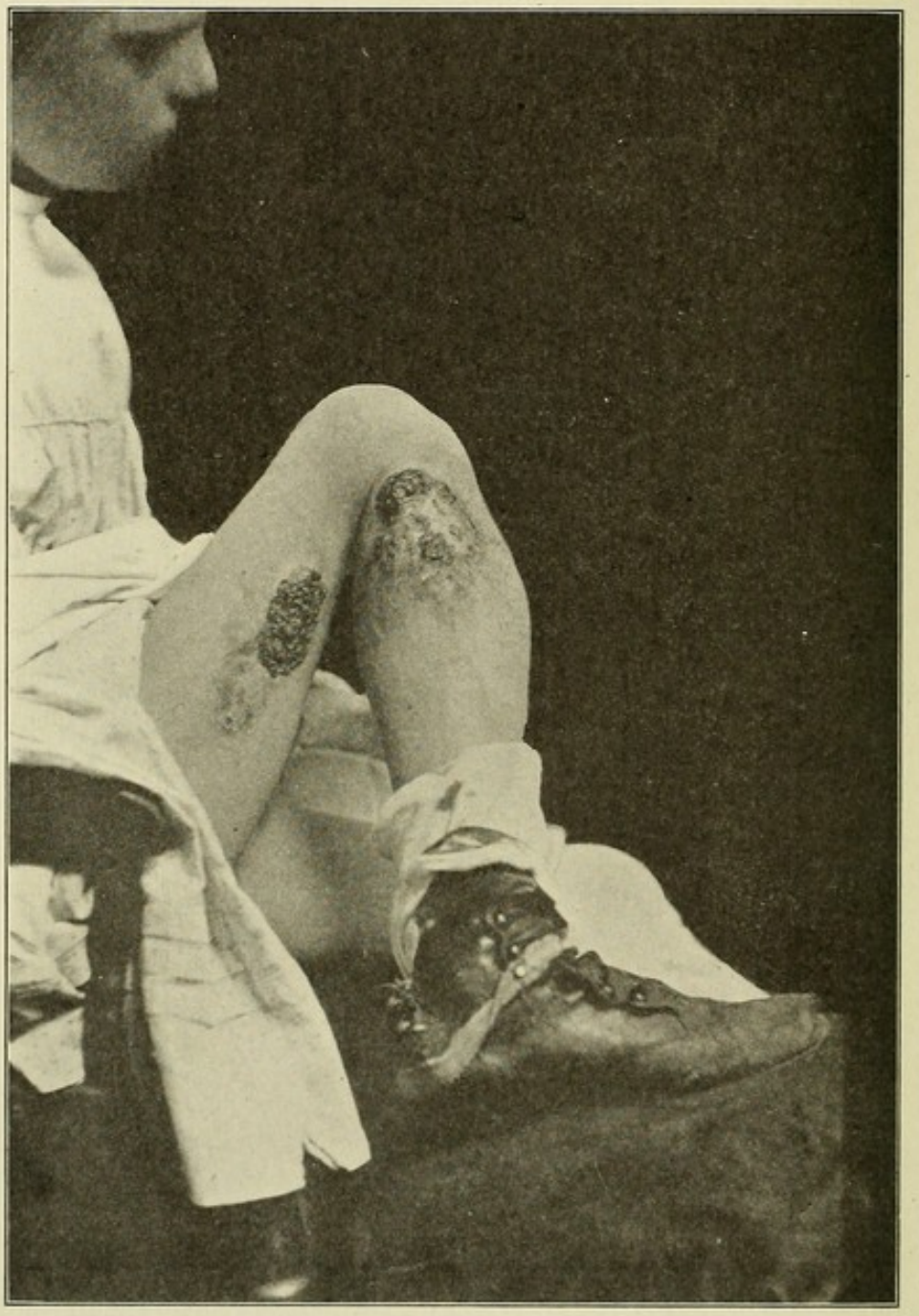

FIG. 37.—SERPIGINOUS TUBERCULO-ULCERATIVE SYPHILID. (FOX.)

The individual tubercles are relatively large. They begin as small tubercles, which soon become pustulous and then ulcerate, all the while growing quite rapidly.

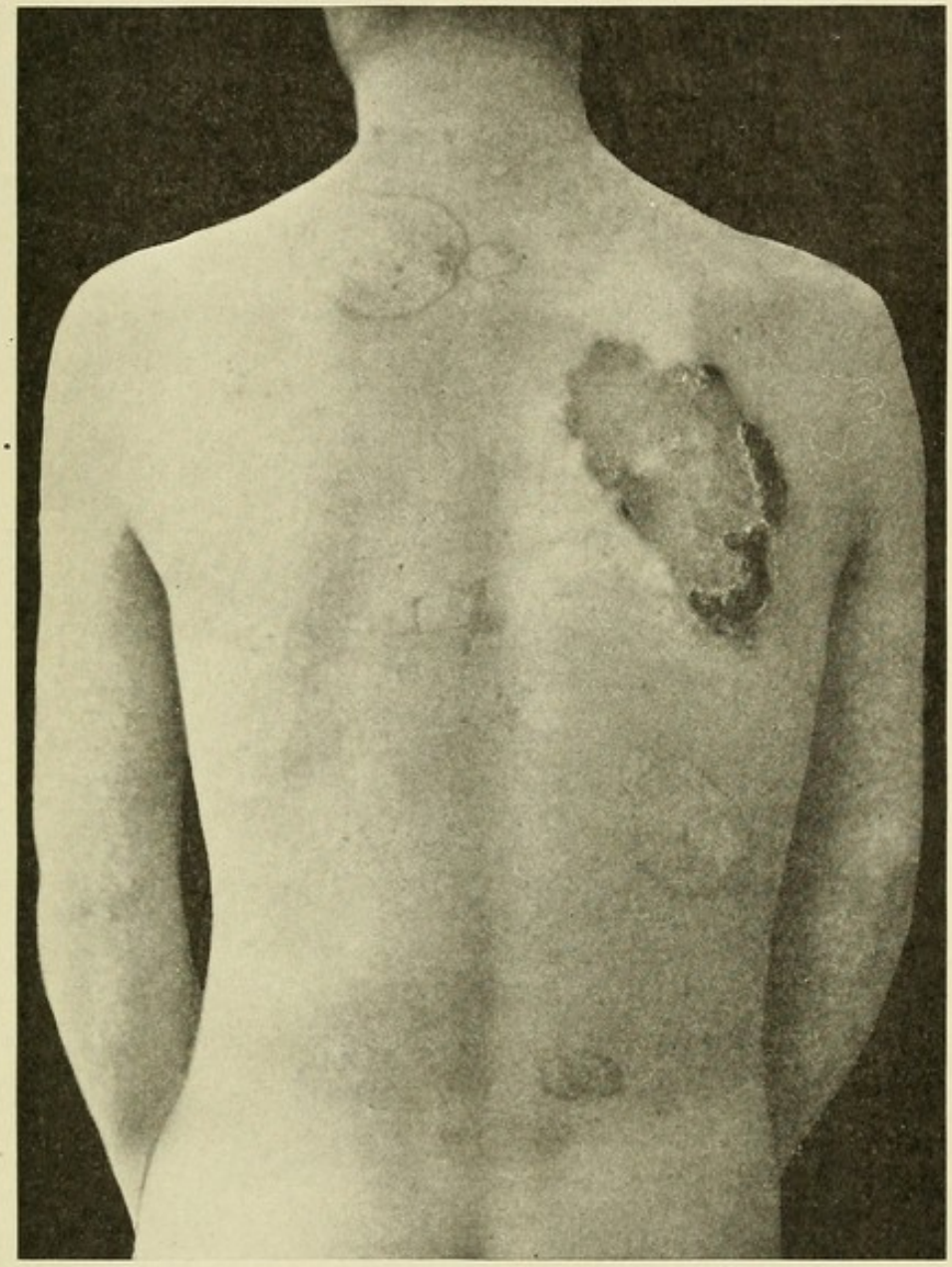

FIG. 38.—SERPIGINOUS TUBERCULO-ULCERATIVE SYPHILID. The sinuous crusted lesion at the edge of a thin, pigmented circinate scar is most typical, as are the circinate scars above and below (marks left by adhesive plaster in middle of back). (Fordyce.)

They may become serpiginous; that is to say, the ulcers and tubercles creep over the surface. The advance may be

centrifugal in all directions or along a narrow track in curves, inclosing portions of healthy skin; or the advance takes place in only one direction, while the opposite edge of the ulcer is cicatrizing.

Thus the *serpiginous ulcer* is usually a flat plane of scar tissue more or less completely surrounded by a ditch of ulceration set in an indurated syphilid as a base (Figs. 36, 37, 38).

The most important of the general characteristics of the ulcerated syphilid is its tendency to phagedena.

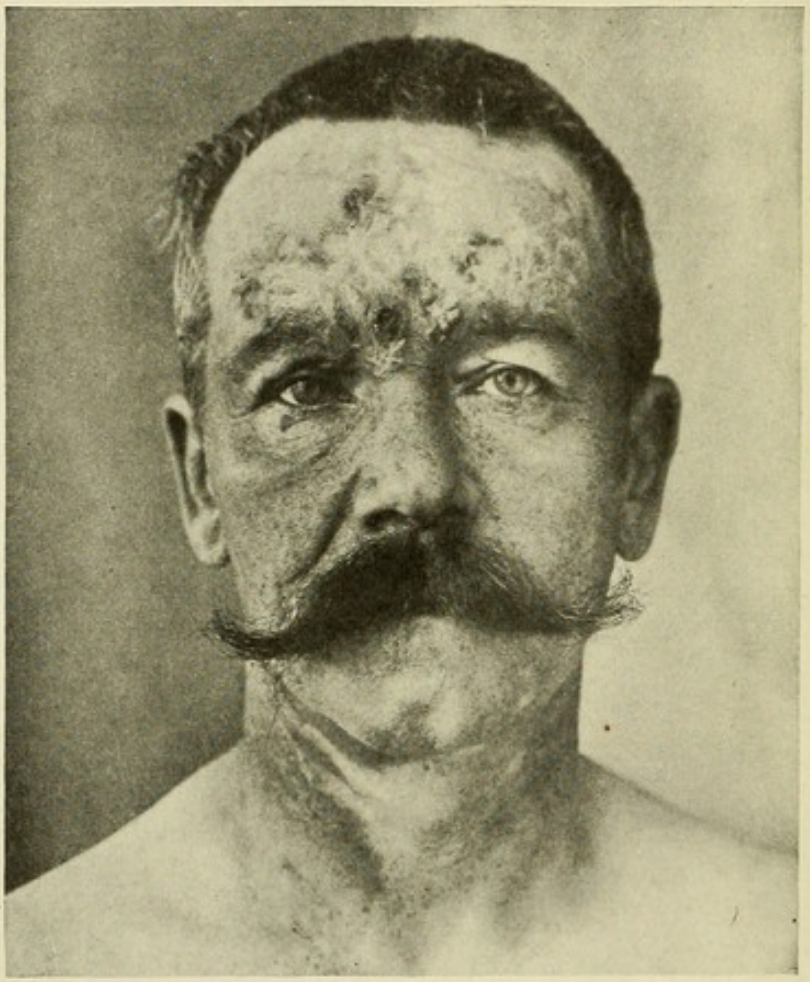

FIG. 39.—ANCIENT SERPIGINOUS ULCERATED TUBERCULAR SYPHILID OF BROW ALMOST HEALED. The scar though very irregular in general outline is thin and in detail circinate. (Fordyce.)

Phagedena.—Phagedena is a malignant, destructive, rapidly spreading gangrene. It is extremely rare except with

syphilis of the soft palate (with which we are not at present concerned). Though a tendency to phagedena is most marked in the cachectic or in those whose constitution has been broken

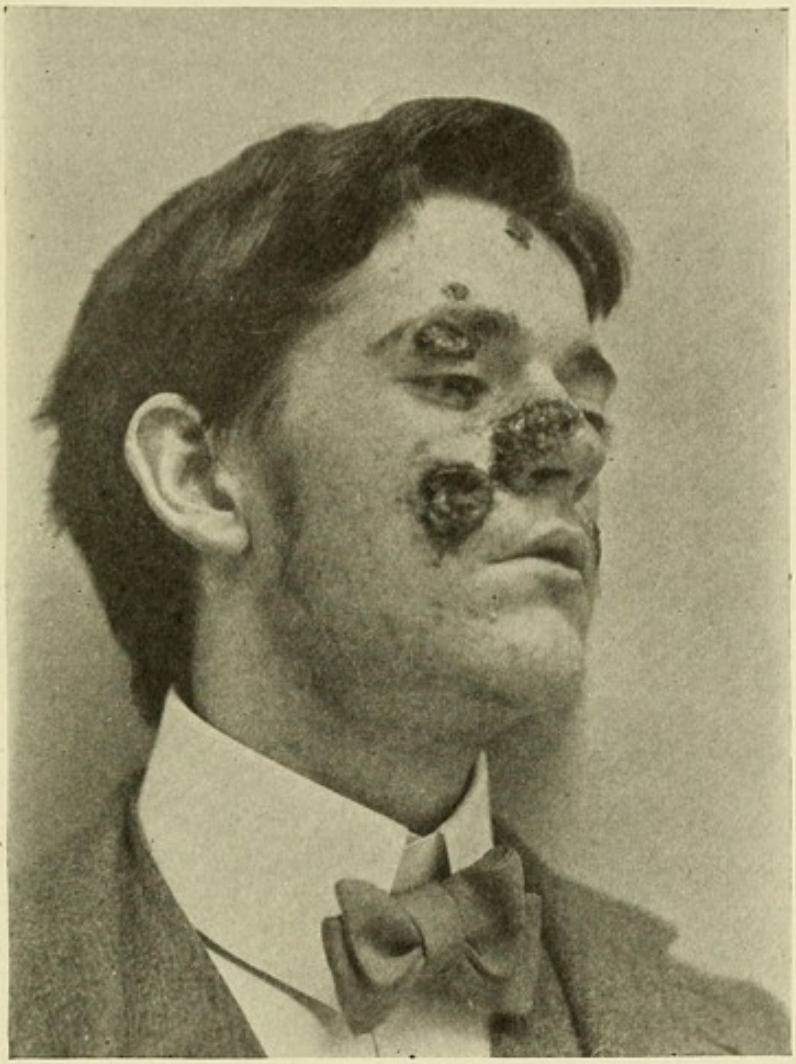

FIG. 40.—PHAGEDENIC ULCERS: THESE MALIGNANT ULCERATIONS APPEARED IN THE FIRST FEW MONTHS OF THE DISEASE. (FOX.)

by alcoholic excess, it may afflict anyone without apparent predisposing cause (Fig. 39).

Phagedena usually occurs very soon after the first appearance of the lesion; and it may progress so rapidly that there is practically no intermediate stage of pustulation or ulceration. The tubercle becomes gangrenous, and immediately the gangrene spreads like wildfire, devouring the tissues in its path.

It may spread chiefly over the surface (superficial phagedena); or may eat its way deeply into the underlying tissues (deep phagedena).

The course of phagedena is most capricious. Even when untreated its rapid spread will suddenly stop and the slough will be cast off and the lesion go on to heal; or, having stopped, it may take a new start.

TREATMENT.—Thus one can never be sure of the effect of treatment. When the gangrene is controlled there may be room for doubt whether it would not have ceased spontaneously quite as soon. Indeed, the Viennese school attack phagedena solely by hygiene and tonics, reserving mercury and the iodids for the cure of the ulcerated lesion left after the slough has been shed.

I have always thought, however, that high doses of iodids help to control phagedena, and, in attacking it, it has been my custom to push this drug rapidly to the point of saturation.

Characteristics of the Lesion.—The ulcerative tubercular syphilid may appear clinically as a *pustular*, a *crusted*, or an *ulcerating* lesion. Pustules are infrequent and rapidly break to take on one of the other types. Whether the lesion is crusted or ulcerated depends largely upon the accident of its situation. Usually it will be found covered with a typical syphilitic scab (page 274); but if it has recently been picked or if it lies in some place subjected to constant friction, the scab may be off and a typical syphilitic ulcer (page 274) disclosed.

Thus the lesion may appear in one of the following forms (we give only the most important ones, since they are useful merely for clinical identification):

Tertiary ecthyma.

Rupia.

Other forms of crusted syphilids.

The ulcerated syphilid.

Ecthyma.—Tertiary, or tubercular, or deep ecthyma differs from the secondary lesion already described only in that the pustules are set upon tubercles instead of papules. The pustules are evanescent; they soon break, and the tubercle is covered with a thick brownish-green scab set into and tightly adherent to the punched-out ulcer. The lesions are usually multiple and small; in healing, they leave flat, rounded, depressed pits.

Rupia.—The rupial syphilid is rare. It occurs almost exclusively in debilitated or debauched persons; it is usually multiple.

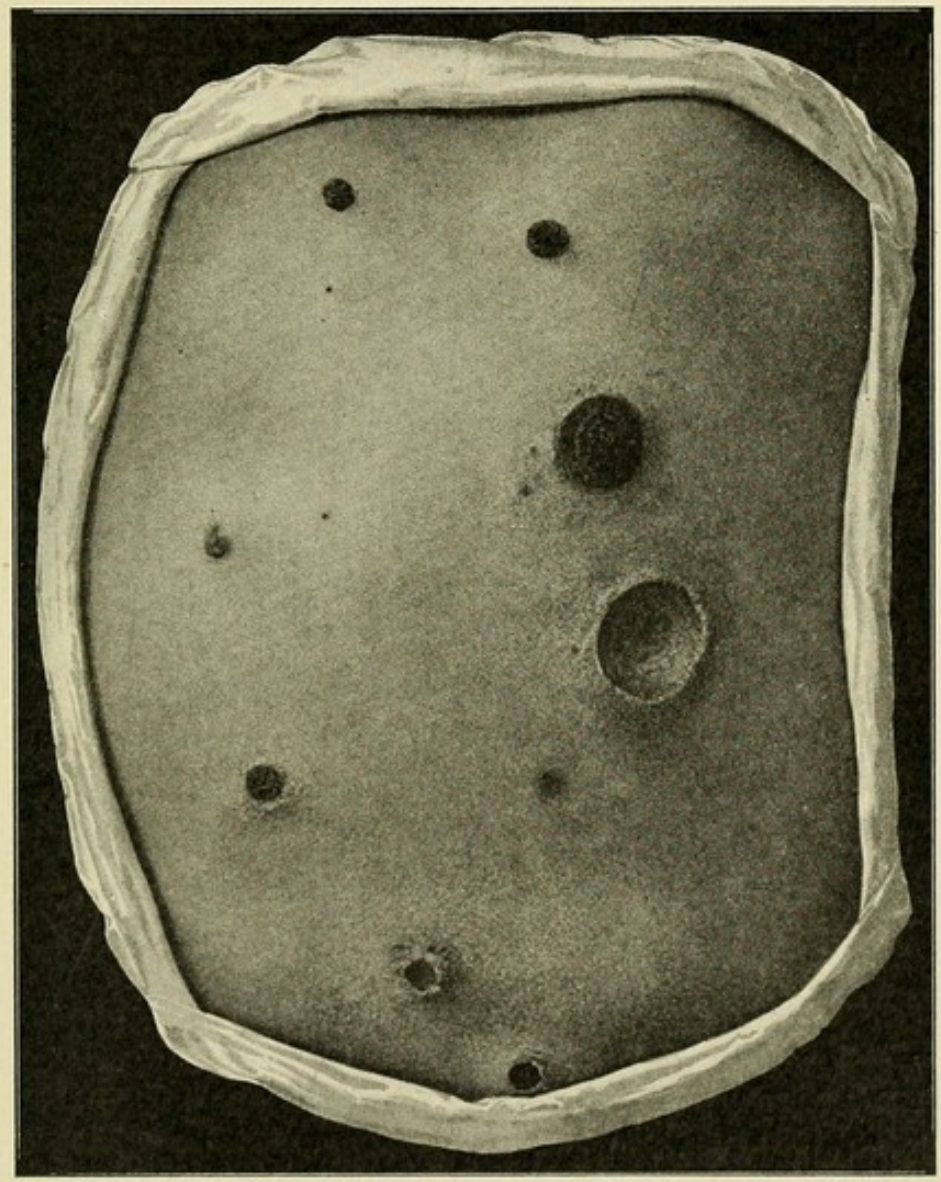

Fig. 41.—Rupia. (Musée, St. Louis.)

The ulcer is commonly a fairly large one (often an inch in diameter) and its differentiating characteristic is the rupial scab. The first crust is elevated by the formation of a layer of scab beneath it, and, as this process goes on for weeks, it becomes finally a prominent, rough, oyster-shell scab marked by concentric layers of brownish or greenish black, projecting above the surface almost in the shape of a horn. If the underlying ulceration is not very active, the scab fits dry and tight over the ulcer; but if the ulceration is active, there is often an ooze of pus from underneath and a tendency to the formation of a bullous ring outside the crust, due to the progressive increase in the size of the ulcer (Fig. 41).

These crusts grow to incredible size before they finally fall. The healing of the lesion leaves a large, rounded, syphilitic scar. Though rupial lesions grow gradually in size, they almost never become serpiginous.

Treatment.—Rupia is one of the most obstinate of syphilids. Its cure often depends more upon tonics and hygiene than upon specifics, and in many cases the vigorous onslaught with mercury and iodids so effective in other lesions is absolutely ineffectual. A much better plan is to keep up a moderate, prolonged, specific course while vigorously attacking with tonics and hygiene any manifest deterioration in the patient's health.

Other Crusted Lesions.—The more common, crusted, ulcerated syphilid does not form the mighty, rupial scab, but tends rather to spread in a circinate way with or without healing in the center. The scabs have the same characteristics as those of rupia, but do not grow to any great height. The lesions may be small, as in syphilitic ecthyma, or large, round ulcers, or serpiginous patches (Fig. 42).

The Tertiary Ulcer.—Any of the lesions just described may be properly classed as a tertiary ulcer and may momentarily become so when friction removes the scab from the sur-

face (Fig. 43). The characteristics of the ulcer have already been described (page 274).

Prognosis.—The ulcerative tubercular syphilid progresses slowly, and in most instances, if untreated, shows a consider-

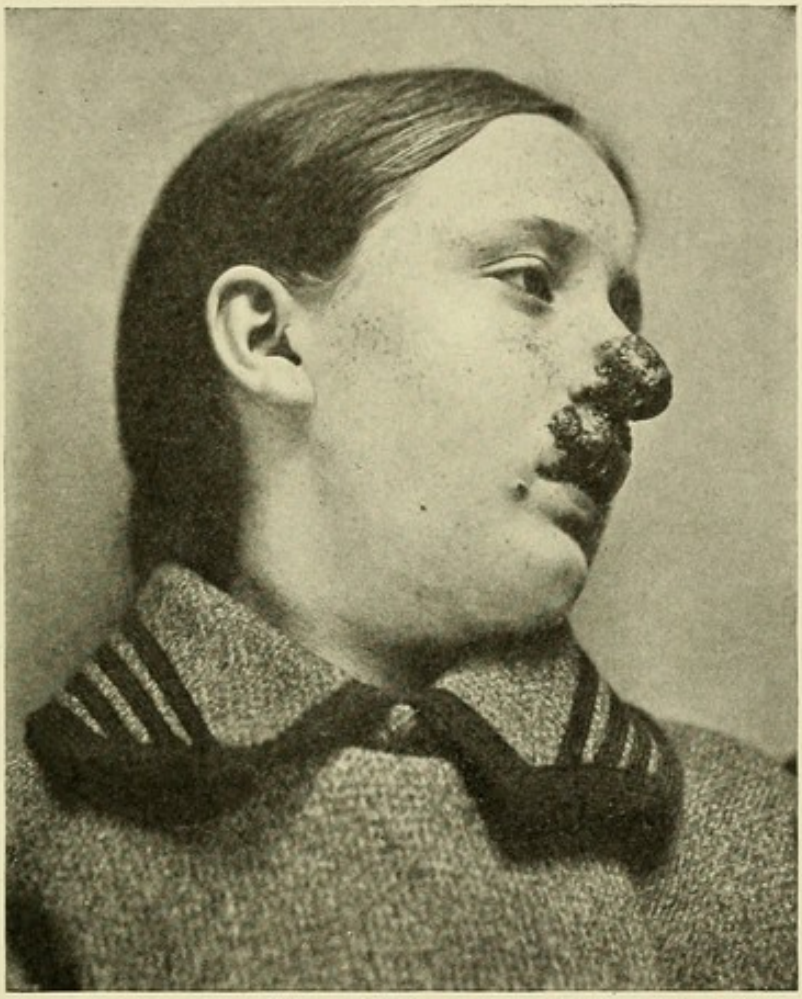

FIG. 42.—CRUSTED TUBERCULO-ULCERATIVE SYPHILID. Lesions confluent on nose and upper lip: a single ulcerated tubercle is seen below the angle of the mouth. (Fox.)

able tendency to heal in some places while advancing in others. Indeed, one may meet old, neglected cases in which certain lesions have healed spontaneously, but even when this does occur, one usually finds other slowly progressive lesions elsewhere on the skin. Years may elapse while this ulceration is slowly spreading, but, as a rule, within a year or two, if no treatment is given, some other syphilitic lesion appears and

forces the patient to submit to treatment. *Gangrene* in the form of *phagedena* may occur at the onset of the lesion, but scarcely ever comes on later. *Inflammation* and *erysipelas* are most exceptional complications.

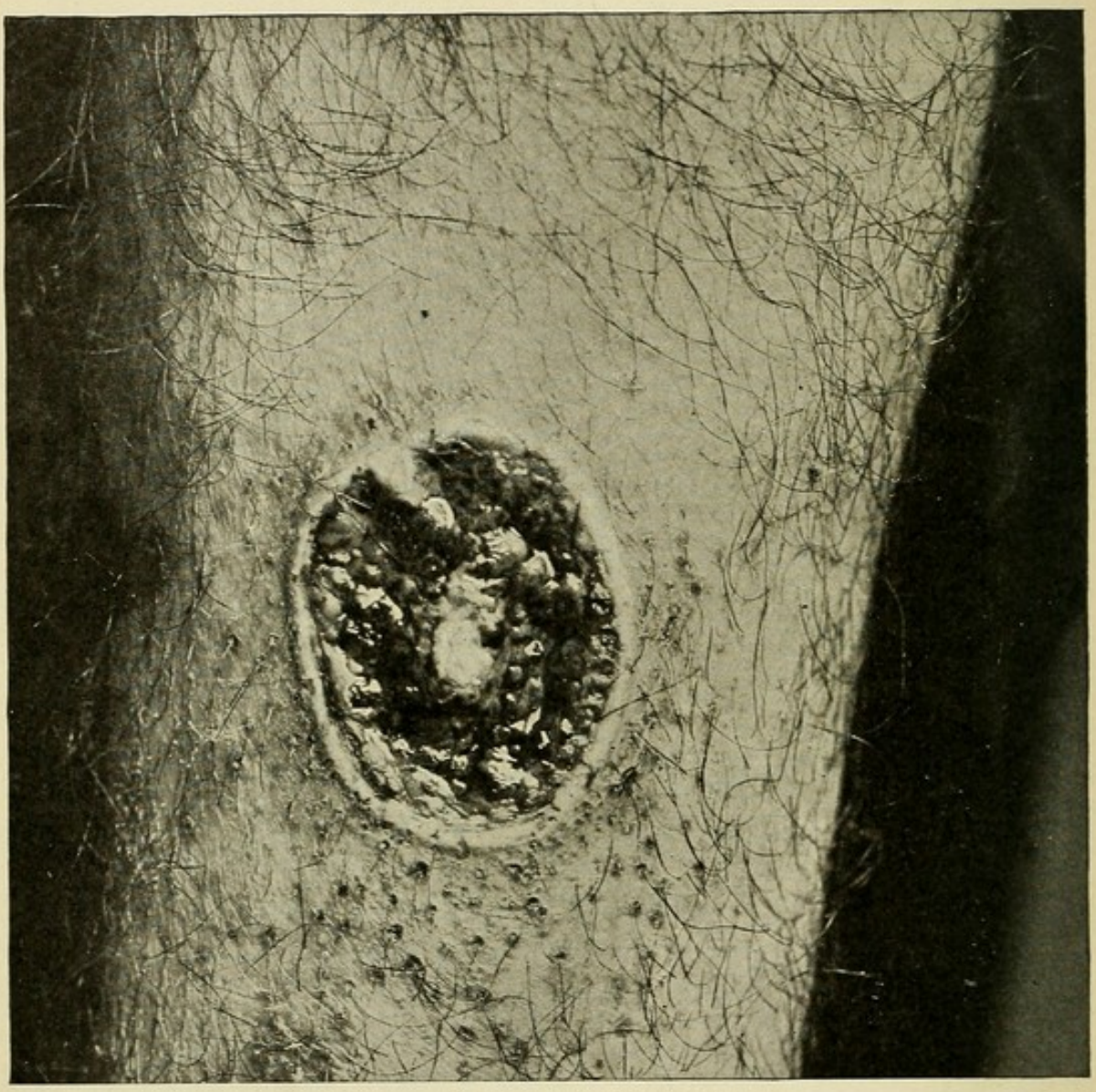

FIG. 43.—TERTIARY ULCER OF LEG.

Diagnosis.—The diagnosis, apart from the history and evidence of other lesions, is made from the appearance of the thick, blackish crusts; from the ulceration itself with its sharp, infiltrated, punched-out edges, its surrounding dark-red areola, and its irregular sloughing or granulating base; from the cir-

cular configuration or circinate grouping of the ulcers; from their insensitiveness; and, finally, by the success of mixed treatment.

Differential Diagnosis.—The differential diagnosis from *ecthyma* and lupus has already been described. The *ulcer of lupus* has a soft, thin border; its areola has a light color; its base is more raw-looking and more superficial, or even vegetating, than that of the syphilitic ulcer. Unfortunately, it is rarely possible to distinguish the tubercular lesions by discovering the bacillus, by inoculating animals, or by biopsy.

Epithelioma and *varicose ulcers* are much more closely imitated by subcutaneous gumma than by the ulcerated, tubercular syphilid.

Leprosy may usually be distinguished by the spots of anesthesia or palpable nodosities on the nerves, the fall of the eyebrows, the rhinitis and epistaxis, and the genital and ocular lesions.

Treatment.—The sole local treatment necessary is to protect the lesion from irritation by covering it with a thick pad of absorbent cotton, or, if it is ulcerated, by powdering with calomel.

SUBCUTANEOUS GUMMA

Subcutaneous gumma is a rather common lesion most frequent between the third and sixth year of the disease (Fournier). Though usually single, gummata may be multiple, and may even occur in great numbers, in which case they may be either grouped or disseminated. Their seat of election is the leg, but they may occur anywhere in the body. They vary from the size of an olive up to that of a hen's egg.

The Pathology of Gumma.—The *subcutaneous gumma* is, as already stated, a deep-set syphilitic tubercle. At its onset it is simply a syphiloma, a localized perivascular granuloma, set in the subcutaneous tissue. But in development it differs

from the lesions previously described in that its center soon breaks down into a gelatinous or gummy mass (whence the name "gumma"). This central gummy mass consists of necrotic tissue held together by a network of fibrous connective tissue, so that even when the necrotic mass is large and completely disorganized it does not become absolutely fluid, but is kept in a characteristic semisolid state by this network of fibers.

This mass, yellow or sero-sanguinolent in aspect, rapidly enlarges and bursts through the skin. Through the ulcer thus formed the gummy center comes away bit by bit, leaving a gummatous ulcer (Fig. 44).

Gumma of the internal organs (liver, testicle, etc.) behaves somewhat differently. It begins as a granuloma and undergoes central degeneration, but it finds no outlet, and is limited in its spread by the change that occurs in its own outer layers. Here the inflammatory exudate changes gradually into scar tissue, which completely envelops the central gummy mass. In this fully developed state the visceral gumma therefore consists of a central gummy mass, surrounded by a dense envelope of fibrous tissue, which itself is infiltrated and surrounded by plasma and small round cells. Healing takes place either by absorption or by encapsulation of the gummy mass. The contraction of the fibrous tissue leaves a characteristic hard, puckered scar.

The *diffuse gumma* forms still another pathologic type, in which the syphiloma is spread over a relatively large area, undergoes no central degeneration, and heals by complete transformation into scar tissue.

The *pathologic diagnosis* of full-blown gumma usually presents no difficulty, for its macroscopic characteristics are quite typical. Nothing can imitate the gummy central mass, held together by bands of fibrous tissue and surrounded by a fibrous or an inflammatory envelope. The shrunken scar is almost

equally typical. But the developing or diffuse gumma may simulate tuberculoma very closely (it is not to be forgotten that the two may coexist).

The features that distinguish gumma are the relative prominence of small round cells, fibroblasts and fibrous tissue, the

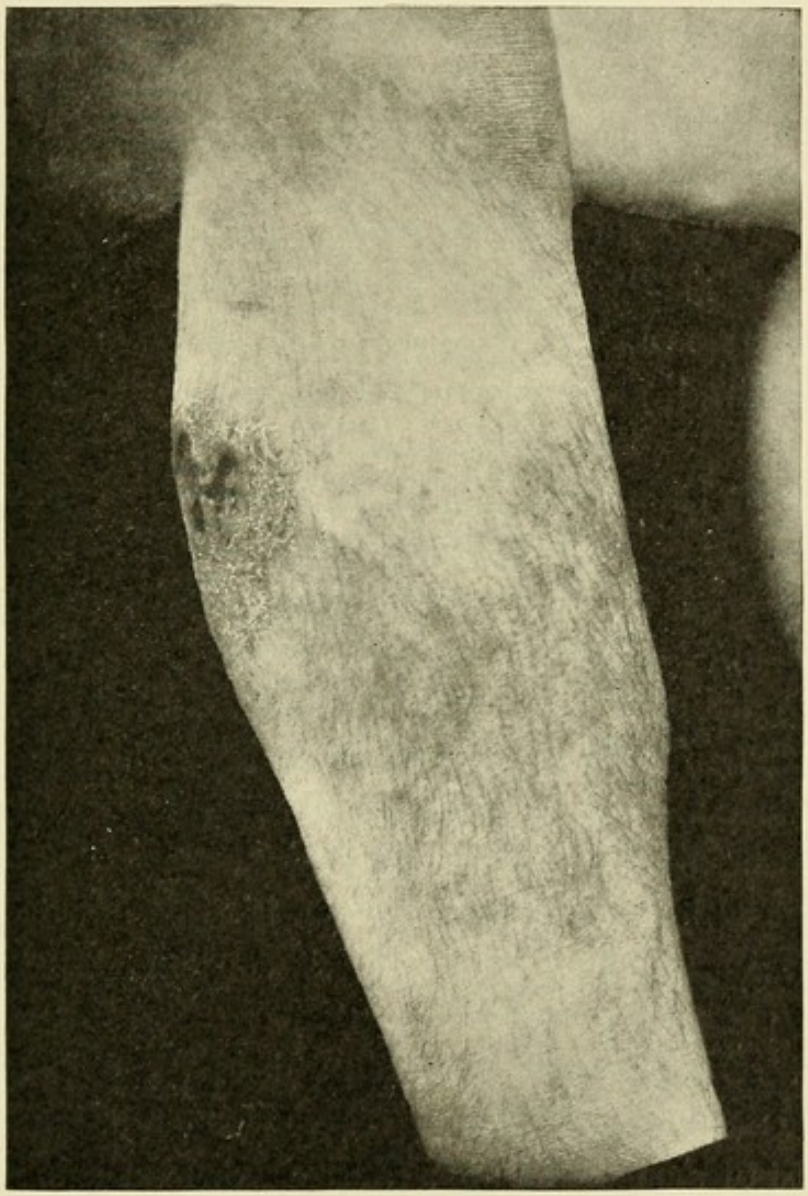

FIG. 44.—GUMMATOUS ULCER: TIBIAL NODE. (Fordyce.)

marked lesions of the capillary vessels, and the relative rarity of epithelioid and giant cells. If a necrotic area can be found this, if the lesion be gumma, will be gelatinous and adherent,

with a fibrous reticulum; while, if it be tubercle, the mass will be cheesy or purulent, homogeneous, and readily detachable. Moreover, in tuberculoma, the bacilli can usually be discerned by microscope or by inoculation.

The subcutaneous gumma affects two types, the single or discrete gumma and the multiple or confluent gumma. These bear the same relation to each other as the furuncle and the carbuncle; the discrete gumma has a single center of syphilitic infiltration, the confluent gumma has many.

The Single or Discrete Gumma.—The gumma begins as an insensitive, little, hard subcutaneous lump, freely movable over the subjacent tissues, the integument slightly movable over it. In this condition it may remain stationary for months, but, as a rule, it grows slowly (or rapidly) in size, and within a few weeks has infiltrated the skin.[1]

The skin becomes red and tense, then, as the gumma begins to soften centrally, purple, boggy, and *tender to pressure.* This tenderness is in marked contrast to the insensitiveness of other syphilitic skin lesions. It may be extreme, and one must not be misled by it into supposing the lesion a simple abscess.

Finally, the softening of the gumma reaches the surface. Actual fluctuation may be determined and may cover quite a large area. As the tenderness continues, and may even be accompanied by intense pain, incision is often practiced to relieve these symptoms. The result is startling. Instead of a gush of pus there oozes forth only a few drops of bloody or purulent serum; the fluctuation persists; the symptoms are unabated; the incision has merely added a disfiguring scar to the list of the patient's woes; for, whether the skin is broken by the surgeon's knife or by the unchecked progress of central caseation, a typical, deep, gummatous ulcer results, whose orifice rapidly enlarges until it covers the whole mass.

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[1] At the same time it usually becomes adherent to the subjacent tissues.

The *gummatous ulcer* has many of the characteristics of the ulcerated tubercle. It is round or, if due to multiple, confluent gummata, polycyclic and surrounded by a dark, indurated areola. But it is too large and too active to be covered by a scab; it is much deeper than the tubercular (syphilid) ulcer, and its base is occupied wholly or in part by the grayish white, irregular mass of caseous degeneration which once was the center of the gumma (Fig. 44).

The gumma may heal at any stage of its development, and it is always our aim to encourage healing before it breaks, or even, if possible, before it softens. The rapid development of the lesion or the carelessness of the patient may defeat these efforts.

If healing occurs in the first or hard stage, there will result from the absorption of the gumma a slightly depressed scar, though the skin over this remains normal. If healing occurs in the second stage, the stage of softening, the infiltrated skin is more markedly depressed and itself is more or less completely transformed into scar tissue. If healing occurs in the third, or ulcerative, stage, the ulcer first sheds the remains of its necrotic center, then becomes shallower both by elevation of its center and by the flattening down of its edges. Then healing takes place rapidly from the periphery toward the center, leaving a flat, thin, round, depressed, pigmented, typical scar (page 274).

The Confluent Gumma.—The confluent gumma begins with many adjacent points of subcutaneous syphilitic infiltration. These run together and progress irregularly, so that, while certain parts of the mass remain hard and scarcely involve the skin, in others there are points of softening, points of ulceration, and even areas of cicatrization adjoining one another. The edges of the mass may be quite sharply defined, or may shade off imperceptibly into the surrounding tissue. These masses of confluent gumma vary widely in size.

Prognosis.—Subcutaneous gumma rarely become phagedenic, and though it may rapidly soften and ulcerate, its further progress is often very slow. For months and even years it slowly enlarges; or if confluent, progresses in one direction while healing in another, so that large, irregular areas of ulceration, induration, and scar are intermingled.

Such lesions are most often seen as chronic leg ulcers. They may be treated as varicose ulcers for years without ever getting much better or much worse.

The *complications* of gumma are chiefly due to invasion of such important structures as may lie adjacent to the growth. Thus involvement of a vein may cause phlebitis and edema; of an artery, hemorrhage; of a nerve, neuralgia or paralysis; of a bone, necrosis; of a muscle or a joint, mechanical interference with function.

The coexistence of varicose veins of the leg with a confluent gumma is peculiarly adverse to a cure. The impaired circulation greatly interferes with healing.

Diagnosis.—In its first stage the hard, subcutaneous gumma is scarcely mistakable for anything else, though the fact that it is a syphilitic product is often not suspected by the patient or by his physician until it has softened or broken.

In the stage of softening gumma often resembles abscess very closely. It is swollen, red, tender, and painful, even fluctuating. Yet the wary observer will note that fever is entirely absent (or in exceptional cases but a degree or so).

If the mistake is made and the mass incised, the few drops of serous or sero-purulent fluid expressed and the persistence of fluctuation immediately reveal the error.

Differential Diagnosis.—The gummatous ulcer may be mistaken for epithelioma or for varicose leg ulcer.

The differentiation between gumma and *epithelioma* is not

always easy; for gumma may be almost as sluggish as the true neoplasm, and, like it, is very slow to cause enlargement of the adjacent lymphatic nodes.

The ulcers themselves are not dissimilar, though epithelioma bleeds more readily and is more shallow and fungat-

FIG. 45.—MULTIPLE GUMMATOUS LEG ULCERS. Neglect and filth have so altered their syphilitic character (cf. Figs. 43, 44) as to make them resemble varicose ulcers in every respect, except in multiplicity. (Fox.)

ing, while gumma is more excavated, and may have some of its sloughing contents in the base. In some instances it is

impossible to make a diagnosis without examination of a piece of tissue snipped from the growth, and, as already stated, I have known even this test twice to mislead, so that the patient who had syphiloma was treated surgically and vainly for epithelioma. When this is done, the lesion extends until by its circinate shape, its areas of surrounding infiltration, or healing, and perhaps by a closer scrutiny of the patient's history, the true diagnosis is finally made.

Varicose ulcer is rarely mistaken for gumma, but old, gummatous ulcers are very often treated as varicose ulcers, especially if the patient has varicose veins of the leg (Fig. 45).

Fournier estimates that twenty per cent of chronic leg ulcers treated as varicose ulcers are actually syphilitic; yet there are certain striking, differentiated characteristics, as shown in the following table:

SYPHILITIC LEG ULCER	VARICOSE LEG ULCER
1. *Situation.* — Anywhere. Often in calf or in upper half of leg.	1. Only in lower third of leg. Habitually on internal surface.
2. *Number.*—Often multiple.	2. Very rarely multiple.
3. *Shape.*—Round or polycyclic.	3. Irregular.
4. *Edges.*—Sharp, deep, or even undermined.	4. Rounded, shallow, never undermined.
5. *Surrounding Tissues.* — Very slight areola, unless there be old scars or other foci of syphilitic infiltration.	5. Extensively and irregularly pigmented, thickened, and adherent.
6. *Base.* — Granulating or sloughing.	6. No special type.

SYPHILITIC LEG ULCER	VARICOSE LEG ULCER
7. *Other Evidences of Syphilis.*—The history is frequently defective. There are often scars elsewhere on the body.	7. No.
8. *Varicose Veins.*—May be present.	8. Constant.
9. *Mixed Treatment.*—Cures.	9. Ineffectual.

Treatment.—The *general treatment* of gumma is by mixed treatment, with especial insistence on iodids. They may not have to be given in very high dose, but they are practically always necessary to cure the lesion, while mercury is useful as an adjuvant and necessary to prevent relapse.

The *local treatment* is almost nil. In the hard stage, mercurial ointment or plaster may be applied. In the soft stage, the part should be protected from trauma and kept at rest; *but must not be incised under any circumstances*, no matter how fluctuating or how tender it may be.

After the gumma has opened, its healing may be hastened by ordinary surgical cleanliness; gently removing sloughs, and keeping the ulcer bathed in a mild (1 : 5,000) solution of bichlorid if it is inflamed; applying mercurial ointment or plaster if it is chronic and sluggish. For syphilitic leg ulcer complicated by varicose veins, rest in bed is often essential in order to encourage the circulation.

Generally speaking, however, the less done to the ulcer beyond keeping it surgically clean, the quicker it will heal.

Scars left by syphilitic ulcers may demand a plastic operation, but this should not be performed until all active syphilis has ceased for several months.

CHAPTER XXIII [1]

SECONDARY SYPHILIS OF THE MUCOUS MEMBRANES

THE early secondary lesions of the mucous membrane are usually spoken of as mucous patches or mucous plaques. But, inasmuch as these lesions are certainly not patches and very rarely plaques, it is more proper to classify them under their true names of *mucous papules and ulcers.*

Distribution.—They may affect any part of the mucous membranes and also any part of the skin which in texture and moisture simulates the physical conditions of mucous membrane. They are commonest in the mouth and throat and in and about the genitals.

These are among the most important lesions of syphilis; for, although less common in the woman who does not smoke than in the man who does, they are generally among the earliest, the most numerous, and the most constantly relapsing lesions of the disease. Moreover, they are often painless, so that the patient may have a mouthful or a vaginaful of them without knowing they are there, and, finally, they are by far the most infectious secondary lesions.

One marvels that so keen an observer as Ricord should have denied that these secondary lesions are infectious; for certainly they inoculate far more victims than do the primary lesions.

Etiology.—Local irritation is the exciting cause. In the mouth the local irritation of tobacco, whether smoked or

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[1] Reviewed by Dr. L. M. Hurd.

chewed, stands preëminent. About the folds of the skin uncleanliness excites them. In the male they are more common in the mouth than elsewhere; in the female, much more common about the genitals on account of the profuse secretions there.

Classification.—The secondary syphilids of the mucous membranes may be classified as follows:

1. Macular syphilid.
2. Erosive or papulo-erosive syphilid (mucous papule).
3. Ulcerative syphilid.
4. Squamous syphilid.

Of these the macular syphilid is unimportant. The erosive papular and ulcerative types are common in the first year of the disease and occur infrequently for several years thereafter, while the squamous types are rarely seen before the second or third year, and may continue indefinitely thereafter.

THE MACULAR SYPHILID

Roseola of the mouth is not recognized by Fournier; but laryngologists recognize a diffuse symmetrical erythema of the velum and the anterior pillars of the fauces that persists for some weeks and is almost always present at the time the first mucous papules appear in the mouth.

THE EROSIVE OR PAPULAR SYPHILID

The syphilitic papule upon a moist skin or mucous-membrane surface is always eroded. Even upon a dry surface it sheds the superficial epithelium, and, when kept moist, the deeper layers are more freely exposed and exudation from the surface of the papule is thus a constant phenomenon in all papular lesions of mucous or moist surfaces. (In the later syphilids the growth of the papule is slow and is usually

accompanied by an epithelial thickening rather than a thinning; hence it assumes a distinctive squamous type.)

The early erosive papular lesion may be so slight as to produce no perceptible irregularity in the surface. Its top is level with the surrounding mucous membrane, but it is an eroded top, and, consequently, the papule appears as a simple erosion. In a second type the papule is elevated above the surface to a slight degree and becomes a typical, eroded papule, while in the third type it is hypertrophic and becomes a vegetating papule or condyloma. Hence the three types of erosive papular syphilids:

The erosion,

The eroded papule,

The vegetating papule or condyloma.

The Erosion.—The syphilitic erosion is the least characteristic of the lesions of syphilis. It may occur on any moist surface; it may appear and disappear within a day or two, or persist for several days. It is sometimes sensitive or painful; oftener not. It consists simply in an oozing surface bereft of its normal epithelial covering. This surface may be only a little redder than the surrounding tissues, or it may be grayish and opalescent, or it may be covered with a yellowish false membrane from mixed infection. It varies from the size of the head of a pin to that of a split pea, or occasionally larger. Its shape is usually rounded, but may be quite irregular. The confluence of several lesions may form a polycyclic lesion.

Given a single lesion or group of lesions of this character about the mouth or genitals and we may be completely at a loss to diagnose their nature. The lightest cauterization cures them.

The Eroded Papule.—The eroded papule is one of the most characteristic lesions of the disease. It is but slightly elevated above the surrounding surface. Its top is flat or very slightly convex. In size it averages larger than the erosion

(it may even be larger than a ten-cent piece). It is strictly circular in shape. Its surface is eroded and moist; sometimes smooth, but oftener slightly roughened. It is rather dark red in color, rarely grayish or covered with a false membrane. The eruption may be confluent and polycyclic. There may be a slight tendency to vegetation, in which case the surface of the papule are broken into little warty projections. The eroded papule is usually quite soft, though about the genitals —especially about the vulva or the preputial frenum—the base

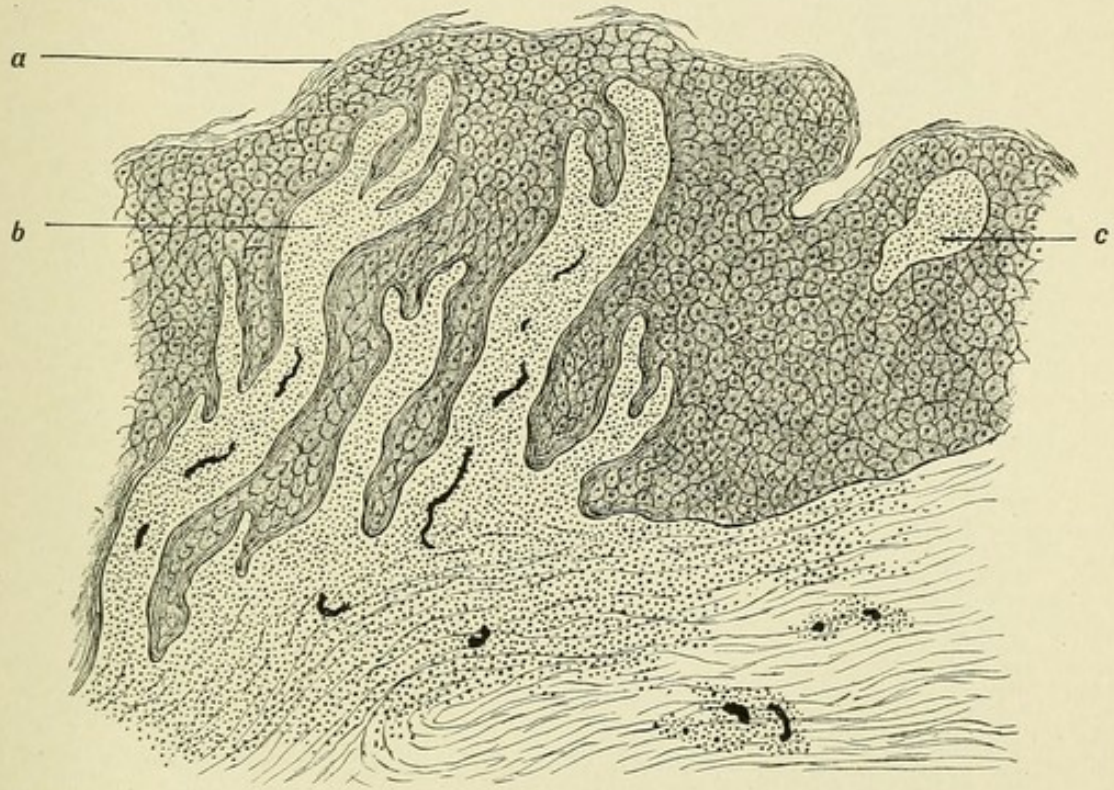

FIG. 46.—PATHOLOGY OF CONDYLOMA. (Diagrammatic.) *a*, desquamating epithelium. *b*, greatly hypertrophied and infiltrated papilla of derma. *c*, tip of papilla in cross-section.

may be slightly indurated (the indurated chancriform syphilid of Fournier).

The exudation from the surface of the papule is serous, irritating, and foul-smelling. The peculiar acrid odor (though scarcely pathognomonic) is very suggestive and very penetrating. The exudation from the lesion irritates the surround-

ing tissues and even irritates the lesion itself, causing the tendency to vegetation so noticeable when the erosive papule occurs upon the skin.

The Vegetating Papule or Condyloma.—The eroded papule upon the skin or about the orifices of the anus or the

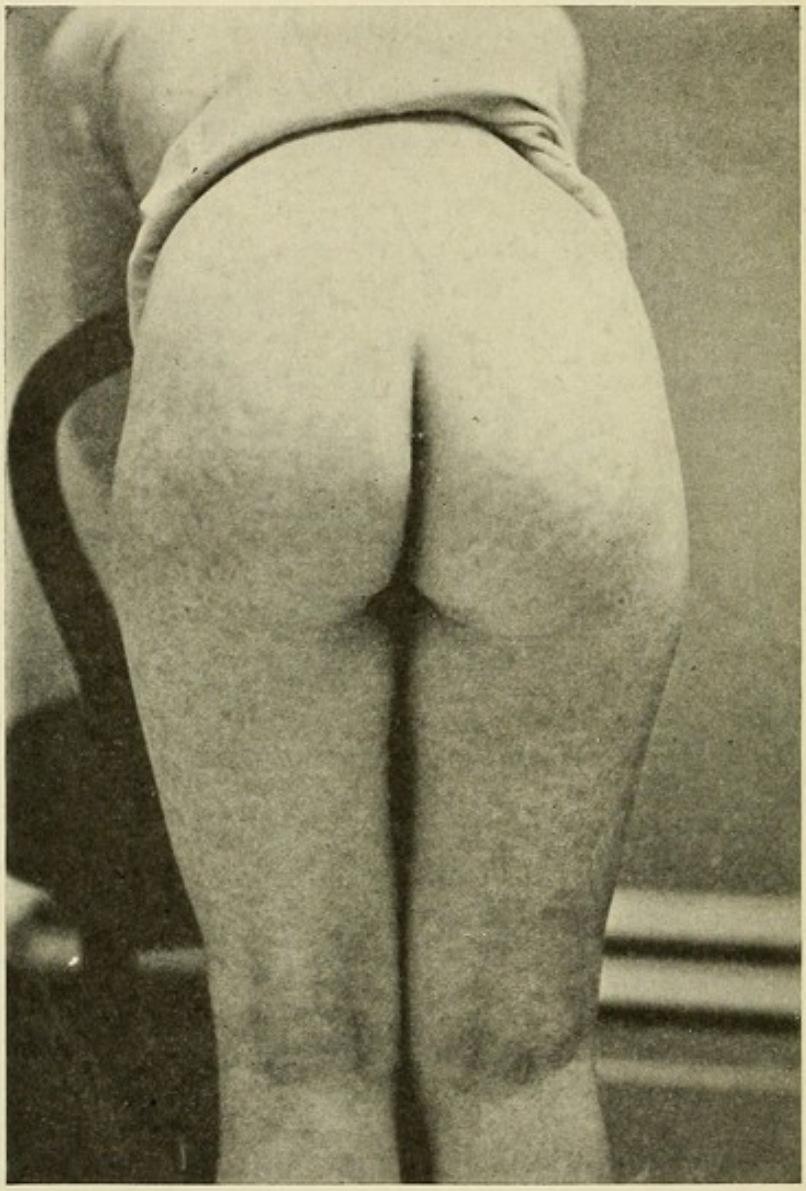

FIG. 47.—MACULAR SYPHILID AND CONDYLOMATA. (FOX.)

vagina, if permitted to bathe in its own secretions, rapidly hypertrophies. The infiltrated papillæ of the skin, instead of remaining distorted and slightly swollen, as in the typical

syphilitic papule, grow and branch out into numerous irregularly shaped fingers, while the deeper layers of epithelium sur-

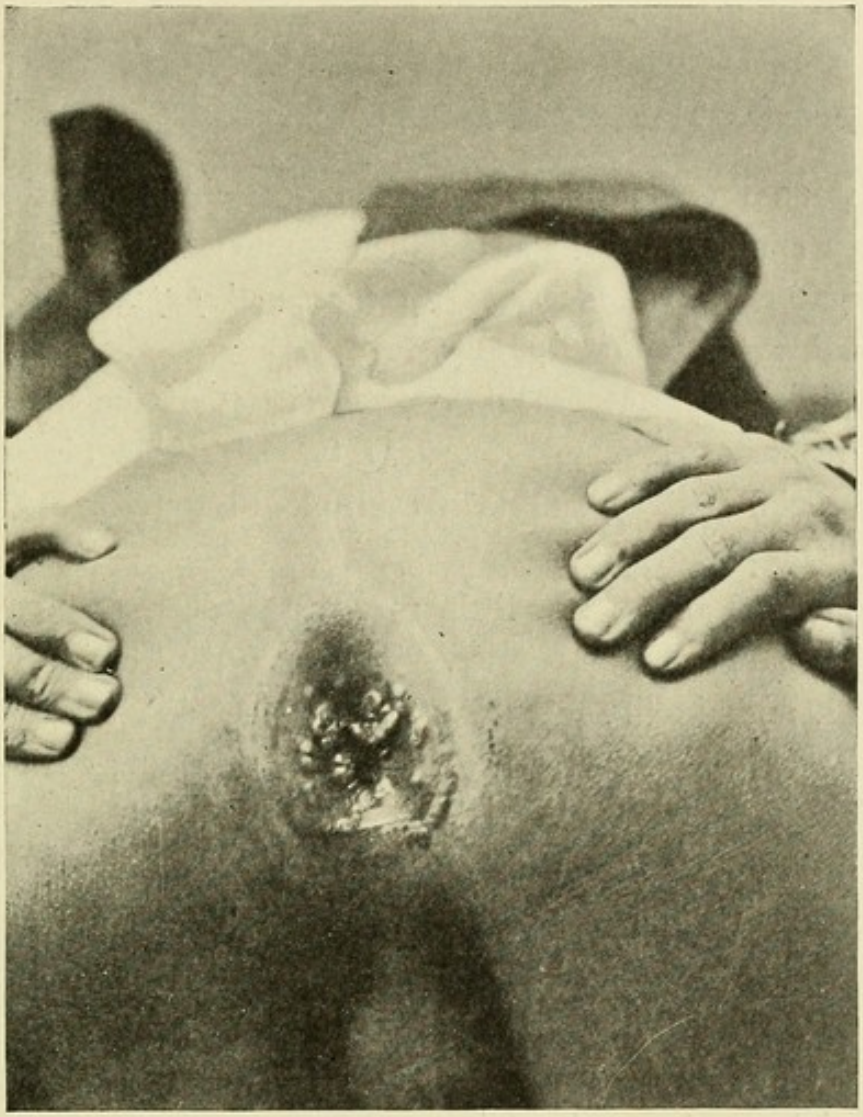

FIG. 48.—CONDYLOMATA ABOUT THE ANUS. (FOX.)

rounding them are much thickened by swelling of their cells (Fig. 46).

These hypertrophic papules usually occur in groups forming irregular masses, which may cover an area two or three inches in diameter and may rise half an inch or more above the surface. The striking characteristic of this lesion is that, though a vegetating one, the vegetation is relatively low compared to the extent of the base; hence it is commonly known

as *condyloma latum*. Its foul secretion, its irregular, broken, vegetating, and eroded red surface constitute amplifications of the characteristics of the eroded papule (Figs. 47, 48).

THE ULCERATIVE SYPHILID

Just as the papule may appear as a simple erosion, an irritated papule, or a hypertrophied papule, so the ulcer may appear as a *simple ulcer*, as an *ulcerated papule*, or as an *ulcerated hypertrophied papule*.

The Simple Ulcer.—The characteristics of the ulcer, like those of the erosion, are not very typical. The ulcers may be superficial or deep, simple or multiple, discrete or confluent, rounded or polycyclic.

Superficial ulcers are little more than deep erosions and have the same characteristics. The deeper ulcers are slow-growing, and, in the mouth, usually collect about the tonsils and fauces. Their edges are raised, sharply cut; their bases smooth, unindurated, and red or yellow. They look a great deal like chancroid, and exude true pus. Exceptionally they are irregular in shape and have no distinguishing characteristics. When occurring at the angle of the mouth, or at any other fold, they are likely to be deeply fissured.

The Ulcerated Papule and the Ulcerated Condyloma.—Superficial or deep ulceration may occur upon an eroded papule or upon a syphilitic condyloma. Such ulcerations have no very special characteristics.

THE SQUAMOUS SYPHILID

The squamous syphilid of the mucous membranes (syphilitic leukoplakia, tylosis, milk patch, pearly patch), though a secondary manifestation, is closely allied to certain more important tertiary manifestations. It is secondary in that it is

curable, like other secondary mucous-membrane lesions, by mild cauterization and mercury, and in healing leaves no scar. Yet it is most commonly seen late in the disease upon the tongue, where it assumes the position of the most superficial form of tertiary sclerosis. It may seem contradictory to speak of a secondary form of a tertiary lesion; but this may serve to impress the fact that the division of the lesions of syphilis into secondary and tertiary is purely clinical and arbitrary. We assume the general distinction for the sake of convenience, and, in this instance, we dismiss it—for the same reason.

The secondary squamous syphilid will, accordingly, be described with tertiary sclerosis of the tongue.

DIAGNOSIS

Erosion and Ulceration in the Mouth.—While the flat, erosive papule of syphilis is usually quite characteristic, the erosion, the ulcer, and even the ulcerated papule, may be indistinguishable from certain non-specific lesions. The most important lesion to be differentiated is the ulceration due to mercurial stomatitis.

Mercurial ulceration may follow close upon the syphilitic ulceration for which the mercury is given. I have known patients to go through this transition quite unconsciously, and since, with additional mercury, the ulceration progressively increases, they rapidly reach a fine state of salivation.

I know no pathognomonic characteristic to distinguish the ulcer of syphilis from that of mercury, but the latter rarely occurs upon the fauces or upon the dorsum of the tongue or in the angle of the lips, where syphilitic ulcers are common; while *it is almost typical of mercurial ulceration to find a lesion on the cheek or gum back of the last molar tooth, or about the upper or lower central incisors.*

The ulceration back of the molars is usually quite large,

deep, and painful. That about the incisors is associated with retraction of the gum, and other ulcers may be found wherever there is a broken or an irregular tooth.

Confirmatory evidence may be looked for in the presence or absence of the usual signs of salivation; yet there may be mercurial ulcers with very slight general salivation. The only way to prove the nature of the lesion is to stop the mercury and redouble the efforts at cleansing the mouth (page 163). All cauterization should be eschewed and one should await developments. If the ulcers are due to mercury they will soon disappear, unless the patient has been severely salivated; if due to syphilis they will multiply. Unfortunately, since mercury decimates the spirochetæ, microscopical examination of a smear is futile.

Aphthous erosions are very common in syphilis and accurately simulate syphilitic ulcers of the mouth, except that the aphthous spots are usually intensely irritable and quite painful, whereas the small superficial syphilitic ulcer is usually, though not always, relatively painless. Aphthæ always remain very small.

Herpes of the mouth is also quite frequent. It begins as a single, little opalescent papule or a group of such papules. It rapidly becomes eroded and its surface whitish. Finally, all the epithelium falls, and it remains a reddish, little, round, superficial erosion. Its distinguishing characteristics—in contrast to the syphilitic erosion—are the smallness of the single lesions, the frequency of a group of lesions, the polycyclic and microcyclic character of the confluent lesions (page 245).

Fournier attaches great clinical importance to this last characteristic. He also states that, at any stage of the eruption, one is likely to find isolated miliary erosions no larger than the head of a pin in the neighborhood, and that if the eroded lesion is surrounded by a collarette of whitish epithelium, it is not syphilitic.

Besides the common lesions of aphthous stomatitis and herpes, many other rarer conditions simulate the ulcerative or erosive syphilid of the mouth. Thrush, diphtheria, and hydroa may produce similar lesions, while simple erosions due to burns, etc., may be quite indistinguishable from syphilis.

The differential diagnosis of any of these lesions must often be left to time, and the ablest diagnostician will confess that, in many instances, a number of lesions have come and gone before he was able to state definitely that they were or were not syphilitic.

Erosion and Ulceration within the Female Genitals.—*Simple erosions due to endometritis* may appear on the cervix or elsewhere within the genital passages, and, for a time, at least, simulate syphilitic erosions; but, like erosions of simple balanitis, they are changeable, fugitive, and readily cured.

Chancroid about the orifice of the female genitalia may suggest syphilitic ulceration. It should be distinguished by autoinoculation.

Mucous Papules of the Skin of the Genitals and Elsewhere.—The soft, flat, eroded papule can scarcely be mistaken for any other lesion unless it has been partly healed by the application of a drying powder. In this semihealed state it may simulate the papules of scabies, but is distinguishable from them by the absence of itching.

Condylomata Lata.—The fungating syphilid may, in some degree, resemble so-called venereal warts, or condylomata acuminata; but the titles of the two lesions suggest their striking difference.

The simple wart is sharp and projects relatively high from a small base, while the syphilitic wart has a broad implantation and a relatively slight elevation. The application of any simple dusting powder to the syphilitic condylomata will rapidly shrivel them and effect a cure while establishing the diagnosis.

TREATMENT

The general treatment of all these lesions consists in the administration of mercury. But, inasmuch as the marked tendency to relapse is due to local causes, the cure is the removal of these local causes and local treatment, rather than vigorous, general antisyphilitic treatment.

Indeed, one often sees cases in which mercury and iodids have been vainly pushed to the limit of toleration promptly recover under mild mercurial treatment aided by appropriate local measures. The *local treatment, therefore, is much the more important.*

Local Treatment of Lesions upon the Skin.—If the lesion is upon the skin it is usually possible to keep it dry, and this drying will cure *erosion or vegetation* as if by magic. In mild cases all that is necessary is to cleanse the affected region thoroughly twice a day with warm soap and water, then dry and apply pure calomel or calomel mixed in equal parts of stearate of zinc or talcum.

For extensive condylomata it may be necessary to paint with a solution of permanganate of potash (1 : 1,000) twice a day and to interpose a layer of absorbent gauze between the various inflamed surfaces after powdering the whole region thickly.

For subpreputial erosions when inflammatory phimosis prevents retraction of the prepuce, one may inject, twice a day, a four per cent resorcin solution, or a 1 : 2,000 permanganate-of-potash solution. If this does not speedily cure, circumcision should be performed.

For *ulcers,* cauterization twice a week with the nitrate-of-silver stick is an important accessory.

Local Treatment of Lesions within the Mouth.—It is impossible to keep these lesions dry, hence they are often more difficult to manage than the external lesions.

The most important point in local treatment is mild cauterization, and the best cauterizing agent is the officinal *liquor hydrargyri nitratis*, either pure or diluted to one half or one quarter strength. The pure fluid can be applied only to small lesions, as it is quite painful. *This application should be made not oftener than twice a week*, and should never be confided to the hands of the patient, since overcauterization makes the lesion worse instead of better.

The *nitrate-of-silver* stick often acts quite as well as the acid nitrate of mercury, and like it may be applied twice a week.

Crystals of *copper sulphate* may be applied to the lesion by the patient himself, and may often be used with advantage every other day, but are not nearly so effective as the mercury or silver.

In order to prevent relapses of secondary syphilids within the mouth, it is often necessary for the patient—

To stop smoking and chewing tobacco;

To keep the teeth and gums in perfect condition;

To employ twice daily an antiseptic mouth wash (page 163).

SECONDARY SYPHILIDS OF THE EAR

The lesions of secondary syphilis in and about the ear are both rare and unimportant.

In the *external auditory canal* mucous papules and condylomata may occur. Careful examination readily distinguishes the latter from sessile polypi.

The *treatment* is by drying powders.

In the *Eustachian tube* mucous papules (usually extending from similar lesions in the naso-pharynx) may cause obstruction, resulting in—

1. Partial deafness, buzzing in the ear, and sometimes vertigo; and

2. Acute otitis media from mixed infection due to the obstruction. (I have record of two such cases.)

The *treatment* consists of douches to the nose, while mercurial treatment is pushed and hot douches applied to prevent otitis (or myringotomy to cure it). No attempt should be made at dilatation, catheterization, or Politzerization of the canal.

In the *inner ear* secondary and tertiary lesions are clinically indistinguishable (page 367).

CHAPTER XXIV [1]

TERTIARY SYPHILIS OF MOUTH, PHARYNX, NOSE AND EAR

In this chapter are collected a great variety of lesions connected only by an anatomical relation. Clinical convenience bids us throw them together. They are the tertiary lesions:

1. Of the lips.
2. Of the tongue, including leukoplakia.
3. Of the tonsil.
4. Of the soft parts of the pharynx.
5. Of the soft palate (velum).
6. Of the hard palate.
7. Of the nose.
8. Of the ear.

Occurrence.—The table on next page shows the frequency and date of occurrence of these lesions (excepting the lips and ears). The last column sums up the gummatous and ulcerative lesions for contrast with the purely sclerotic lesions in the first column. The only striking difference thus shown is that the former are relatively much more frequent than the latter in the first two years and after the fifteenth year of the disease. The occurrence of each class becomes about fifty per cent less frequent with each half decade.

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[1] Reviewed by Dr. L. M. Hurd.

ONSET OF TERTIARIES ABOUT THE MOUTH AND NASO-PHARYNX

YEAR	Tongue Leukoplakia and Sclerosis	Tongue Gumma and Ulcer	Tongue Sclero-Gumma	Pharynx	Nasal Septum	Tonsil	Velum	Palate	Total Gummata
1.....	2	4	.....	5	1	3	5	3	21
2.....	14	.....	3	4	6	7	3	4	27
3.....	20	4	1	8	6	2	4	.....	25
4.....	19	4	1	4	5	2	2	2	20
5.....	11	.....	2	1	3	3	1	1	11
6.....	12	2	.....	4	7	4	2	.....	19
7.....	9	1	.....	5	3	2	1	1	13
8.....	2	1	.....	1	1	.....	.....	3	6
9.....	2	1	.....	.....	3	.....	1	1	6
10.....	7	.....	.....	3	.....	1	.....	1	5
11.....	4	1	.....	3	2	.....	2	1	9
12.....	2	.....	2	2	1	1	1	2	9
13.....	3	.....	.....	3	1	.....	.....	.....	4
14.....	3	1	1	1	.....	.....	1	.....	4
15–19.....	2	2	2	.....	4	2	1	1	12
20–24.....	3	2	1	2	.....	.....	.....	1	6
26–41.....	2	.....	.....	1	3	.....	.....	.....	4
Indefinite..	12	1	.....	7	10	.....	5	7	30
Total...	129	24	13	54	56	27	29	28	231

Influence of Tobacco.—The influence of tobacco in exciting leukoplakia and sclerosis is great, while it excites (tertiary) ulceration much less and gumma not at all. The most striking evidence of this is the fact that only two cases of leukoplakia are recorded among women, while of the gummatous and ulcerative lesions, 31 (tongue, 4; pharynx, 7; septum, 8; tonsil, 3; velum, 6; and palate, 4) occurred in women.

Relapses.—Though leukoplakia is hard to cure and may defy treatment for many years, only four relapses are recorded (six to seven to nine to twelve years, four to six years, four to twelve years, six to fifteen years) in cases cured and remaining well one year.

Gummata, though readily curable, show relapses in tongue (four to six years, twelve to seventeen years, sixteen to eighteen years), pharynx (two to six years, four to five to eight years, eleven to thirteen years), and tonsil (two to four years, seven to nine years). No relapses are recorded in cured gummata of velum; palate and septum show one each (one to four years and two to six years, respectively).

Concurrence of Lesions.—The relative frequency of simultaneous or successive outbreaks in two or more of the regions under discussion is not great. The 360 lesions [1] occurred in 321 patients, there being 36 duplications and two triplications (velum-tonsil-pharynx and velum-palate-septum).

This concurrence is usually due to direct extension of a phagedenic lesion from one tissue to an adjoining one, but sometimes to what might be termed a "regional" tendency in the outbreak. The case already related [2] (page 20) is an excellent example of this. The tongue gumma and the necrosis of the mandible were regionally, but not directly, connected with the tonsil-velum lesions.

These concurrences are separable into three classes:

Sclero-gumma of tongue—13 cases.

Leukoplakia and lesions of tonsil or pharynx—9 cases.

Septum and palate or velum—7 cases.

TERTIARY LESIONS OF THE LIPS

The striking characteristics of *diffuse, tubercular* syphilitic *infiltration* about the lips and nose have already been mentioned (page 312). The distinction between gumma and chancre of the lip has been dwelt upon (page 249), and an instance of phagedena in lip chancre cited (page 231).

Finally, there may be added to the other diffuse infiltra-

[1] Excluding recurrences.

[2] Not included in these statistics.

tions grouped under the head of "leontiasis," a *thickening of the lips without a tendency to gummatous degeneration and without surface lesions*, though it may be accompanied by them. This process is a diffuse, interstitial sclerosis. The lips look as though enlarged by edema, but, though they yield to pressure, they do not pit. The condition is often associated with sclerotic glossitis.

TERTIARY LESIONS OF THE TONGUE

Tertiary syphilis of the tongue appears under two forms, the sclerotic and the gummatous. Inasmuch, however, as sclerosis of the tongue is closely allied to the secondary, superficial squamous syphilid of the tongue, this will be included. Accordingly, the lesions to be discussed are leukoplakia, interstitial glossitis, and gumma.

LEUKOPLAKIA

By leukoplakia (tylosis, milk spot, etc.) is meant a chronic inflammation of the mucous membrane characterized by the appearance of pearly white or bluish white patches. These patches may appear anywhere inside the mouth, but, while extremely common upon the tongue, they are very rare elsewhere. I have record of only seven cases occurring on the mucous membrane of the cheeks, and I have recently seen one case in which the change was entirely confined to the gums; but it is seen most constantly and typically upon the tongue.

Etiology.—It is still a matter of dispute what proportion of cases of leukoplakia are due to syphilis.[1] Of late years we have appreciated more and more how important a factor syphilis is; for leukoplakia of the mouth, like the squamous

[1] Cf. Marshall, *Treatment*, 1906, January; also Chichkoff, *These de Paris*, 1902.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

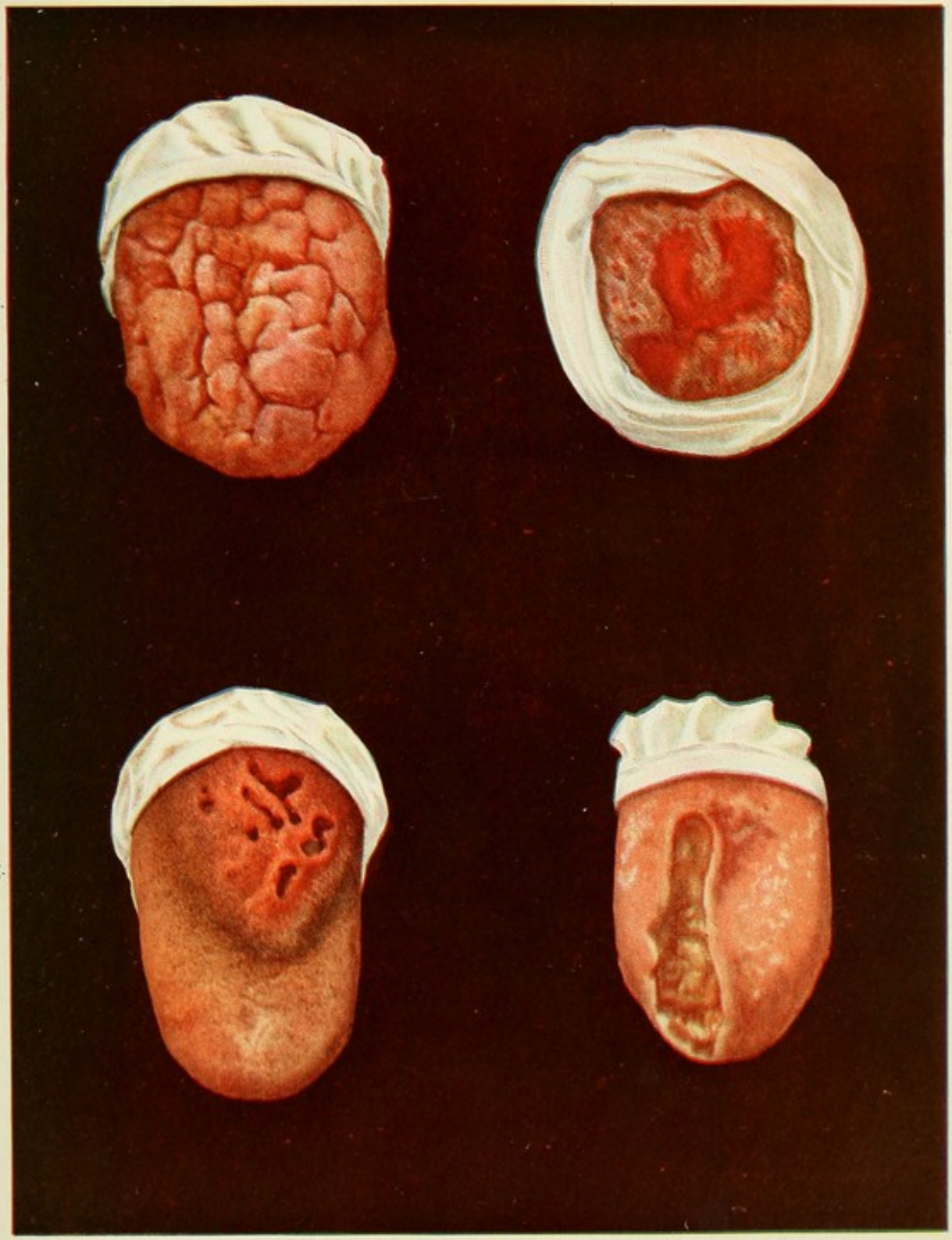

PLATE IX.—Tertiary Lesions of the Tongue.

Fig. 1.—Interstitial glossitis. (Musée, St. Louis.)

Fig. 2.—Leukoplakia (superficial glossitis). (Musée, St. Louis.)

Fig. 3.—Gumma of the tongue. (Musée, St. Louis.)

Fig. 4.—Superficial tertiary (gummatous) ulcer of the tongue. (Musée, St. Louis.)

syphilid of the palm, is very rebellious to specific treatment, and it is only since the introduction of the injection method that the majority of these cases have been proven curable by mercury, and therefore shown themselves to be syphilitic.

But if most are syphilitic, a certain few surely are not; and, while it may be impossible in a given case to say beforehand whether or not the leukoplakia is due to syphilis, one encounters certain cases in which all history of syphilis is denied, in which no antisyphilitic treatment has any curative effect, and in which the lesion itself has a slightly elevated and papillomatous character, not in the least suggestive of syphilis. Indeed, I have seen one case in which leukoplakia preceded the chancre and continued throughout the syphilis that followed.

Smoking is the real exciting cause. Leukoplakia is recorded only twice among women (one per cent), as against 141 times among men (six per cent). Yet one occasionally sees it in the mouth of a syphilitic who has never smoked.

Characteristics of the Lesion.—The typical lesion of leukoplakia consists in a whitish, grayish, or opalescent discoloration of the mucous membrane. It has a thickish look but no palpable density, and is not perceptibly raised above the surrounding surface.

The lesions are usually multiple and vary widely in size and shape. The smaller ones are usually oval, and the larger confluent lesions may have circinate edges; but, in many instances, although this circinate character may be discerned at some part of the patch, *the general outline is quite irregular.*

Intermingled with these typical lesions one commonly sees reddish areas where the mucous membrane of the tongue has lost its papillary character (the *glossite dépapillante* of Fournier), and presents a glistening, polished aspect. In other places the surface may be eroded or ulcerated and the tissues

of the tongue thickened by sclerotic glossitis, or (rarely) destroyed by gumma.

These lesions occupy the terminal third of the tongue and are usually grouped about its edge, whence they extend commonly over the dorsum, rarely very far on to the under surface.

All of these lesions are based on the one pathological process. The depapillated patches represent areas of squamous syphilid. The leukoplakia is the same process with a greater thickening of the epithelium; while sclerotic glossitis simply represents a deep extension of the same process, and erosion, ulcer, and gumma are expressions at different depths of the degenerative tendency of the sclerotic tissue.

Diagnosis.—The appearance of such a polymorphic lesion as that just described is pathognomonic of syphilis. But when, as sometimes occurs, the whitened patches of leukoplakia appear unaccompanied by any of these kindred changes, and unassociated with syphilitic lesions elsewhere in the body, one may doubt their nature. It is safe, however, to assume that every leukoplakia is syphilitic until it is proven not to be. Presumptive evidence of syphilis is afforded by the history, the presence of other syphilitic lesions, the coexistence of ulcer or gumma upon the tongue, while the absence of syphilis is presumptive if the lesion is papillomatous. The test of treatment alone decides the case; but in chronic severe cases it may be impossible to cure a truly syphilitic lesion.

Prognosis.—Leukoplakia is one of the most inveterate lesions of syphilis. Cases lasting four or five years are not uncommon, and one occasionally sees them continue for ten or twenty. The most important element in prognosis is the use of tobacco. If the patient will give this up while the disease is yet young he may expect an entire cure; but if he keeps up the irritation of smoking or chewing tobacco, no amount of treatment is likely to cure the lesions. On the other hand, after the case has been neglected for five or ten years, the

lesions become so chronic that they are often incurable under any circumstances.

The most important question in the prognosis of leukoplakia is the occurrence of *epithelioma*. Most authors speak of it as common. But among my cases I have known only two to undergo epitheliomatous change. Yet I have seen 24 cases in which the lesions had lasted more than three years, while in 8 of these the lesions had lasted from ten to fifteen years.

The two cases of epithelioma occurred one twenty-seven years, the other thirty years after the chancre. In the latter case the leukoplakia was known to have existed for twenty years.

Treatment.—There are three essentials to treatment:

1. *Removal* of irritation, notably the irritation of *tobacco*; less frequently that of a jagged tooth or of spicy viands.

2. *Repeated cauterization* with liquor hydrargyri nitratis, as for the secondary lesions. The more superficial the leukoplakia, the more effective this cauterization. If, after repeated efforts, cauterization fails, it is vain to continue it indefinitely. The actual cautery has been employed (Barthélemy, Perrin) to burn out the lesions with alleged excellent results, but I have never used it.

3. *Specific treatment.* Unless there is ulcer or gumma accompanying the leukoplakia, little good may be expected from the use of iodid. Small doses of iodid may be employed with the mercury, but this latter drug is our chief reliance. In early and superficial cases mercury, administered by mouth or by inunction, together with the stopping of tobacco, and cauterization with the liquor hydrargyri nitratis will often cure. But if these fail, or if the condition persists in relapsing, recourse should be promptly had to injections of insoluble salts of mercury, lest too great delay permit the lesion to become so ingrained that no cure is possible.

DIFFUSE SCLEROTIC GLOSSITIS

The lesion is a diffuse sclerosis in the muscle of the tongue. It is quite rare and is always accompanied by superficial glossitis, either simple depapillation or leukoplakia.

It appears as a thickened and dark red induration within the substance of the tongue, and surmounted by an area of leukoplakia or of glossy infiltration. There are usually several such indurations, and the process may involve the whole tongue.

In such cases the tongue is much thickened; its dorsum is lobulated and shining, with only here and there an irregular area in which the normal papillæ remain. These papillæ are rather abnormally white. The tongue itself has a hard, cartilaginous density; it is insensitive and clumsy. The sclerosis is not painful, but secondary fissures and excoriations occur which are quite sensitive. With cessation of the active syphilitic process there is a gradual and progressive shrinking of the scar tissue, which leaves the tongue much smaller than normal.

The *prognosis* of interstitial glossitis, like that of all syphilitic interstitial inflammations, is extremely bad. If attacked early and vigorously by hypodermic injections of mercury, it may be cured in many instances, and at least checked in most; but if neglected, and especially if irritated by continued smoking or chewing of tobacco, it becomes absolutely intractable to treatment; for, though the syphilitic process may still be stayed, the scar remains indelible.

GUMMA OF THE TONGUE

Gumma of the tongue is relatively rare. One third of the cases are associated with sclerosis of the tongue.

Gumma may be single or multiple, superficial or deep. Its general characteristics are comparable to those of the same lesion in the skin.

In its hard stage it appears as a deep, submucous lump, which softens, resembles an abscess, and then breaks, leaving a characteristic deep syphilitic ulcer.

Gumma commonly opens in the dorsum of the tongue, rarely upon its edge, never upon its under surface. It may affect any part of the tongue from the epiglottis to the tip. It is usually a rapidly growing lesion.

It is curable by mixed treatment, with insistence on iodids.

It is to be distinguished from epithelioma by the characteristics indicated in the following table:

EPITHELIOMA	GUMMA
1. *Occurrence.* — Almost exclusively in men over forty-five years of age.	1. Any age; either sex. History of previous syphilis.
2. *Situation.* — Usually on edge; may be on under surface.	2. Usually on dorsum. Never on under surface.
3. *Number.*—Single.	3. May be multiple.
4. *Leukoplakia.* — May precede.	4. May accompany.
5. *Characteristics.* — A shallow, fungating ulcer, with thick, everted edges, upon a base of wooden hardness. It bleeds very readily; its discharge is foul.	5. A deep, sloughing ulcer, with sharp, undermined edges, upon a moderately indurated base. It does not bleed readily; its discharge is not very foul.
6. *Pain.*—Severe and lancinating.	6. Slight or absent.

EPITHELIOMA	GUMMA
7. *Lymph Nodes.*—Soon and progressively enlarged.	7. Enlargement slight or absent.
8. *Biopsy.*—Epithelial pearls.	8. Gumma.
9. *Treatment.*—No medicines have the least effect.	9. Mixed treatment cures.

The differentiation from tuberculosis has already been given (page 251).

TERTIARY LESIONS OF THE TONSIL

The tonsil, like the tongue, is subject to gumma and to interstitial infiltration. But infiltration of the tonsil is only slightly more common than gumma. It is not accompanied by any leukoplakia upon the surface and is usually quite readily curable. It does not, therefore, assume the importance of the sclerotic lesions of the tongue, and is generally overlooked.

Infiltration of the Tonsil.—The lesion comes on quite rapidly and either causes no sensation at all or else excites a slight soreness in the tonsil.

Inspection reveals a characteristic picture. The tonsil looks as though it had been compressed from before backward. Instead of projecting as a round mass it has a sharp, notched edge. We may characterize it as "hatchet-edged." In color it is dark, its surface is dense and somewhat glossy. The normal pittings are few and shallow.

TREATMENT.—This lesion yields kindly to mixed treatment. Cauterization is harmful.

Gumma.—I have never seen tonsillar gumma before ulceration. When ulcerated it appears as a deep, sloughing, sharp-edged mass surrounded by a dark indurated areola. Contrary to the usual rule, the glands behind the angle of the

jaw may be inflamed and sensitive (mixed infection). The tissues about the tonsil are usually somewhat swollen; exceptionally there is a complicating acute tonsillitis, with fever and great pain. In such cases it is impossible to make the diagnosis until the acute attack has passed by.

Phagedena is rare, but I have recently seen a case in which gangrene of the velum originated in a tertiary lesion of the tonsil.

TREATMENT.—Mixed treatment. No cauterization.

TERTIARY LESIONS OF THE PHARYNX

The tertiary lesions of the pharynx may originate in the mucous membrane, in the submucous tissue, or in the bones. The lesion always begins as an infiltration, and this quickly breaks down.

Thus the three types are: Ulcerative tubercular syphilid, submucous gumma, and gumma of the bone.

The lesions may be circumscribed or diffuse, single or multiple. Though phagedena is infrequent, the pharynx is sometimes involved with the velum in a phagedenic process (see below). The lesions are sometimes quite painful, and may, by shutting off the Eustachian tube, give rise to suppurative complications in the middle ear.

TREATMENT.—Cauterization is harmful; mixed treatment effects a cure. The treatment of adhesions following phagedena is mentioned on next page.

TERTIARY LESIONS OF THE VELUM

The velum, or soft palate, may be the site of *interstitial sclerosis* or of superficial (tubercular syphilid) *circumscribed ulcers*, but these lesions are extremely rare and relatively unimportant.

The typical and terrible lesion of syphilis in the velum is *the phagedenic gummatous ulcer* (Fig. 49).

This begins as a diffuse infiltration, producing redness, tumefaction, and relative immobility of the velum. But it causes scarcely any pain, and the patient usually pays no attention to it until, suddenly, ulceration and phagedena set in.

The onset of phagedena is exceedingly rapid. The patient goes to bed at night thinking himself well, and awakes with

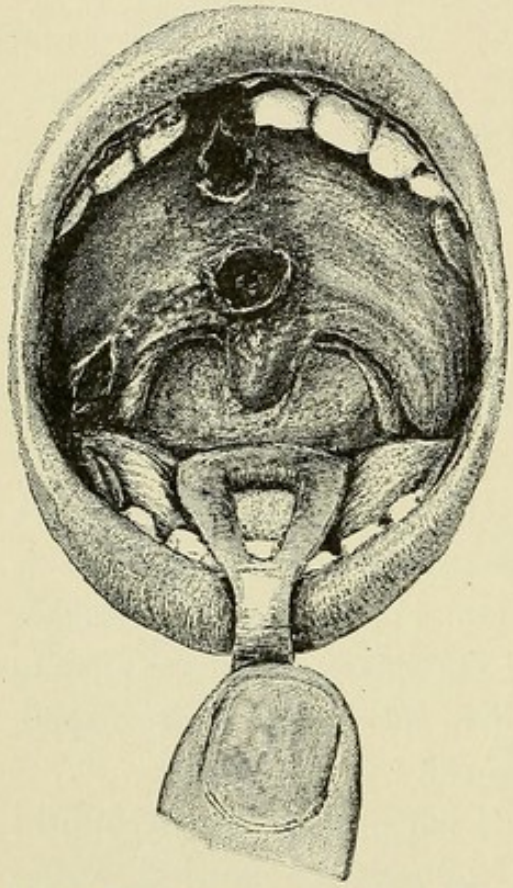

FIG. 49.—ULCERS OF THE SOFT PALATE. (Shurley, after Du Blois.)

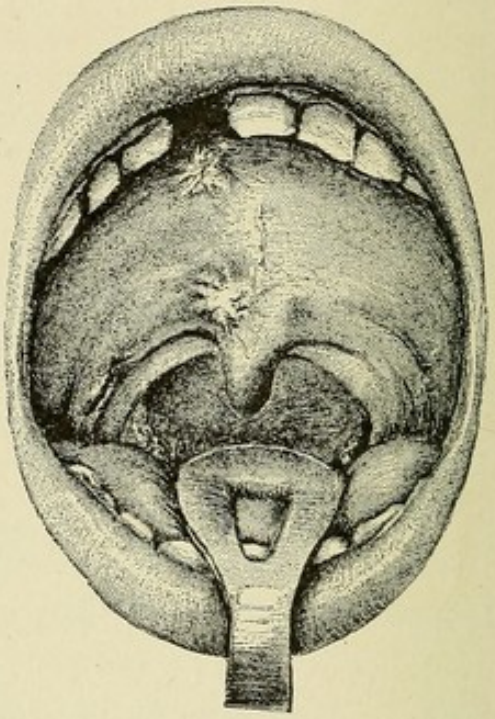

FIG. 50.—THE SAME: HEALED. (Shurley, after Du Blois.)

a hole in his palate, or with great patches of necrosis replacing what the day before was a comparatively innocent, painless infiltration. Even now the pain is not great, and the discomfort is inconsiderable. But the foul odor from the sloughing tissue, or the sense of a hole in the velum, soon brings the patient to his physician.

Examination then reveals either an area of dark greenish slough in the palate, or else a perforation surrounded by a slough. If the perforation is of any size the voice assumes a nasal quality and the breathing may be accompanied by a rattling gurgle, due to the flapping to and fro of partially detached bits of the palate. When the patient tries to swallow he has great difficulty in preventing fluids from regurgitating through the perforation into his nose. Before long, if the lesion is a severe one, the surrounding tissues, the palate, the pharynx, the pillars of the fauces, the tonsils, even the base of the tongue are invaded, and, on account of a mixed infection of this large, sloughing area, the patient is racked with fever and his neck is swollen by inflamed, sensitive nodes.

Prognosis.—The prognosis of many of the lesions of syphilis is absolutely under the control of the physician. But that of gangrene of the velum is often a matter of fortune. Some cases, even though untreated, produce only a slight gangrene with infinitesimal perforation; indeed (most exceptionally) there may be no perforation at all. But in the ordinary case a considerable part of the palate is destroyed, leaving a deformity which consists either of a perforation or of an extensive erosion of the free border of the palate with destruction of the uvula. If the perforation of the palate is small, not more than one quarter of an inch in diameter, its ultimate healing may be expected (Fig. 50), and in order to encourage this healing *after the active process has been controlled*, the perforation may be lightly touched every third day with the solid stick of nitrate of silver. In the meanwhile the patient may wear an obturator in order to prevent irritation of the hole by reflux of fluids through it.

Those unfortunate victims whose lesions extend to the pharynx are left in a truly pitiable condition. For not only does the deformed velum permit reflux of fluids into the nose,

but also the healing of the extensive ulceration is followed by adhesion of the remains of the velum to the lateral or posterior pharyngeal wall. As these scarred tissues heal they contract until the posterior nares, or even the pharynx itself, is almost completely obliterated, gravely interfering with deglutition, speech, and even breathing.

TREATMENT.—As stated above, the extent of the lesion depends more upon good fortune than upon medical skill. So rapidly does the gangrene spread that it is impossible to check it immediately by any drugs.

The patient should be put in the best hygienic surroundings possible and overdosed with milk if he can swallow it. In the meanwhile mercury is given by inunction or by hypodermic, and iodids pushed as rapidly as may be. In a severe case it is proper to push the iodids to the point of rendering the patient moderately delirious; but, once this point of intoxication is reached, all internal medication must be stopped, whether the lesion is conquered or not; for to keep the patient poisoned with iodid will never cure the lesions of syphilis. When the active process is checked, either by the lapse of time or the effect of medicine, the patient must be closely watched to prevent adhesion of the palate to the pharynx. This may be in large measure prevented by occasionally hooking out the palate; but if such efforts fail, it may have to be freed by a plastic operation, the detail of which differs with every case. An obturator [1] may be applied after the active process subsides, and kept in place until all perforations are healed. For a persistent perforation the obturator may be worn permanently, or an attempt may be made to close the hole by a plastic operation.

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[1] Cf. Schadle, *St. Paul Med. J.*, 1905, March.

TERTIARY LESIONS OF THE PALATE

Superficial infiltrations and ulcerations of the hard palate, like those of the velum, are both rare and unimportant, while *perforating gumma* of the palate is as insidious, as rapid in evolution, and almost as destructive in its results as the same lesion in the velum.

Gumma of the hard palate usually begins upon its nasal surface as a submucous infiltration. Since it causes neither pain nor obstruction, the patient does not notice it until he is surprised to find a hole through the roof of his mouth.

The earliest one sees these lesions is when the necrotic bone has not been separated, but appears as a black, sloughing spot, or a black or white sequestrum of dead bone surrounded by an angry inflamed circle in the midst of the palate. Happily this perforation is usually very small. Exceptionally (seven times in 28 cases) it is associated with necrosis of the nasal septum;[1] still more rarely (twice) with gangrene of the velum.

TREATMENT.—Sequestra must be removed as soon as they separate. High doses of iodid must be depended upon to control the progress of the lesion. Its healing (after the active process has been checked) may be encouraged by an obturator and applications of nitrate of silver, and if the perforation cannot be closed by this means, the patient must choose between obturator and operation.

TERTIARY LESIONS OF THE NOSE

Gumma.—Gumma of the nasal bones is always[2] insidious and rapidly destructive in its onset, like that of the palate and the velum.

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[1] The frequency of such complication depends chiefly upon the length of time the lesion is neglected.

[2] Though superficial secondary mucous papules and ulcers are not uncommon they are habitually overlooked and heal spontaneously.

The tissues most frequently involved are the bony septum (51 cases), the palate, i. e., the floor of the nose (28 cases), the cartilage (7 cases), the turbinates (3 cases). Septum or palate may be implicated alone. Cartilage or turbinate is never involved except with the bony septum.

Symptoms.—Exceptionally the gumma is seen as a little dark-red tumor before perforation; usually it is not seen until more or less extensive destruction of the bones has occurred. The symptoms that bring the patient to the physician are obstruction of one nostril (rarely both), slight pain (though this may be increased by secondary septic involvement of the accessory sinuses), and the casting off of a great quantity of foul, bloody scabs, amidst which the patient rarely recognizes fragments of bone.

Close questioning reveals the fact that *the first symptom noticed by the patient is an increase in the discharges from his nose and the presence of blood in these discharges.* He will also confess that the sense of smell is either blunted or completely lost.

Examination (anterior rhinoscopy) reveals a perforation in the septum, which is usually situated at least an inch from the anterior nares, though it may appear at the junction of bone and cartilage. This perforation is either surrounded by a sloughing sore or else plugged with scabs and dead bone. There are usually superficial ulcerations in the adjacent tissue, and there may be a sloughing, necrotic mass on the turbinate or a perforation in the floor of the nose.

Severe or neglected cases go on to total destruction of the nasal septum, and may be identified the moment they enter one's office by the peculiar snuffling, purring sound due to the flapping of necrotic tissue excited by the breathing. The result of such extensive destruction is at first a considerable edema and soreness of the nose externally. When in this condition the nose, if gently moved from side to side, emits

a crackling sound indicative of the multiple, irregular perforations in all its bony structures.

With the healing of such a lesion the bridge of the nose sinks in, and may even fall flat to the level of the patient's face if all of the bones have been destroyed. The deformity, if chiefly due to bone destruction, is saddle-backed (Fig. 51),

FIG. 51.—LOSS OF SEPTUM: ACQUIRED SYPHILIS.
(Cf. Figs. 63, 64, p. 511.) (Fordyce.)

while the more rare destruction of the cartilage produces a deformity *en lorgnette* (like an opera glass), which looks as though the lower third of the nose had been partially pushed up inside the upper two thirds.

Still more virulent cases resulting in extensive perforation of the accessory sinuses, cicatricial obstruction of the lacrymal ducts or of the Eustachian tubes, complete destruction of the soft parts of the nose, and invasion through the ethmoidal cells of the meninges, with consequent fatal septic meningitis, are so uncommon as to deserve only a passing mention.

Diagnosis.—The importance of an early diagnosis cannot be overestimated, and the only way to achieve this is to put the patient on his guard. Bid him, *if he blows bloody scabs from his nose*, report at once.

Treatment.—Mixed treatment, with high doses of iodid, is required in the virulent cases, though, in most instances, a mild course is quite sufficient. The nose must be kept clean by douching or spraying with oil or by the application of boric-acid ointment. Sequestra must be removed as rapidly as possible, but no other local treatment should be attempted.

Other Rare or Obscure Conditions.—The destructive lesions above described tell almost the whole story of tertiary nasal syphilis. But other rarer conditions also merit mention.[1]

Syphiloma.—This name has been given by the Germans to a very rare tumor, usually a complication of syphilitic septal perforation and growing from the septum (though it may grow from the inferior turbinate or in the nasopharynx). It is grayish or reddish in color, usually soft in consistency, and may attain a considerable size. It is usually confused with tuberculoma. It disappears promptly under mixed treatment.

Fibroid Degeneration of the Turbinates.—Described by Mackenzie.

Atrophic Rhinitis.—The healing of a septal gumma almost always leaves more or less atrophy of the mucous mem-

[1] Cf. Renner, *N. Y. Med. J.*, 1904, February 8, 15, 22.

brane, which is interpreted by Lang, Gerber, and other German authors as truly syphilitic (not a mere complication).

Moreover, there seems reason to believe that cases of atrophic rhinitis beginning at about the age of puberty *may* bear some relation to antecedent hereditary syphilis.[1]

TERTIARY SYPHILIS OF THE EAR

Apart from the *tertiary skin lesions* which may affect the outer ear, and *otitis media* which may result from syphilis of the adjacent bones, the only type of tertiary syphilis of the ear is the *sudden, absolute deafness* due to disease of the inner ear.

Syphilis of the Internal Ear.—The lesions of syphilis in the inner ear are not well known.[2] Indeed, the one symptom is *deafness*, without any local evidences of the disease.

Since this deafness is, in my experience, often associated with other lesions of the nervous system, I think it preferable to defer its consideration to another chapter (page 395).

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[1] Cf. discussion of Richards's paper, *J. Am. Med. Ass'n.*, 1907, vol. xlviii, p. 42.

[2] Hennet (Fournier's *Traité de la syphilis*, 1904, vol. ii, p. 620) relates one autopsy showing a secondary periostitis.

CHAPTER XXV

SYPHILIS OF THE NERVOUS SYSTEM[1]

GROUPING the lesions of syphilis according to the tissues involved, one may affirm that syphilis of the skin is the most frequent, syphilis of mucous membranes the most infectious, syphilis of the nervous system the most malignant manifestation of the disease. Syphilis of the nervous system is indeed malignant in that it occurs in a large proportion of syphilitics and that its lesions are always grave and often incurable.

Thus, among my 2,500 cases, 504 (twenty per cent) were afflicted with syphilis of the nervous system, and of all patients showing tertiary and parasyphilitic lesions, forty-two per cent were affected in the nervous system. Moreover, 39 died paretic; 113 were more or less disabled by tabes; 10 (at least) died insane, and 7 others of various paralyses; while, of the remainder, about one fifth were permanently and gravely disabled. To admit (in round numbers) 60 deaths and 175 cases of permanent, though often moderate, disability, is hard, but necessary. Such are the results of syphilis of the nervous system as we find it recorded.

ETIOLOGY

One must distinguish the effects of race, sex, age, treatment, and alcohol.

Race.—Not only are full-blooded members of the yellow, brown, and black races almost entirely immune to the para-

[1] Reviewed by Dr. E. G. Zabriskie.

syphilids, but they also possess a relative immunity to all syphilids of the nervous system. Despite utter neglect of medicine or hygiene they escape in far larger proportion than we do.

Sex.—Only 21 cases (ten per cent) are recorded among women, against 483 (twenty-one per cent) among men.

Age.—The effect of age is marked. Comparing lesions of the nervous system with all other tertiaries:

Of men infected under twenty years of age, 18 = 31 per cent N. S.[1]

Of men infected between the ages of twenty and twenty-nine, 199 = 43 per cent N. S.

Of men infected at or after thirty, 147 = 51 per cent N. S.

Moreover, the older the victim at the time of infection, the earlier the outbreak of his syphilis in the nervous system, thus:

Of men infected before twenty, only one had N. S. within two years.

Of men infected between twenty and twenty-nine, 12 had N. S. within one year, 41 within two years.

Of men infected after thirty, 31 had N. S. within one year, 51 within two years.[2]

Thus the older the man at the time of infection, the more likely is he to have N. S., and the sooner will it begin.

Treatment.—As far as the prolonged taking of mercury is concerned, I find that 120 were well treated, in that they took mercury at least eighteen months; while 124 were ill treated, in that they took it not at all or for a shorter time than eighteen months. But, dividing the patients into three classes by age, as in the preceding computations, I find that:

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[1] Syphilis of the nervous system.

[2] Or, to express it still more forcefully—in the youngest class, half of N. S. began in the eleven years following chancre; in the medium class, half began within five years; in the older class, within three years.

Of the youngest class, 4 were "well" treated, 9 "ill" treated.

Of the medium class, 45 were "well" treated, 72 "ill" treated.

Of the older class, 51 were "well" treated, 43 "ill" treated.

This is what one might expect: the older man takes better care of himself than the younger; nevertheless, the older man is more afflicted with N. S. than the younger.

This proves, if it proves anything, that mercurial treatment, as currently employed, has a relatively slight inhibitory effect upon N. S.[1]

Alcohol.—My records relate only to marked and persistent alcoholism. Of this class, 151 had tertiary lesions and only 61 N. S. Improbable as it may seem, three fifths of the alcoholics who showed tertiary syphilis escaped as to their nervous system. Thus, however much alcohol may predispose a patient to N. S., it alone cannot be invoked as a constant and essential cause.

Summary.—If it be not alcohol, what is it that makes men rather than women suffer from syphilis of the nervous system, white men rather than those of another hue, older men rather than younger men, and that in defiance of mercurial treatment?

The question is not an easy one to answer. Alcohol is in large measure answerable for tertiarism in general, and in some degree for syphilis of the nervous system as well; just as mercurial treatment is surely in some measure preventive. But the chief etiological factor is doubtless the "civilization" which Kraft-Ebing has linked with syphilis as the cause of paresis.

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[1] The statistics of most Continental writers suggest a marked beneficial influence from proper treatment. The insurmountable difficulty is to learn just what constitutes "good treatment," and whether it has been employed in a given case.

One naturally supposes neurotic or neuropathic persons more liable than others to nervous syphilis. Yet if this be so, I know some glaring exceptions to the rule.

But the men most susceptible to syphilis of the nervous system appear to be those who live hard or high; those who slave or sport; those who live the fierce life of cities rather than those who dwell in the better hygiene and the calmer surroundings of the country. The underlying idiosyncrasy which causes some such men to fall victims, while others escape, we do not know.

Date of Onset.—Although all authorities agree that syphilis of the nervous system is by no means an essentially late process, as was supposed by the earlier writers, yet there is considerable dispute as to the exact date of maximum frequency. This dispute is founded upon a looseness in interpreting the exact nature of the early symptoms which we might call secondary.

These symptoms, consisting almost exclusively of headache and neuralgias of various kinds, are very common at the onset of syphilis, and it is highly probable that many cases of syphilis of the nervous system have their origin with the short-lived headache of the early stages of the disease.

Yet, in speaking of syphilis of the nervous system, it is impossible to take this feature accurately into account; for the greater number of sufferers from early headache or neuralgia have no subsequent grave lesions of the nervous system.

Our cases, collated as accurately as possible, show a maximum frequency of onset in the third year of the disease, and also show that the number of lesions of N. S. occurring in each of the first three years far exceeds the number occurring in any subsequent year.

The effect of age upon the onset has already been dwelt upon. It is to be noted, however, that some of the most important lesions of the disease are most common in the first and

ONSET OF SYPHILIS OF THE NERVOUS SYSTEM

Year of Onset	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15–19	20–24	25–29	30–32	1	2
117—Ocular	6	7	10	7	10	8	6	7	3	6	4	4	4	4	8	4	3	0	16	33
113—Ataxia	0	0	6	2	5	6	5	8	6	4	5	3	1	3	8	9	3	1	38[3]	88[3]
89—Hemiplegia (and apoplexy)	10	13	10	9	2	3	4	2	3	5	3	1	0	2	5	2	2	0	13[4]	52[4]
68—Neuralgia (and paresthesia)	6	11	10	5	8	3	4	4	0	6	0	0	1	0	2	3	0	0	5	22
62—Headache	7	11	11	6	6	3	4	1	0	1	0	3	1	0	2	1	0	0	5	14
50—Vertigo	8	4	7	1	2	4	0	2	3	1	3	3	0	0	5	2	0	1	4	17
39—Paresis	0	0	3	2	3	3	2	3	1	3	2	1	1	2	0	5	2	0	6[5]	32[5]
29—Auricular	9	2	5	1	2	1	2	0	1	2	0	0	0	0	1	1	0	0	2	9
22—Epilepsy	2	2	4	3	1	2	1	0	1	1	0	0	0	1	2	1	0	0	1	5
20—Syphilis of cord	3	7	1	0	0	1	2	0	0	0	1	0	0	0	1	0	1	0	3	18
21—Aphasia	0	0	4	3	1	1	1	0	2	1	1	2	1	0	1	0	0	0	3	2
17—Dementia (and melancholia)	1	1	1	2	0	0	1	0	0	0	1	0	0	0	2	1	0	0	7	8
17—Facial paralysis	5	0	1	1	0	0	0	0	1	0	0	0	2	0	1	1	0	0	5	4
12—Mania	1	0	2	1	0	0	1	0	0	2	0	0	0	0	0	2	0	0	3	3
13—Monoplegia (and irregular paralyses)	1	1	2	1	0	1	0	2	0	0	0	0	0	0	0	2	1	0	1	4
5—Laryngeal paralysis	0	1	0	0	0	0	0	2	0	2	0	0	0	0	0	0	0	0	0	3
Total number of lesions .....698[6]	59	60	77	44	39	36	33	31	21	34	20	17	11	12	40	38	12	2	112	...

[1] Date of onset unknown.

[2] Number of cases in which the lesion mentioned was not associated with, preceded by or followed by other symptoms of N. S.

[3] Seven deny syphilis.

[4] Two deny syphilis.

[5] One denies syphilis.

[6] Occurring among 504 cases.

second years. Such are myelitis, (hemiplegia [1]), deafness, and facial paralysis; while ataxia and paresis begin to appear only in the third year and run an average maximum about evenly from the third to the eleventh year.

PATHOLOGY OF BRAIN LESIONS

The pathological changes produced by syphilis in the brain were, until this generation, ill understood. Before the researches of Heubner (1874) they were reducible practically to two classes:

1. Cases of diffuse or localized infiltration (sclerosis and gumma), most common in the meninges about the base of the brain, less frequent at other parts of the meninges, or arising from the bone, or apparently within the brain itself.

2. Cases *sine materia,* in which the pathological findings were nil.

Arterial Lesions.—Heubner showed the nature of the lesions in this second class of cases by discovering syphilitic arteritis of the main arteries about the base of the brain and their branches, *even to the arterioles within the brain.*

The arteritis begins with an exudation and proliferation of the cells of the intima (says Heubner), which exudation eventually becomes organized into new connective tissue. This thickening may involve considerable areas and causes partial or complete obstruction of the lumen. By destroying the elasticity of the vessel wall it leads to thrombosis, rupture, or aneurysm.

Köster suggested that the process originated not in the intima, but in the vasa vasorum; and Baumgarten, in 1878, affirmed that the usual process was primarily in the outer

[1] The onset of hemiplegia is early in the experience of the syphilologist, though the neurologist, meeting it as an isolated lesion, does not see many cases before the third year.

coats of the artery, the invasion of the intima being secondary. This periarteritis is certainly the more common lesion, and is probably the underlying one.

Berkley [1] describes the gross changes observed as follows:

"Primary syphilitic disease of the large vessels of the brain basis presents some peculiar characteristics which serve to distinguish it, even to the naked eye, from diffuse arteriosclerosis and other degenerations, though in fact many of these are indirectly caused by the luetic poison. At an early stage the blood-vessels lose their semitransparent character; they become opaque and acquire a peculiar, whitish cast, which later assumes a gray tint. On cross-section a vessel no longer collapses, but remains triangular, or at least irregular, in outline. To the touch the walls feel harder, and show a tendency to break on slight tension. On a closer examination of the lumen it is found to be narrowed to perhaps one half of its former caliber by what is apparently a zone of newly formed substance of gray or whitish appearance, which has a dry and cartilaginous consistency. Finally, the lumen of the vessel may be entirely obliterated by the advancing growth of the new formation, or thrombi may form in the narrowed channel.

"Other forms of syphilitic degeneration or neoplastic formation may now and then be found. Miliary gummata may be strewn along the periphery of the larger vessels, or the elements of the adventitia, nuclear or fibrous, may proliferate, inclosing the vessel in a thickened sheath, which gradually involves the inner coats, weakening the walls, and by extension of the proliferating elements narrowing the lumen."

Meningeal Lesions.—Apart from acute early meningitis, of which the lesions are not definitely known, syphilitic meningitis is a tertiary process; either a diffuse infiltration or multiple localized gummata. These latter vary in size from that

[1] "Mental Diseases," Appleton, 1900.

of a pin's head to a robin's egg, while the diffuse infiltration may cover a considerable area.

On the base of the brain the lesions usually begin in the pia mater and extend very widely. The greatest infiltration is usually about the optic chiasm, and may extend as far back as the meninges of the cord. The cranial nerves are affected either by compression or infiltration (they may be swollen to several times their natural size), while the brain itself becomes adherent.

On the convexity of the brain the meningitis is commonly quite localized; and originates either in the dura mater or in the skull, thence extending inward, causing adhesion, infiltration, and compression of the underlying brain.

Arterial and meningeal lesions often coincide.

Gumma of the brain always originates in the meninges or in a vessel wall. It is extremely rare.

Secondary Changes.—The frequent invasion of the cranial nerves is a direct extension from the common syphilitic meningitis. Hemiplegia is due to the arterial changes, while mania and dementia are associated primarily with circulatory impairment, ultimately with actual secondary degeneration or infiltration of the brain.

PATHOLOGY OF LESIONS OF THE SPINAL CORD

The pathology of the spinal lesions of syphilis is quite the same as that of the cerebral lesions. The spinal meningitis is both diffuse and circumscribed (gummatous); arterial disease is quite constant and leads to sclerosis (or gumma) of the cord, or permits hemorrhage into it. But gumma of the cord is usually of meningeal origin.

As in the brain, the dura is rarely attacked, and invasion from a syphilitic bone lesion is most uncommon, since efficient protection is afforded by the intraspinal fat.

The distribution of lesions is usually both irregular and general; moreover, with the spinal lesions, cerebral lesions often coexist.

LYMPHOCYTOSIS OF CEREBRO-SPINAL FLUID

"The normal cerebro-spinal fluid contains no cells, or at least only rarely a mononuclear lymphocyte or an endothelial cell" (Dana and Hastings [1]). Therefore, when Nageotte and other French observers noted a marked *lymphocytosis of the cerebro-spinal fluid* in cases of cerebral syphilis as well as *in the great majority of recently infected syphilitics*, even in those who showed no evidence of cerebral or spinal lesions, they concluded that this lymphocytosis is due to syphilis (though the reaction is by no means universal). They also ventured the opinion that it is an early sign of meningeal involvement. But more recent investigations have failed to confirm this theory. Merzbacher [2] has summed up the evidence at hand, from which it appears that:

1. The lymphocytosis of syphilis is due to the disease itself (toxemia), not to a meningitis; for

2. This lymphocytosis is very frequent in certain conditions that have nothing to do with meningitis. Some of these are syphilitic, e. g., tabes, paresis, Argyll-Robertson pupil; some non-syphilitic, e. g., herpes, multiple sclerosis, and mumps.

3. Precisely what causes this lymphocytosis we do not know. *But it certainly is not a warning of an impending meningeal outbreak.*

From another point of view, however, this knowledge is extremely useful. For such conditions as hysteria, neuras-

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[1] *Med. Record*, 1904, January 23.—This contribution contains an excellent description of the technic of lumbar puncture and cytodiagnosis.

[2] *Centralbl. für Nervenheilk.*, 1906, vol. xxix, pp. 304, 352.

thenia, and alcoholism are *not* accompanied by lymphocytosis; hence *the existence of cerebro-spinal lymphocytosis is presumptive evidence in favor of paresis, tabes, or some syphilitic lesion, as against hysteria, neurasthenia, and alcoholism.*

More recent studies of the chemico-physiological qualities of the cerebro-spinal fluid, though not yet thoroughly worked out, suggest that the lymphocytosis is evidence of a specific reaction excited by the presence of spirochetæ. Thus a recent contribution [1] concerning the complement-deviation test reports positive results (presence of syphilitic antibodies) from experiments with the cerebro-spinal fluid in secondary syphilis and in paresis.

The application of these tests opens up a new field for investigating the relationship of syphilis to paresis and tabes. Thus far the evidence is uniformly favorable to linking the two great parasyphilids more closely than ever with syphilis. We may at least affirm that the *cytological and bio-chemical changes usually found in the cerebro-spinal fluid of known syphilitics are identical with those usually found in tabetics and paretics, while differing from those common to other diseases of the nervous system.*

EARLY SECONDARY MENINGITIS

The localized headache and neuralgias that so often accompany the first outbreak of secondary symptoms are perhaps due to acute meningitis or neuritis.

The clinical type of these pains has already been described (page 264). Lang has noted that they may be accompanied by mydriasis and bradycardia. Schnabel (quoted by Lang) found retinal irritation in 14 and actual retinitis in 7 among 40 patients examined at this period of the disease. The re-

[1] Virchow's *Archiv*, 1907, vol. clxxxviii, No. 1.

markable feature of these investigations is that the process found, though one would expect it gravely to impair the sight, had no such effect.

Prognosis.—The importance of these evidences of meningeal irritation is the warning they afford. Every syphilitic headache or neuralgia merits respect and attention whether it occurs at the onset or late in the disease. Whether it disappears after a few doses of iodid or proves intractable, it is always an evidence of a definite lesion which may, if neglected, lead to grave central lesions.

Treatment.—Iodids cure. Mercury prevents relapse. Moderate doses of each suffice, but the treatment should be continued for weeks after the symptoms have subsided.

SYMPTOMS

Apart from this early secondary meningitis, the lesions of syphilis of the nervous system have two striking clinical characteristics, viz.:

1. The occurrence of prodromes.
2. The variety and diversity of symptoms.

Prodromes.—There are five types of preliminary symptoms, one or all of which invariably, or almost invariably, precede the graver forms of cerebral or spinal syphilis. These are:

1. Pain.
2. Intellectual and moral derangements.
3. Impairment of the general health.
4. Impairment of the sexual power, and
5. Fugitive nervous symptoms.

Pain.—Syphilis of the spine may be preceded by sharp pains, or girdle sensations, or sensations of numbness or paresthesia in whatever nerves are becoming affected. But the

most constant and premonitory pain is the *headache* which precedes brain syphilis. This headache, like all other syphilitic pains, has three marked characteristics:

1. Nocturnal exacerbations with partial or complete relief of the pain by day.

2. Resistance to all the ordinary remedies for neuralgia, and persistence of the pain under their use, and

3. Prompt amelioration or relief under moderate doses of potassium iodid. The headache is often occipital and intense and sharp in character, but variations in situation and type are extremely common.

Sleeplessness results from this headache.

Intellectual and Moral Disturbances.—These are common preliminaries of cerebral syphilis, but do not precede a *purely* spinal lesion. They consist in a diminution of all forms of cerebral activity. The patient becomes gloomy, even melancholy. He is irascible. His power of concentration is diminished; his memory fails him; attention and ratiocination are both impaired.

Unlike the premonitory pains, which are usually persistent, these premonitory intellectual and moral disturbances are usually somewhat irregular. They may apparently improve from day to day for quite a time, only to relapse again, or they may amount to actual dementia or mania. They are quite frequently lacking.

Impairment of the General Health.—This, though the least definite, is perhaps the most constant of the premonitory symptoms of syphilis of the nervous system. It consists in a progressive loss of weight, often associated with a sensation of muscular weakness and anorexia.

Impairment of Sexual Power.—There is also commonly a slight but progressive diminution of sexual power and desire in the male; though this is often not called to the attention of the physician until, as improvement sets in, the patient

recognizes a marked increase in, or rather a return of, sexual vigor. This decrease of sexual vigor is often an important early sign of syphilis of the nervous system, and should always be sought for.

Fugitive Nervous Symptoms.—Certain irregular nervous symptoms precede almost every outbreak of syphilis in the nervous system; but this type of prodromes is essentially common and characteristic before hemiplegia and insanity. Apart from certain vague, abnormal sensations, which are common enough but difficult to characterize, syphilitic hemiplegia is invariably preceded by one or more attacks of vertigo, aphasia, or momentary paralysis.

The patient, while talking to a friend, suddenly finds himself *unable to speak intelligently* (*aphasia*). He is conscious of what he wishes to say and of his inability to say it. His mouth emits only incoherent words or syllables, and, ashamed at this inexplicable disturbance, he endeavors to hide it by silence or by a pretended nausea or giddiness. After a few moments the attack passes, and he readily persuades himself that it was nothing. If he is fortunate enough to have several such attacks, he may come to the physician before the onset of any graver lesion; but, as a rule, and perhaps on account of a concomitant intellectual deficiency, he pays no attention to these warnings.

In other cases the attack is a momentary *vertigo*. While walking along the street he suddenly loses his balance, clutches a railing or a friend's arm for a few moments, and then it is all over, and he is as well as ever.

Or he is *temporarily paralyzed*. While sitting at the table, he suddenly notices that he cannot hold his knife or fork; or, while walking, his leg gives way beneath him; but, as a rule, a few moments suffice to set the weakness aright.

Convulsive prodromes are less common.

Duration of the Preliminary Period.—These warning symptoms may be few and brief, or multiple and prolonged. Severe syphilitic headache or repeated attacks of vertigo may continue for months, uncontrolled by treatment, and without leading to any further evidence of cerebral syphilis. But in a majority of instances the warnings are so few and so slight that the patient does not consult a physician until the full attack has burst upon him (Cases XXXII, XXXIII, XXXIV).

Onset.—In spite of prodromata the onset of symptoms is always sudden. The patient's story is that "suddenly—" something happened. Be the attack a dementia, a paralysis, or a convulsion it develops suddenly, and to the untrained observer unexpectedly.

This sudden onset may or may not launch the patient into the midst of his attack. As a rule, the more extensive lesions require at least a few hours for their full development, and may even progress for several days. Thus syphilitic hemiplegia usually begins with a sudden, complete or partial, loss of power in a limb or a group of muscles, which loss of power soon spreads to the whole side. But as this spreading often occupies but a few hours, an unintelligent patient usually reports the whole affair as having occurred suddenly (Case VII, page 20).

Course.—The course of the manifestation is, as remarked above, characterized by a marked—even pathognomonic—variety of symptoms dependent upon the diversity and irregular distribution of the lesions from which they spring.

The lesions of cerebral syphilis are many and complex. More or less widespread gummatous meningitis and more or less widespread arterial changes may occur together. Hence results an almost infinite diversity of clinical symptoms.

The table on page 372 shows, it is true, that certain clinical types (e. g., hemiplegia and the parasyphilids) often stand

alone, while others (e. g., ocular paralysis and insanity) are usually only one element in a many-colored and confused picture of nervous disorder. But at the beginning some one lesion stands out most prominently, and, if this is vigorously attacked, there may be no further symptoms. Thus a patient in the prodromal stage may usually be cured by sharp treatment.

Moreover, a single lesion, such as a ptosis or an epilepsy, may remain unique, even though untreated for years, but this is very rare. The rule is an intermittent progress from one lesion to another, unless the process is checked by treatment.

Relapses are therefore very common and are the measure of treatment. The better the treatment, the fewer and further apart are the relapses.

Although it is not possible absolutely to separate all cases according to the underlying pathological lesion, one may attempt a general disposition of the cases under the following heads:

Cerebral Arteritis.—This leads usually to hemiplegia, to aphasia, to nuclear paralyses of the various nerves, and to mental obfuscation, drowsiness, and loss of memory, or epileptiform attacks, finally to insanity.

Basilar Meningitis.—There is usually involvement of one or more of the cranial nerves. There may be headache, vertigo, coma.

Meningitis of the Convexity of the Brain.—Headache, Jacksonian epilepsy, and localized cortical paralyses indicate the site of the lesion. Melancholia and mania are often associated with diffuse lesions of the cortical meninges.

Prognosis.—The statistical prognosis, as I have found it, has already been given (page 368). But these bare figures require interpretation. If taken in time, syphilis of the nervous system, however grave, however fulminating, may often be controlled and actually cured, so that no trace of it remains.

The wildest mania, the deepest coma, the most complete paralysis may yield like magic to prompt and efficient treatment. Medicine can boast no miracle greater than the effect of mercury and iodids upon syphilis of the nervous system. The blind see, the lame walk, the deaf hear, and those who are given up for dead arise again (cf. Case XXXIV).

But such wonders cannot always be accomplished even by prompt and efficient treatment. If the lesion is a diffuse syphilitic arteritis the impairment to circulation (and to function) is in large part permanent when the patient first consults his physician. And in any case, if treatment is delayed until the nerves have degenerated and muscles atrophied, it is vain to ask of any treatment the real miracle that would be required to restore the patient to perfect health. Much may still be done to prevent further destruction, and, even after months of neglect, the sight may be in some measure restored to an eye, the strength to a limb; but perfect restoration may not be looked for.

On the other hand, an occasional case goes from bad to worse in spite of our best efforts. We are not infallible (Cases VII, XXXII, XXXIV).

Treatment.—*At the onset* of the attack the treatment should be *intensive* and *specific*. Mercury is to be given by intramuscular injection and at a maximum dose. Iodid is begun at ten drops (minims) three or four times a day, and run up to the point of conquering the lesion or gravely poisoning the patient. By such a course the patient should be slightly salivated, more or less iodized, and much improved within three weeks. Specific treatment is then stopped.

The severity of this first course may be tempered somewhat according to circumstances. Undue sensitiveness may bid us substitute fumigations or inunctions for injections; a delicate digestion may forbid too vigorous use of iodids. *Prudence in the use of mercury is surely profitable*, for severe

salivation always does harm; *but prudence in the use of iodids in severe cases may lead to failure.* If the emergency is great the patient may be severely (but briefly) iodized with benefit.

The routine of rest, light diet, and urinary analysis need not be repeated here (page 198).

After these first weeks all specific medication should be stopped, and a course of hygiene instituted. If the patient lives in the city he should sojourn in the country, live on the most nourishing diet possible, spend his days in the open air, and take an iron tonic to which, in some cases, the addition of strychnin sulph., gr. $\frac{1}{50}$, t.i.d., is useful.

This hygienic course should last about three weeks, and then be interrupted by a second course of specifics. By this time the patient has usually greatly improved, and, accordingly, the specific course may be a mild one. If he has been salivated, one must omit the mercury altogether. The iodid need rarely be increased above 15 to 25 minims (1 to 1.5 gm.).

Thus the treatment is continued in interrupted short courses, alternating hygiene with specifics until, at the end of the second or third course of specifics, the patient has obtained the maximum benefit.

The reason for this interrupted method of treatment has already been given (page 141).

If the patient is not entirely cured, it is highly probable that the remains of his trouble are irremediable, and it is, therefore, vain to inundate him with specifics. But, if the symptoms continue to show real activity (or if the patient is unable to take adequate treatment in the first place), he may be sent to Arkansas or to Aix.

But this is not all. After the attack has been relieved every effort must be made *to prevent relapse.* The patient must be put on a routine preventive course (page 158) and carefully watched for evidence of relapse of any kind. The cases cited in the following chapter illustrate the difficulty here encoun-

tered. The patient's mind is often—very often—just enough disordered to induce a false sense of security. He may consider his attacks of some importance, but once these are controlled he has no further care, no fear for the future, and not the least wish to submit to repeated and prolonged courses of treatment. One must always be prepared for this mental condition, and ready to assume a high-handed mastery of the situation.

If the patient is first seen after his symptoms have existed for some time his history should be carefully investigated in reference to treatment. In many instances it will be found that he has been sadly mishandled and perhaps irretrievably harmed by prolonged and excessive specific medication. In such a case mercury and the iodids must be absolutely prohibited for a time, at least, however much the patient may long to continue wallowing in them.

But if he has been inefficiently treated, or if he is suffering from some acute relapse, the specific treatment may be resumed in short courses, but should usually be employed with much less vigor than in a fresh case.

In these chronic and long-standing cases, hygiene, hydrotherapy, mechanotherapy, and electricity—in fact, all the resources of neurology—are far more to the point than mercury and iodid.

Operative Treatment.—Quite a number of cases of brain gumma have been subjected to operation. Stransky,[1] in a recent review, concludes that it is quite justifiable if prompt relief does not result from specific treatment. But it is open to question whether the operative results as yet justify such an opinion.

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[1] *Centralbl. f. d. Grenzgebiete*, 1905, vol. viii, p. 1.

CHAPTER XXVI

SYPHILIS OF THE NERVOUS SYSTEM[1]

CLINICAL TYPES: DIAGNOSIS

DESPITE the utter irregularity of the symptoms of syphilis of the nervous system, there are certain recognizable types to which a case, for a time, at least, usually adheres.

We may distinguish:

Syphilitic paralysis.
- Ocular paralyses.
- Hemiplegia and apoplexy.
- Aphasia.
- Deafness.
- Facial paralysis.
- Other forms of paralysis.

Syphilitic epilepsy.

Syphilitic insanity.

Syphilis of the spinal cord.

Parasyphilids.
- Tabes.
- Paresis.
- Erb's spastic spinal syphilis.

Syphilitic neuritis.

OCULAR PARALYSES

One hundred and seventeen cases, of which 63 involved the motor oculi (8 bilateral), 34 the optic (neuritis) (6 bilateral),

[1] Reviewed by Dr. E. G. Zabriskie.

14 the sixth (1 bilateral). In only a few cases are several ocular nerves recorded as simultaneously or successively involved. The right eye was affected 28 times, the left 21 times, both 15 times. Ten cases were bilateral. Both eyes were paralyzed within a short period at one, three, four, five, six, ten, eighteen, and twenty-one years. Twice the eyes were involved successively at four and nine, eighteen, and twenty years.

The ocular lesions were associated with other lesions of the nervous system in 85 cases; with tabes [1] 23 times, with hemi- and monoplegia 22 times,[2] with aphasia and auditory paralysis each 9 times, less often with every lesion of the list.

Paralysis of the Motor Oculi.—Paralysis of the third cranial nerve is almost the hall-mark of cerebral syphilis, so common and so characteristic is it, whether occurring alone or in connection with other and graver lesions.

The subjective symptoms are diplopia and ptosis. The former I find about thrice as common as the latter. There is no pain unless from eye strain. Because of the difficulty in fixing the location of objects the gait may be uncertain and there may be vertigo (14 cases).

Examination reveals some or all of the following changes, depending upon the more or less complete paralysis of the nerve.

1. *Ptosis.*—This is usually complete and hides the ocular deviation. On lifting the lid we find:

2. *Deviation of the Eye.*—The eye is drawn outward by the action of the external rectus and the superior oblique muscle.

If the paralysis is partial, affecting only certain of the muscles innervated by the motor oculi, the eye is fixed in various positions according to the muscles paralyzed.

[1] Exclusive of the changes in pupillary reaction.

[2] And probably oftener.

3. *Fixation of the Eye.*—The eye is almost immobile, and can be moved only slightly in the direction of the unparalyzed muscles.

4. *Diplopia.*—The usual type of diplopia is crossed with the false image slightly elevated and inclined away from the paralyzed eye. Incomplete or complex paralyses vary the relative positions of the image as they do that of the eye itself.

5. *Mydriasis* (*Internal Ophthalmoplegia*).—This is due to paralysis of the sphincter iridis and the ciliary muscle. As a result the patient finds it impossible to read, though the sight at distance is relatively little affected.

Paralysis of the Abducens.—The only subjective symptom is diplopia, which examination reveals to be homonymous and with both images erect and at the same elevation. The eye is fixed in internal strabismus.

Paralysis of the Fourth Nerve.—Extremely rare: homonymous diplopia with the false image below the true and inclined obliquely. The eye is raised and turned inward.

Optic Neuritis.—Next in frequency to paralysis of the motor oculi. This lesion is so intimately related with visible disturbances within the eye that it is better to consider it in relation to these (page 419).

Site of the Lesion.—Extension to the nerves of syphilitic lesions in the *bones of the orbit* is very rare, and is accompanied by local tenderness, pain, or even swelling. All the nerves are usually involved. Invasion from gumma of the *sphenoidal fissure* may, according to Rochion-Duvigneaud, be diagnosed from the simultaneous involvement of all the nerves that pass through this fissure: viz., the third, the fourth, the first branch of the fifth, and the sixth, the remaining branches of the fifth being uninvolved. The optic nerve may be implicated.

Excepting the optic, these nerves are very little subject to syphilitic *neuritis.*

Gummatous *meningitis* is by far the commonest cause of ocular paralysis. The nerves are compressed or inflamed. There are usually associated lesions of mental, paralytic, or epileptic sort.

Finally, the nerves may be assailed at their origin *within the brain* by disease of the posterior cerebral arteries.

It has been supposed that *partial paralysis* of the motor oculi must be due to cerebral lesions. But autopsies have abundantly proven that it is as often, perhaps oftener, due to basilar meningitis.

Prognosis.—Prompt mixed treatment, of moderate severity (administering the mercury by injection, inunction, or fumigation) cures almost all syphilitic paralyses of the external muscles of the eye in six to eight weeks. The internal muscles yield rather more slowly, if at all.

Relapses among my cases after at least one year of health are recorded eight times (intervals of one, two (twice), three, four, five, twelve, and thirteen years).

HEMIPLEGIA AND APOPLEXY

Eighty-nine cases, of which five are apoplexies without paralysis, and of the remainder about two thirds paralysis without and one third paralysis with apoplexy (though in few was the loss of consciousness complete).

Onset.—The attack is preceded by prodromes (page 378). *The onset usually occupies several hours*, perhaps several days; *beginning without loss of consciousness* by a slight weakness of one limb, which gradually steals over the whole side. Thus a woman remarks one afternoon that her husband drags his left leg in walking, though he himself is unconscious of this; the next morning he awakes with left hemiplegia (see also Case XXXII).

On the other hand, the onset may be sudden and complete, with or without apoplexy.

Finally, syphilitic apoplexy may be the severe apoplexy characteristic of cerebral hemorrhage, lasting for hours or days, or a mere fleeting unconsciousness.

Clinical Type.—Syphilitic hemiplegia is characterized by incompleteness, varying intensity, and association with other lesions of the nervous system, and by yielding readily to treatment.

Incompleteness.—Careful examination of the paralyzed limbs reveals that the muscles are very unequally involved. Certain muscles or groups of muscles are weakened rather than absolutely impotent. Thus the face is often but little affected, and the lower extremity may retain some power of movement while the upper is completely inert.

Varying Intensity.—But more striking still is the fact that, if the exact condition is noted carefully at the onset, a second examination a few hours later reveals many minor changes. While one limb has perhaps gained strength another may have become paralyzed, or perhaps the paralysis has everywhere progressed or everywhere diminished. Or the two may alternate in a " recurrent " paralysis. I have seen a patient remain completely hemiplegic for three hours, and then rapidly and spontaneously regain his strength. (Such a condition is undoubtedly due to vascular spasm.)

Such variations, however, are exceptional. Usually one must watch sharply to note the variations after the first few hours, unless these be due to other lesions of the nervous system.

Other Lesions.—Hemiplegia, like any other of the clinical types of syphilis of the nervous system, may stand alone or may be but one of many lesions. Even if unique, as is so often the case, it is not absolutely so. We have studied the warnings that precede it. Similar phenomena usually accom-

pany it: viz., psychic or intellectual disturbances, vertiginous or epileptiform seizures, outlying or discordant paralyses.[1] Among these latter, aphasia, dimness of vision, and paralysis of the motor oculi are common.

As examples of crossed paralyses I may mention a case of right hemiplegia and left ptosis and one of right facial paralysis, followed a year later by left hemiplegia. The ensuing two cases of double hemiplegia merit more extended mention as examples of what untreated syphilis of the nervous system can do.

Case XXXII.—H. W., aged thirty-two, reports that, twelve years before, he had four genital sores, followed by suppurating bubo, indefinite secondary symptoms, and four or five months' internal mercurial treatment. Since then no symptoms until five months ago, when he began to have severe headaches, much worse at night. He has lost appetite and sleep, and has sciatica, worse at night. He gives no alcoholic history.

Examination reveals diffuse thickening of the inner half of the right clavicle, and slight thickening with crackling on pressure of the inner half of the left. He had not discovered these lesions. The right side of the cranium, the clavicle, the shins, and a rib on the left side are tender.

He was put upon mixed treatment, under which his pains promptly disappeared, and he continued the treatment but five months.

Five years later, while dining at a restaurant, in his usual health, he discovered that he had lost the power of speech. Much alarmed, he endeavored to rise, but found the *left side* helpless. His companions thought him drunk, and took him home to bed. After a few hours he regained all his faculties, and though he has had several similar relapses since, none have been as sharp as the first.

Examination reveals dilatation of the right pupil, tenderness of both shins and of the ribs. No paralysis apparent.

He was put upon mixed treatment again, and was not seen until *two months later*, when, without warning and without loss

[1] Hemianesthesia is unusual.

of consciousness, he had a spasmodic attack in the right side, followed by diplopia and partial paralysis of the *right leg*, the left remaining unaffected.

Examination reveals great weakness of the right leg, but no complete paralysis. Arms and face, rectum and bladder normal. Slight converging strabismus of the right eye; pupil normal; mydriasis of the left eye.

He again took treatment irregularly and inefficiently, and, *a month later*, these lesions having been only partially relieved, his mind was markedly impaired, his memory defective, and he had another attack of paralysis in the *right leg*.

He was then taken in hand and given fifteen grains of iodid three times a day. In four weeks he was apparently well. He continued taking the iodid for *six months*, and *six months* thereafter had another *left hemiplegia* without headache or loss of consciousness; but this time his mental and physical condition was extremely unpromising, and it is probable that he died soon after.

Case XXXIII.—W. H., aged thirty-seven. Was first seen lying senseless and almost motionless at his home. From his friends, and subsequently from himself, the following history was obtained:

Chancre and a few early symptoms seven years before; treatment by mercury six months. Three years later, *left hemiplegia* and aphasia, followed by various ulcerations and periostitis of the external condyle of the left femur. He was treated irregularly and inefficiently for four years, during which time he had two more attacks of aphasia; then the present attack came on.

He arrived home at eleven o'clock completely aphasic, and then gradually went into a state of almost complete unconsciousness. His pupils both reacted to light, but were unequal. The only evidence of intelligence he gave was to shake or nod his head when questioned. There were several active ulcerations over the body and many circular pigmented scars. The urine was of a specific gravity of 1.040, loaded with urates, and contained albumin and casts. The right arm and leg were partially paralyzed.

He was given ten grains of potassium iodid every three hours. During the day he came out of his lethargy and went into an

active delirium. That night he did not sleep, and the next day he became maniacal.

Mercurial inunctions were begun. The mania began to lessen on the second day, and on the third he slept ten hours, and was fairly quiet and rational most of the time. On the eleventh day he was dressed and walking about, his hemiplegia much improved, his mind almost normal. He was taking about one hundred grains of iodid a day. This was rapidly diminished, and he continued to improve. A month from the beginning of the attack he was entirely well, except for slight slowness of thought and articulation; slight weakness on the left side and a severe bronchitis.

After this he was seen no further for a month, when he suddenly relapsed into his condition of stupor and paralysis. This improved for a day or two, and again gave place to mania, and, on the fourth day of the attack, his *right arm and leg* became paralyzed, the onset of the attack being preceded by a slight convulsion. His right pupil was dilated, his intellect very confused. The right side was completely paralyzed, as was the bladder.

He was promptly put upon 100 grains of iodid a day; but, in spite of this, five days later he had a sharp epileptic fit, followed by severe pain in the head; but the following day the paralysis in the right hand had disappeared.

Inunctions and iodid, at the dose of a dram a day, were continued for several weeks, the patient constantly improving.

At the end of a month from the last attack he had three convulsive spasms in his right hand, and became aphasic again, and very depressed, but not stupid.

No paralysis accompanied this attack, and he again improved steadily under treatment, until one month later—four months after he was first seen—he had another general epileptic attack, followed by acute mania and rapidly progressing debility.

In this attack he died in two weeks.

Such cases require no comment. They show the full picture of cerebral syphilis; the sudden onset of the most diverse symptoms; the marvelous effect of treatment in relieving these;

the unreasoning carelessness and confidence of the patient; the inevitable relapse of the case that has been for years irregularly treated; the final fatal issue.

Prognosis.—To find such cases in a record of private practice I had to look back thirty years. Syphilis of the nervous system we still constantly see, but these ill-treated, poorly nourished, alcoholic victims appear only in our city hospitals. In private we encounter every lesion, perhaps every combination of symptoms, but very rarely, as in the above instances, all symptoms in one case.

The well-treated case of syphilitic hemiplegia usually recovers rapidly, leaving little weakness. Be the underlying lesion a meningitis, or a thrombosis, or simply a syphilitic arteritis (the common lesion), it yields kindly to mixed treatment.

Under two circumstances, however, a cure may not be obtained:

1. If the artery ruptures, the paralysis, due to cerebral hemorrhage, is no better in prognosis than any non-syphilitic apoplexy.

2. If the case is neglected until the paralyzed muscles atrophy, or the arterial or meningeal exudate becomes organized, permanent paralysis becomes inevitable.

It is surprising, to be sure, how slow these changes are to occur, and how many years the patient may remain in some degree amenable to mixed treatment; but it is only one degree less marvelous to find a patient to whom the highest possible doses of iodid and mercury have been administered in vain, keeping himself poisoned with these drugs year in and year out in the hope that they will ultimately benefit him. Yet such victims of misguided enthusiasm are pitifully numerous.

APHASIA

Aphasia, while one of the commonest symptoms of cerebral syphilis (more frequent than is shown in my statistics), rarely stands alone; only twice in my series was it not attended, preceded, or followed by other symptoms of cerebral syphilis.

Syphilitic aphasia is often incomplete and intermittent. It may cease spontaneously. Agraphia, word-blindness and word-deafness are likely to be associated with it.

Nine of my cases of aphasia accompanied hemiplegia [1] (e. g., Case XXXII); epilepsy and ocular paralyses are also frequent concomitant lesions.

DEAFNESS [2]

Almost half the cases of syphilitic deafness occur in the first two years of the disease.[3] While the deafness may be incomplete or may assert itself insidiously, the typical deafness of syphilis is sudden and absolute. One patient related that, at a given moment, without any warning, he was stricken suddenly and absolutely deaf in one ear. Others stated that the deafness became absolute within an hour or two.

Pathology.—The lesion may be in the internal ear or somewhere along the course of the nerve. The aurist places it usually in the inner ear, and autopsy has several times revealed exudative and sclerotic changes therein, together with syphilitic exostoses. None of these changes are peculiar to syphilis.

On the other hand, the association with other lesions of cerebral syphilis in our cases has been striking. Only thirty-one per cent were free from such concomitant lesions. These

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[1] Usually but not always on the right side.

[2] Auditory hyperesthesia has been noted; it is extremely rare.

[3] Politzer has seen a case in the first week.

were many [1] or few. The eye was involved 9 times (thrice with the ear, 6 times before or after it); other paralyses occurred 5 times, neuralgia 5 times, tabes thrice.

Moreover, severe vertigo was only four times noted at the onset of deafness [2] (though slight vertigo is always present).

Hence it seems proper to associate deafness with lesions of the nervous system rather than with those of the ear.

Symptoms.—The onset is usually *without prodromes* (contrary to the general rule) and *painless*; *within a few hours the patient is stone-deaf* in one ear (rarely in both). Slight vertigo is usually present, but this is not a prominent symptom. The characteristics are suddenness of onset, rapid progress of the deafness, and loss of bone conduction.[3] But these three symptoms, though very suggestive of syphilis, if they occur in a person under forty-five years of age, are not absolutely pathognomonic.

Prognosis.—Deafness occurring in the first two years of the disease may often be cured if attacked promptly by injections or inunctions of mercury (I prefer the former) and iodid.[4]

If treatment is delayed or the onset of deafness is late in the disease the prognosis is not good.

Treatment.—The only special treatment for deafness is that by pilocarpin. If a short, sharp course of mixed treatment fails, a course of hypodermic injections of pilocarpin hydrochlorate should be instituted. Using a two per cent solution, daily injections should be given, beginning with

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[1] E. g., hemiplegia at one year; paralysis at two years; demented at four; deaf at five; deaf at two years, ocular paralysis at four, neuralgia at five, paralyzed vocal cord at ten.

[2] Though in three other cases it is noted at intervals of two, six (preceding), and eight years from the deafness.

[3] Tested by holding a tuning fork against the teeth.

[4] Of 15 cases followed, 7 were cured, 4 somewhat improved, and 4 unimproved; of the 7 cures, 5 were obtained in the first two years of the disease.

ɱiv, and increasing one minim at each dose for a week; continue the daily dose at or about ɱxij for another week. Politzer advises employing this treatment before the antisyphilitics.

FACIAL PARALYSIS

The only peculiarity to distinguish facial paralyses from similar lesions due to other causes is the concurrence of other syphilitic lesions and the amenability to antisyphilitic treatment.

When it occurs in the first year of the disease it is rarely (one in five of my cases) accompanied by any other lesion in the nervous system, though such lesions often (three cases) follow in later years.

Facial paralysis is usually incomplete, whether syphilitic or not.

The prognosis is relatively good; some improvement is almost always obtained.

OTHER FORMS OF PARALYSIS

Since any nerve or any combination of nerves may be paralyzed by syphilis, it were futile to attempt the enumeration of all the forms or combinations of syphilitic paralysis.

Besides the thirteen monoplegias and irregular paralyses of the extremities and the laryngeal paralyses mentioned in the table on page 372, one case of olfactory and three of glossopharyngeal paralyses have been noted.

Acute bulbar paralysis, due to rupture or thrombosis of the basilar artery, causes apoplexy followed by Cheyne-Stokes's respiration or epileptiform attacks. If death is not immediate there follow labio-glosso-laryngeal paralysis, more or less complete paralysis of all the extremities, and various respiratory and cardiac irregularities. Albuminuria, glycosuria, and

hyperpyrexia may also coexist and the patient soon dies. The condition is an extremely rare one.

SYPHILITIC EPILEPSY

The epilepsy of syphilis, if one includes every convulsive phenomenon under this head, is extremely common. A transitory and Jacksonian epileptiform attack accompanies the majority of paralytic or vertiginous seizures, but marked or persistent attacks of *grand mal* are relatively uncommon. It is often associated with mania (5 cases [1]), dementia (3 cases [1]), or ocular paralysis (6 cases). Indeed, neurologists consider syphilitic epilepsy to be commonly a symptom or prodrome of paresis.

Grand Mal.—Syphilitic *grand mal* differs from the idiopathic type in four particulars:

1. It begins in adult life instead of in childhood.
2. The aura and initial cry are often absent.
3. There is usually history or other evidence of syphilis.
4. The epilepsy yields to antisyphilitic treatment.

Otherwise there is nothing to distinguish them.

Petit Mal.—Uncommon. Age, history, and treatment alone distinguish it.

Jacksonian Epilepsy.—Localized convulsive seizures are almost exclusively due to syphilis or trauma, and are almost always associated with paralysis of the affected nerves.

SYPHILITIC INSANITY

Insanity, like epilepsy, is far more frequent than my records show; for a brief dementia or mania often accompanies the onset of some other lesion; e. g., hemiplegia, but is over-

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[1] One duplicate.

shadowed by it, while mental inefficiency, impairment of memory, and loss of emotional control precede and follow almost every grave paralytic attack.

Berkley [1] states that syphilitic insanity depends rather upon the tissue starvation and congestion due to vascular disease than upon the meningeal processes.

The *prodromes* are nocturnal headache, "fleeting pareses and paralyses, epileptiform convulsions, sudden faints, abrupt attacks of furibund mania, followed by half comatose conditions. . . . The pupillary symptoms, though not so frequent as the pareses, are of the same order; also irregularities in the size of the pupils and mydriasis are more often met with than spastic myosis."

The following is Berkley's classification:

1. Sudden furibund delirium of short duration, followed by half comatose states.

2. Delusional insanity.

3. Slowly progressive dementia, attended by incomplete paralysis of the extremities and external ocular muscles.

4. Dementia, attended not by paresis, but by epileptiform attacks.

5. Syphilitic epilepsy.

Mania.—A neglected and fatal case in which mania was a prominent symptom has already been cited (Case XXXIII). In this instance the brilliancy of the temporary reaction to treatment was overmatched by persistent neglect, and the issue was fatal. But only three other maniacal cases died under our care, while at least four were promptly and perfectly cured. The others (five) remained feeble-minded or demented.

Delusional Insanity.—"Found especially in persons who have inherited a strong predisposition to insanity, and may closely simulate paranoia. The delusions are varied. . . .

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[1] "Mental Diseases," D. Appleton, 1900.

Aural hallucinations are rare. . . . Treatment is ineffectual, the lesions being organic and permanent."

Thus Berkley: the syphilologist sees such cases chiefly under the guise of melancholia. It is important to remember that sexual, alcoholic, and other excesses may lead to this condition quite as definitely as does syphilis. Indeed, the few cases of delusional insanity which I have seen follow upon syphilis have seemed to be due rather to alcoholism than to the disease.

On the other hand, one should not take the incurability of such cases too seriously. It is impossible always to determine that the lesion is organic; the test course should, therefore, always be tried.

Progressive Dementia with Paralysis.—The two cases of bilateral hemiplegia cited above are of this class. Indeed, most neglected hemiplegias of syphilitic origin seem to lead to insanity. But such insanity is by no means always fatal—witness the following case, which illustrates, moreover, the important fact that symptoms of cerebral syphilis *may* disappear without specific treatment:

Case XXXIV.—J. W., a physician, aged thirty, when first seen, in September, 1871, gave the following history:

August, 1870.—Chancre of left index finger. Took mercury. No secondaries noted.

January, 1871.—Squamous lesions of hands and face, iritis, fever, and nocturnal headache. Later, "ulcerated sore throat and blotches on genitals." Continued mercury.

May, 1871.—Began potassium iodid (0.5 gm. t.i.d.), and the headache, which had become intolerable, was promptly ameliorated, but did not disappear.

June, 1871.—While continuing iodid the headache became much worse, and ten days later, while starting on a journey, he was seized with vertigo and vomited his breakfast. He, nevertheless, boarded his train; but as he seated himself was surprised to find he could not put his left hand in his pocket. Alarmed, he attempted to leave his seat, but fell, on account of paralysis

of his left leg. He was helped from the train, had another violent attack of vomiting lasting half an hour (during which time he noticed paralysis of his face and tongue), and then, his paralysis in great measure relieved, walked half a mile to a ferry-boat. After the attack the headache was much less severe, and the paralysis almost entirely disappeared in the course of a week.

August, 1871.—Increased iodid to 4 gm. a day for a few weeks; then went to the country. Headache promptly left him, though he stopped all medicines.

September, 1871.—Headache has relapsed. Consults Dr. Keyes, Sr. The patient is unduly emotional, but intellectually apathetic and slow. Still slightly weak on left side, and face is somewhat drawn to right. His memory is defective. Any attempt to study makes his head feel "queer." His sexual desire is greatly diminished. Urinates thrice at night.—He is ordered to resume iodid at 4 gm., and to return to the country.

October 3d.—Back to town. Stopped iodid after two weeks (indigestion). Headache worse, slight internal strabismus of *right eye* (intermittent), mental condition still unsatisfactory, left leg weak.—Inunctions: sodium iodid 1 gm. t.i.d.

December 15th.—Much improved. Has continued iodid and rubs, and is just salivated: stop. The eye is normal, the mental condition satisfactory, the leg stronger. But the headache, which had disappeared, has relapsed during the last few nights.

January, 1872.—Relapse of headache with internal strabismus of *left eye*. "Mentally unsteady and greatly depressed."—Resume treatment.

September, 1873.—His symptoms were promptly relieved and he neglected treatment. After ten days of diarrhea he passed gradually into a comatose condition. He could neither speak himself nor understand others; he could not so much as raise his hand; he was delirious at times; the bladder was paralyzed, the bowel inactive. He was at this time under the care of a Dr. P., who prophesied his early demise, and gave *no specifics*. Yet after a month his condition began to improve, until on—

December 20, 1873.—Having been out of the house two or three times, he relapsed (without diarrhea) into a stuporous condition, somewhat less than before, but sufficient to keep him in

bed six weeks. Then again he began to mend without the aid of mercury or iodid, and in three months was able to get about.

June 17, 1874.—Returns to Dr. K., having taken no specifics whatever. He is improving rapidly and has gained forty pounds in the past year. His intellect is quite clear, his bladder sound; he looks very well, but has static ataxia and mydriasis, and his head is beginning to ache again. He is still a little emotional; his memory is good, his capacity for work fair.

A child born to him eighteen months ago is fat and well; wife not infected.—Resume treatment.

October 3, 1878.—Has taken very little treatment. He soon became, and has remained, absolutely well. He now has an ulcer of the pharynx which does not yield to bichlorid, gr. $\frac{1}{16}$, t.i.d. He can now take no iodid in any form except tr. iodi; under this and local treatment the ulcer promptly heals.

1881.—Node on sternal end of clavicle "as big as a pullet's egg." Slight static ataxia.—Soon relieved by tr. iodi and bichlorid.

1895, 1898, 1899.—He reports entirely well but for a little dyspepsia. His urine is normal; his daughter sound.

April, 1900.—Left hemiplegia without loss of consciousness.

March, 1901.—Reports to me. He has taken no mercury or iodid since 1881. He retains only a slight limp as a token of his hemiplegia. Otherwise in perfect health.

November, 1906.—Condition remains the same.

It is to be noted that this case, like the two previously cited, may be used as illustrations of syphilitic hemiplegia or syphilitic insanity. The two clinical types are thus inextricably intermingled in many instances, the one predominating at one time, the other at another.

Dementia without Paralysis.—Rare. It often accompanies epilepsy, as noted above.

The recorded outcome of 17 cases of insanity (excluding mania and epilepsy) was 8 "cures," 6 deaths, and 3 unimproved. This proportion of cures is very high, and was possible only because the patients were seen at the very onset of

the attack. Most of these so-called "cures" were doubtless only temporary.

SYPHILIS OF THE SPINAL CORD

The symptoms of syphilis of the cord are more irregular even than those of syphilis of the brain.

Not only are the meningeal and arterial lesions often active over various regions of the cord at once, but they are often associated with active cerebral lesions as well. Hence the clinical picture is apt to be very confused. Indeed, syphilis imitates every disease of the spinal cord.

The chief symptoms of syphilis of the cord are:

Disturbance of the reflexes.
Loss of sexual power.
Pain, anesthesia, and paresthesia.
Paralysis of the bladder and bowel.
Paraplegia and other paralyses.
Ataxia.
Ocular symptoms.
Trophic disturbances.

Disturbance of the Reflexes.—Every syphilitic lesion of the cord causes some disturbance in the reflex presided over by the diseased segment. Inasmuch, however, as syphilitic lesions are usually disseminated, the most superficial examination often suffices. Loss of knee-jerk, without paralysis, suggests tabes. Exaggerated knee-jerk (often associated with ankle clonus) shows a break in the association tracts.

Meningeal lesions not involving the cord (rare) may cause no reflex disturbance.

Loss of Sexual Power.—This prodrome of syphilis of the nervous system is peculiarly common and severe in lesions of the cord.

Disturbances of Sensation.—Pain is due to meningeal lesions or to tabes. Arterial lesions of the cord rarely cause pain. The pain may be of any variety or may affect any nerve. Meningeal pain usually shows the syphilitic nocturnal exacerbation; tabetic pain does not. Tabetic pain is intermittent and sharp or stabbing in character. It is usually most marked in the lower extremities.

Anesthesia may be localized, unilateral, or bilateral. In tabes there is often a band of anesthesia to touch in the mid-thoracic region, which may be associated with analgesia.

Paresthesia and *numbness* are fairly common both in syphilis of the cord and in tabes. They may suggest the level of the lesion. The tabetic usually has numb feet and a constricted "girdle sensation" at the thoracic area of anesthesia.

Paralysis of Bladder and Bowel.—Retention of urine and feces or, far less commonly, inability to retain the dejecta, may be caused by a lesion in the sacral portion of the cord or in any part of the motor tract above. Clinically, obstinate constipation and more or less complete retention of urine are among the commonest, the earliest, the most persistent, and the most threatening effects of spinal syphilis and tabes. The necessary catheterism always infects the bladder, and, if the trophic nerves to bladder and kidney have suffered, these organs fall an easy prey to the infection, and the resulting pyelonephritis may prove fatal. Such a condition must be combated by urotropin, systematic catheterism, and vesical lavage.

Paralysis.—Paraplegia, spinal hemiplegia (Brown-Séquard type), monoplegia, and various irregular paralyses may occur, dependent upon the location and extent of the lesion. If the pyramidal tracts are involved the paralysis is soon followed by a tendency to rigidity and cyanosis, but does not cause atrophy, reaction of degeneration, or bed-sores. Lesions in the anterior horns of gray matter, on the other hand, pro-

duce a paralysis which is often confined to a few muscles, varies in intensity, is not followed by rigidity, and does result in rapid atrophy, electrical reaction of degeneration, and bedsores.

Both the pyramidal tract and the anterior horns may be involved simultaneously.

Ataxia.—That incoördination or disturbance of the associated muscular action essential to the maintenance of equilibrium which we term ataxia "is due to an interference with the reception of sensations of muscular sense . . . sent in from the skin, joints, or muscles. These sensations may be intercepted as they pass through the nerves, for ataxia is a symptom of multiple neuritis; they may be intercepted as they pass through the column of Burdach, as is the case in locomotor ataxia; . . . they may be intercepted in the cerebellum. . . . Ataxia from neuritis is usually accompanied by tenderness in the nerves and muscles. Ataxia from cerebellar disease is present only in the act of walking, and is attended by vertigo" (Starr).

Ocular Symptoms.—Reflex iridoplegia (Argyll-Robertson pupil), which Babinsky and Charpentier [1] consider pathognomonic of syphilis, and which is so early and characteristic a sign of tabes, is sometimes found with syphilis of the cord, though a total paralysis of accommodation to distance as well as to light is much more typical of the latter condition. This total paralysis of the iris is extremely rare in true tabes.

Paralysis of the external muscles of the eye, so frequent during the course of tabes, is even more common in spinal syphilis.

Trophic Disturbances. — Excepting the relative frequency of dystrophic joint changes (Charcot joint) in tabes,

[1] *La syphilis*, 1904, vol. ii, p. 545.

the trophic disturbances due to syphilis of the cord are in no way distinctive.

Despite its great irregularities, syphilis of the cord usually conforms, at least in some measure, to one of the following types:

Meningitis and meningo-myelitis.

Paraplegia.

Syphilitic ataxia with exaggerated reflexes.

Meningitis and Meningo-myelitis.—The meningitis may be localized, diffused, or cerebro-spinal; it may be accompanied or succeeded by lesions of the cord.

Its prime symptoms are pain (with nocturnal exacerbations) at the seat of the lesion and rigidity of the spine (excited by the pain), especially notable if the lesion is seated in the neck.

Secondary symptoms arise from compression or infiltration of the nerves and cord.

Severe syphilitic spinal meningitis is extremely rare. The pain is so marked an early symptom that vigorous early treatment is encouraged, and the prognosis is therefore good.

Paraplegia.—Transverse myelitis, whether due to gumma, thrombosis, or embolism, is uncommon.

The usual type is characterized by the sudden onset (within a few hours) of paraplegia accompanied by anesthesia. The reflexes are exaggerated; bladder and bowel are paralyzed. Within a few weeks contracture of the paralyzed muscles occurs; the muscles atrophy and bed-sores appear as the end approaches. The site of the lesion may usually be determined by an area of hyperesthesia, if not of actual pain, and also by the extent of the paralysis and anesthesia.

Some improvement, if not a complete cure, may be looked for if the case is vigorously treated before the muscles become contractured. After this little can be done.

Brown-Séquard Paralysis.—Hemiplegia with crossed hemianesthesia, due to disease of one half of the cord, is usually caused by syphilis.

Syphilitic Ataxia with Exaggerated Reflexes.—I have employed the above stupid title because the disease is so commonly mistaken for *true ataxia with abolished reflexes*. The two conditions differ in many of their clinical details; but recognition of the fact that *true tabes never produces exaggerated reflexes* will save one from the gross error of confounding them.

Prodromes.—The onset is usually extremely insidious. The first symptom may be sexual impotence, paralysis of the bladder or bowel, or irregularly distributed paresthesia. This first symptom may remain unique for many months, and by proper treatment the disease may often be checked before it progresses any farther.

Ataxia.—The chief subjective symptom is a slowly progressive ataxia. This ataxia is due to voluntary spastic contractions of the muscles. The patient walks stiff-legged, with little steps, scarcely lifting his feet from the ground. But put his muscles at rest and they appear entirely normal, neither spastic nor paralyzed; their electrical reaction is unchanged.

This mincing ataxia is utterly different from the incoördinate kicking ataxia of tabes.

Exaggeration of the Reflexes. — The knee-jerk is greatly exaggerated and ankle clonus is marked. Babinski reflex is almost always present.

Vesical Paralyses.—The vesical and rectal paralyses, though usually present, may be slight.

Course.—The progress of the disease is generally very slow. It often requires repeated courses of treatment to effect a cure—and a cure, all too often, means nothing more than checking the progress of the disease. I have seen a sailor, after many months of spinal syphilis, return to the shrouds, a builder to his ladder; but such cases are exceptional. The

best we can do, as a rule, is to prevent the symptoms from becoming any worse.

PARASYPHILIDS

An adequate brief description of the parasyphilids is impossible here. Rather than attempt a hasty sketch of them, let us merely refer to the few remarks in Chapter VII.

SYPHILIS OF THE NERVES

Syphilis of the nerves is usually secondary; an involvement from disease of the brain, cord, or meninges at their origin, or from the bones or soft tissue that surround them in their course.

Primary [1] syphilitic neuritis is quite rare. One or more nerves may be involved. Its symptoms suggest deficient nerve conduction: thus there is paralysis, anesthesia, loss of reflexes, but, above all things, *pain*. Neuritis and neuralgia are almost synonymous.

The *diagnosis* is often obscure. The only distinction between syphilitic neuritis and that due to another cause is the nocturnal exacerbation characteristic of the syphilitic pain.

The *treatment* is especially by iodids.

DIAGNOSIS

Nerves.—The diagnosis of syphilis of the nerves depends, as we have seen, upon the character of the pain, the history, and the effect of treatment. But one caution must be added: however positive one may be that the inflammation is syphilitic, one can rarely be absolutely certain that it is in the nerves

[1] I. e., beginning in the nerve; not a secondary extension from some other tissue.

alone and not in the central nervous system as well. Hence one must always be on the lookout for symptoms of central involvement.

Brain and Cord.—The features differentiating syphilis from other diseases of the central nervous system are:

1. Prodromes.
2. Irregular course.
3. Irregular involvement.
4. Age of the patient.
5. Occurrence of certain lesions.
6. History and evidences of previous syphilis.
7. Examination of the cerebro-spinal fluid.
8. Amenability to treatment.

Prodromes.—These have already been discussed (page 378).

Irregular Course.—The three case-histories cited are absolutely typical of syphilis in their confusing advance and retreat from one to another symptom of nervous disorder.

Irregular Involvement.—Equally characteristic is the multiplicity of heterogeneous symptoms; e. g., the juxtaposition of epilepsy with facial paralysis, of hemiplegia with dementia—and the incompleteness of these symptoms; e. g., partial paralyses, irregular areas of anesthesia, temporary or incomplete loss of the mental functions.

Age of the Patient.—As we have seen, the later the syphilis is acquired, the earlier do its nervous symptoms appear. Hence, syphilis of the nervous system begins almost always between the ages of twenty-two and forty-four,[1] at the very time of life when other lesions of the nervous system are most uncommon.

Now this fact is of especial import in diagnosing two

[1] Out of 368 cases, 2 began at nineteen, 1 at twenty-two, 311 (85 per cent) between twenty-two and forty-four, 28 between forty-five and fifty, 24 between fifty-one and fifty-nine, 1 at sixty-one, and 1 at sixty-three.

lesions: viz., epilepsy and hemiplegia. *Epilepsy beginning after the thirtieth year is almost always syphilitic*, for "idiopathic" epilepsy is a disease of early youth. Moreover, *hemiplegia occurring before the forty-fifth year is almost always syphilitic*, for hemorrhage, thrombosis, or embolism due to other causes is extremely rare until the patient is more than forty-five years old.

Typical Syphilitic Conditions.—Certain symptoms are very suggestive of brain syphilis: such are Jacksonian epilepsy, paralysis of the third and to a less degree of the second cranial nerves, and, as we have just seen, hemiplegia before the age of forty-five, or epilepsy beginning after the age of twenty years. All partial paralyses, hemiplegias without loss of consciousness, as well as all complicated, irregular, and abortive symptoms are also gravely suspect.

History.—Excepting tabes and paresis, syphilis of the nervous system almost always gives evidence of previous syphilitic lesions.

Examination of Cerebro-spinal Fluid.—The characteristic lymphocytosis caused by syphilis is usually (though not always) present, and may confirm the diagnosis of a doubtful case.

Treatment.—The test of treatment is always the court of last resort. Let us once again insist that apparent improvement under treatment is not *absolute* proof that the lesion is syphilitic nor is resistance to treatment *absolute* proof that it is not.

CHAPTER XXVII

SYPHILIS OF THE EYE[1]

Syphilis strikes the eye so frequently in certain regions, so rarely in others, that it is more convenient to consider its lesions in order of their frequency rather than in any cut-and-dried anatomical precedence.

Accordingly, we may take up in turn iritis, chorio-retinitis, and optic neuritis, closing with a brief survey of the rarer manifestations of acquired ocular syphilis.

IRITIS

Inflammation of the iris occurs in at least half of all cases of ocular syphilis. It is said to be more frequent in men than in women. It affects some three to four per cent of all syphilitics (Terrien[2]). It occurs usually between the fourth and the twelfth month of the disease. Half the cases of iritis seen by the ophthalmologist are said to be syphilitic.

Etiology.—While the connection between eye-strain and syphilitic iritis is by no means established and grosser trauma can hardly be invoked as a cause, it is nevertheless evident that a severe case of syphilitic iritis predisposes the patient to relapse. But this predisposition is due rather to the permanence of adhesions than to inefficient treatment of the first attack.

Double iritis, the inflammation in one eye following close upon that in its fellow, is very common.

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[1] Reviewed by Dr. John Izard Middleton.

[2] "Syphilis de l'œil," Paris, 1905.

Pathology.—The inflammation is usually exudative in type (plastic iritis). There is exudation within the iris, thickening it and dulling its luster; the iris is contracted and almost immobile. The congestion always extends to the ciliary body, though there is rarely any symptom of its involvement. The vessels about the cornea are dilated and form the characteristic circumcorneal congestion.

If the inflammation is severe desquamation into the anterior chamber produces a sediment behind the cornea which occasions a spotted appearance upon its lower segment, and is therefore inaccurately entitled "punctate keratitis."

A neglected case results in adhesions between the iris and the lens, which, if marked, may destroy vision.

Exceptionally, there occurs what is known as the papillomatous or condylomatous [1] iritis, characterized by the eruption of a papule (rarely more than one), the size of the head of a pin, projecting from the pupillary border of the iris or from its anterior surface. True gumma of the iris is extremely rare.

Symptoms.—The subjective symptoms are *hazy vision* and *pain*, which, if severe, excites photophobia and a constant lacrymation. These symptoms vary in intensity, but are usually slight. The pain is situated in the eye, brow, or temple indifferently.

The objective symptoms upon which a diagnosis of iritis is based are found in the iris, the pupil, the cornea, and the sclera.

The *iris* is dull in color, looks thick, and often is more greenish or rusty than its fellow. It is fixed in contraction (does not expand in darkness), and reacts much more slowly to mydriatics than its fellow. If adhesions (synechiæ) have already formed between it and the lens, instillation of atropin

[1] Erroneously termed gummatous, for these little tumors scarcely ever degenerate. But they are yellowish and therefore *look like* gumma.

either does not dilate the pupil at all or, more often, does dilate it somewhat, but in a characteristically irregular manner. The adhesions binding portions of the iris impart to the dilated pupil all sorts of fantastic shapes (clover shape, heart shape, etc.).

An exceptionally mild inflammation shows only a slight discoloration or thickening, which may be confined to the pupillary border.

A grave lesion, on the other hand, apart from the marked discoloration and thickening of the iris with salient blood-vessels and its prompt and firm adhesion to the lens, may produce the yellowish pinhead papules projecting from the pupillary border or the broader yellowish papules on its anterior surface.

The *pupil* is, as already stated, small, fixed, and, if dilated, often irregular. It is also blurred in severe cases by the iritic exudate.

The *cornea* may be seemingly spotted (the so-called punctate keratitis) or blurred by exudate into the anterior chamber.[1] There may, moreover, be actual keratitis.

The *sclera* reveals to the examining eye and finger two most important objective symptoms of iritis—circumcorneal injection and the presence or absence of tenderness.

Circumcorneal injection consists in dilatation of the anterior ciliary vessels surrounding the cornea. The cornea is surrounded by a bright-red circle of congestion, which gradually thins out toward its periphery into a series of dilated little vessels, extending for some distance radially over the sclerotic. This sign is constant and characteristic of iritis. In order to distinguish this congestion from true conjunctivitis one has but to touch the conjunctiva lightly, moving it from side to side upon the eyeball, whereupon the immobility of the vessels shows that they are beneath and not in the conjunctiva.

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[1] Gelatiniform exudate is rare: purulent or bloody exudate most exceptional.

By pressure upon the sclera through the upper lid we discover the tenderness and tension of the eye as compared with its fellow. In the ordinary mild case of iritis ocular tension is slightly diminished, and there is little or no circumcorneal tenderness. The presence of extreme sensitiveness to pressure shows cyclitis and suggests the possibility of grave involvement of the choroid or retina, while increased tension is an even more ominous sign, since it is a threat of glaucoma.

Complications.—Let us not forget that iritis may be the most obvious though the least ominous lesion of syphilis of the eyeball. It is not proper to speak of such important lesions as chorio-retinitis, optic neuritis, keratitis, gumma of the ciliary body, etc., as complications of iritis. It is the iritis rather that complicates them.

The most important complication resulting from iritis is the persistence of adhesions with the implied impairment of vision and danger of relapse and of ultimate glaucoma.

Diagnosis.—The physician must learn whether there is iritis, whether the iritis is syphilitic, and whether the syphilitic iritis stands alone or is but a complication of other and graver lesions.

The *diagnosis of iritis* need not be discussed here. Suffice it to remark with Theobald that " speaking generally, the presence of iritis is to be suspected whenever, without increase of intra-ocular tension or other evident cause, pain in and around the eye, usually worse at night, is complained of and is accompanied by circumcorneal injection and a contracted pupil." Such a suspicion is confirmed by the discovery that the iris is thick, discolored, contracted, and sluggish. The presence of adhesions and of punctate keratitis drive the conclusion home.

But in all this there is nothing to show that *the iritis is syphilitic*. Indeed, except for the yellow papules (that occur so rarely) there is no sign to distinguish syphilitic iritis from

that due to whatever cause. Yet the diagnosis is almost invariably established by the presence of other syphilitic lesions and a history of recent "early syphilis." The iritis standing alone is very likely to be dubbed rheumatic.[1]

Is the iritis but one of a number of ocular lesions? This question always merits an accurate answer; for, while no lesion is more benign than the mild iritis of early syphilis, no lesion is more fatally misleading than that same iritis when it conceals the onset of chorio-retinitis.

Hence every syphilitic iritis demands a thorough ophthalmoscopic examination at the outset.

Prognosis.—The prognosis of uncomplicated syphilitic iritis is absolutely good. Dilate the pupil to prevent adhesions, and the iritis will soon disappear spontaneously in most instances.

This tendency to spontaneous resolution has persuaded many ophthalmologists to neglect the specifics in the treatment of iritis.[2] But their arguments apply equally to the early secondary syphilids which iritis accompanies. Moreover, one can never be absolutely sure that an apparently benign iritis is not a warning of graver lesions to come.

Treatment.—The iris must be immediately dilated by the instillation of two drops of a three per cent solution of atropin sulphate twice a day (or oftener if this does not suffice) until full dilatation is attained, and then kept dilated by a one per

[1] Fournier lays great stress upon the insidious onset and benign character of syphilitic iritis as a diagnostic feature. "The less the iritis disturbs the patient the more it should disturb the physician," is his rule.

[2] Thus Noyes ("Genito-Urinary Diseases with Syphilis," Keyes, 1888, p. 621) says: "I have no hesitation in stating that the usefulness of either mercury or iodin to cure iritis is exceptional and not the rule. . . . I have several times observed patients with active iritis in one eye, who have already been brought under the influence of mercury, attacked with the same inflammation in the other. This certainly proves that no preventive virtue can be ascribed to the mercury, and argues against the beneficial influence of quick mercurialization in curing the acute attack."

cent solution until the inflammation subsides. In case the dilatation is not begun until light adhesions have formed, these will gradually break up; but if they do not yield, and are dense and broad, they require iridectomy after the subsidence of the attack.

The eye must be protected from the light by smoked glasses.

Local applications, such as leeches, boric acid or lead and opium compresses, may be employed *secundum artem.*

If there is hypertension atropin should not be used, and an operation will probably be required to relieve the increased tension.

General treatment is mercurial; mild if the iritis is slight, pushed severely if it is grave, accompanied by iodid if other lesions require it.

CHORIO-RETINITIS AND RETINITIS

Syphilis is the most frequent cause of chorio-retinitis,[1] one of the rare causes of retinitis. Yet, though syphilitic chorio-retinitis is far less rare than syphilitic retinitis, it is not, for all that, a frequent lesion. It is said by some authors to be most often due to hereditary syphilis, while others attribute it usually to the acquired form of the disease. It may be accompanied by iritis. It is usually unilateral.

Pathology.—The vessels of the choroid are congested and infiltrated, there are foci of yellow or white exudate upon its surface, which are later surrounded by pigment deposit, and a fibrous exudate into the vitreous.

The changes in the retina are a hyperemia and edema. The ophthalmoscope shows haziness and discoloration of the retina; the arteries appear thickened and surrounded by a whit-

[1] The choroid can scarcely be said to be inflamed without some affection of the superposed retina.

ish exudate at first, while late in the disease they are obliterated and reduced to fibrous threads.

Hemorrhages, sclerotic patches, pigmentary (unilateral) retinitis, detachment of the retina, De Graefe's relapsing central retinitis, etc., are among the rare results of ocular syphilis.

Symptoms.—*Dimness of vision* is the cardinal subjective symptom of chorio-retinitis and retinitis. Since this dimness of vision progresses very slowly and painlessly, and is always unilateral at first, the patient usually does not seek relief until it has existed for some time and wrought organic and uncurable lesions in the retina. Therefore, the prognosis is generally bad.

Analysis of this dimness of vision shows it to be a haziness, a fog, a dense, constantly moving cobweb, through which objects are more or less obscurely seen. Moreover, objects are likely to appear diminished in size (rarely deformed), and certain colors are distinguished only partially or not at all. Vision is markedly impaired when the light is at all dim, and the patient's outlook upon the world may be still further confused by the presence of dancing flames and flecks of light (photopsia).

Pain is absent or slight.

The ophthalmoscope shows a haziness in the vitreous, obscuring the papilla,[1] but clearing toward the periphery. This haziness is constituted by flakes of fibrin, varying widely in size in different cases, or in the same case from time to time. Indeed, great variations occur in the density of this haze that obscures the sight, but these remissions do not necessarily imply a good prognosis.

Implication of the choroid is evinced by the exudative foci. These foci at first form yellowish projections surrounded by a pigmented circle; they lift up the retina. They last a few

[1] The papilla looks like "a moon seen through clouds."

weeks, and then disappear, leaving pitted, bluish-white scars amidst irregular masses of pigment. The scars represent complete destruction of the retina and choroid by the exudate; so that when it withers the white sclera appears, more or less obscured by little agglomerations of pigment.

Meanwhile the papilla is slowly fading. It becomes a dirty yellow, then gray, and finally is completely atrophied. This process usually takes many months, and by the time it is well advanced the other eye is also involved.

Varieties.—In order of frequency the chief varieties are:

Disseminate Chorio-retinitis. — The inflammation is general, the papules are numerous around the periphery of the fundus, rarer toward the center.

Anterior Chorio-retinitis.—Especially common in hereditary syphilis accompanying interstitial keratitis (page 531). The papules affect only the extreme periphery, and require a very careful examination to discern them. If the papules do not invade the fundus, the prognosis, as regards vision, is good.

Irido-choroiditis.—Attacks of iritis in the course of a choroiditis are not uncommon, and are an evil omen unless the choroiditis is extremely slight (anterior chorio-retinitis, areolar chorio-retinitis).

Areolar Chorio-retinitis.—The papules are few and irregularly distributed. This, like the anterior chorio-retinitis, is a partial and benign type of "disseminate" lesion.

Central Chorio-retinitis.—The choroid lesions are centered at the papilla and macula; sight is rapidly impaired.

Retinitis.—The lesions are those of chorio-retinitis less the exudate. Special varieties, such as relapsing central retinitis (De Graefe), retinitis with annual scotoma (Perlia, Bull), etc., are chiefly of interest to the specialist. Pigmentary retinitis is often due to hereditary syphilis.

Prognosis.—The prognosis is bad. The lesions are usually so far advanced before treatment is instituted that checking the disease only partially restores sight.

Neglected cases not only lose the sight of both eyes, but may go on to cataract or to glaucoma, requiring enucleation.

Treatment.—These graver lesions of ocular syphilis require sharp mercurialization, preferably by intramuscular injection combined with repeated short, sharp courses of iodids.

For local treatment little can be done.[1] Terrien speaks well of pilocarpin hypodermically.

OPTIC NEURITIS

The optic nerve is said to contribute twenty-five per cent of ocular syphilis. It is one of the common nerve lesions (cf. page 372).

The symptoms of optic neuritis: viz., amblyopia, blindness, flittering scotoma, and hemianopsia, may or may not be due to lesions visible through the ophthalmoscope. Accordingly, some cases (and they are the milder ones) are distinguished only by subjective symptoms, while others (and they are the majority) show visible lesions of the papilla.

On the other hand, one may occasionally discover a choked disk in a patient who shows no ocular symptoms.

Pathology.—Lesions of the optic nerve may be primary or secondary; i. e., due to syphilis of the nerve itself or to lesions of the surrounding parts. It is thus often associated with other evidences of basilar meningitis, or gumma of the base of the brain, or of the sphenoidal fissure, or within the orbit. The commonest syphilitic cause of optic neuritis is basilar menin-

[1] Subconjunctival injections of normal salt solution, gelatin (five per cent), cyanid of mercury (1: 1,000), and iodo-iodid solution) iodin 0.02; potass. iodid. 2.; aq. ad 40) are commended for various ocular lesions. But the best authorities do not favor such treatment. Cf. Bull, *J. Am. Med. Ass'n.*, 1906, vol. xlvii, p. 823.

gitis. Although primary (intraocular) lesions of the nerve are habitually associated with chorio-retinitis, and secondary lesions with other evidences of syphilis of the nervous system, it is not always possible to ascertain precisely the site of the lesion; nor is it always a simple matter to decide whether a secondary optic neuritis is due to syphilis or to brain tumor.[1] Apart from the diagnostic signs alluded to in the preceding chapter (page 408) one must often apply the therapeutic test.

The lesions visible by ophthalmoscope are neuro-retinitis, papillitis, and optic atrophy. There is nothing distinctively syphilitic about any of them. They may, therefore, be dismissed with brief mention.

Neuro-retinitis.—Already alluded to; varies from simple hyperemia to marked congestion, edema, and blurring of outline.

Papillitis.—Papillitis, or *choked disk*, is a condition of marked edema. The nerve is much swollen and projects forward from the surrounding retina. It is opaque, red, and striated, its arteries small, its veins much congested, its edge indistinct.

The cause of choked disk is interrupted venous return, due in part to pressure upon, in part to inflammation of the nerve. Its outcome, if neglected, is optic atrophy.

Optic Atrophy.—The optic nerve may atrophy "primarily" or subsequently to chorio-retinitis or papillitis.

When the atrophy is "primary" the papilla gradually loses its rosy color and becomes white or gray[2]; its outline is clean-cut, and the retinal vessels are not markedly changed.

Optic atrophy following papillitis, on the contrary, pro-

[1] Bilateral optic neuritis is almost an infallible sign of basilar tumor or meningitis.

[2] Gray atrophy is usually due to tabes, white atrophy to cerebral cause; but this rule is not without exceptions.

duces a white or gray papilla with irregular vague outline, and with tortuous retinal veins and slender sclerotic arteries.

Optic atrophy following chorio-retinitis shows the scars of the choroidal exudate scattered over the fundus.

Symptoms.—There may be symptoms without ophthalmoscopic signs of optic neuritis, less often signs without symptoms, most often both together. The symptoms are similar to those of chorio-retinitis, and consist chiefly of a *gradual loss of vision.* Sudden and temporary improvement or diminution of vision is characteristic of disease of the optic nerve rather than of the retina.

The disease may be checked by mixed treatment. But if actual atrophy has occurred no treatment will restore sight.

Diagnosis.—The diagnosis is made by the ophthalmoscope, with the reserve that the lesions are not always visible at the onset.

Treatment.—As for the cerebral lesions, with which it is usually connected.

GUMMA OF THE UVEAL TRACT

Iris.—Gumma of the iris has been mentioned; it is extremely rare.

Ciliary Body.—Panas [1] has collected thirty cases. The gumma appears as a complication of iritis. The tumor involves and pierces the sclerotic, appearing as a little granulating mass; it also invades the iris. It may be distinguished from tuberculosis by the history and associated lesions. Instillations of atropin and sharp mixed treatment are demanded.

Choroid.—Terrien has collected six cases, and says: "The ophthalmoscope reveals a white neoplasm in the fundus. The gumma is soon complicated by iritis, optic neuritis, and even by rupture of the sclera at the point affected. It may be mis-

[1] *Arch. d' Ophthal.*, 1902, August, p. 485.

taken for sarcoma, but the progress of the disease and efficiency of (mixed) treatment should prevent this error."

CONJUNCTIVAL SYPHILIS[1]

Slight *congestion of the conjunctiva* may accompany the first general secondary outbreak; severe congestion, simulating trachoma, is extremely rare.

Papular conjunctivitis is a rare complication of iritis (or of a papular eruption upon the skin of the eyelids). The papules may be movable or attached (episcleritis).

Mucous papules and ulcers are sometimes seen upon the edge of the eyelid.

Conjunctival gummata have been noted. They are usually single and situated at some distance from the cornea; they arise from the subconjunctival tissue.

KERATITIS AND SCLERITIS

Interstitial keratitis is so uncommon in acquired syphilis,[2] and so characteristic of inherited syphilis that we may defer its description (page 531).

Episcleritis without conjunctivitis is extremely rare. One or more small tubercles appear near the cornea and adherent to the sclera. It is usually a complication of iritis. It is differentiated from rheumatic episcleritis by the absence of sensitiveness (Terrien).

Scleritis is constituted by one or more similar tubercles embedded in the sclera. Panas has suggested that these are actually an extension of a gumma of the ciliary body.

Gumma of cornea and sclerotic are alleged, but not proven to exist.

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[1] Cf. Pusey, *J. Am. Med. Ass'n.*, 1907, vol. xlix, p. 828.

[2] My records contain only two cases.

SYPHILITIC TARSITIS

Occurs in the latter stages of syphilis, and is to be regarded as a gummatous infiltration of the tarsus. Develops gradually, and without noticeable pain. At its height there is great enlargement of the lids, and their skin is reddened and stretched. The tarsus itself feels hard, and does not allow eversion of the lid, for examination; also appears pale and lardaceous. The lashes fall out. The preauricular glands swell. The course is tedious, affection lasting some weeks, but yielding to specifics.

THE LACRYMAL APPARATUS

The Lacrymal Gland.—Syphilitic dacryoadenitis is extremely rare. It is characterized by a painless infiltration of the gland, forming a little tumor in the upper and outer portion of the lid. It is usually mistaken for tuberculosis.

The Tear Duct.—Syphilis of the lacrymo-nasal duct occurs only in connection with extensive ulcerations of the nose or destruction of the nasal bones. The resulting stricture (after the inflammation has been controlled) requires dilatation.

ORBITAL LESIONS

Osteoperiostitis and gumma of the orbital bones are important chiefly on account of their effect upon the eye.

Inflammation in the edge of the orbit excites the usual symptoms of bone syphilis, i. e., localized pain and swelling, and involves the lower lid in the inflammation. Deeper in the orbit it may cause exophthalmos, optic neuritis, diplopia (by muscle or nerve involvement), and even neuro-paralytic keratitis, etc. It may terminate in resorption, gummy degeneration, or suppuration.

CHAPTER XXVIII

SYPHILIS OF BONE

Bone syphilis and brain syphilis resemble each other in this important particular; either may manifest itself first by pain (osteocopic pains, headache) at the time of the first general outbreak of symptoms, and later the disease may follow one of three courses, viz.:

1. The pain having been relieved by time or treatment no further bone (or brain) lesions may appear, or

2. The pain may be followed (with or without an interval of remission) by grave bone (or brain) lesions, or

3. The grave lesions may attack a bone (or brain) in which no pain was felt at the onset of secondary symptoms.

Thus the early osteocopic pains, which have been already described (page 263) need concern us here only inasmuch as they may precede a graver lesion.

The bone may be *secondarily* involved by syphilis of the surrounding tissues, or the inflammation be *primary* in the bone itself or in its periosteum. Of the secondary lesions, the only important ones are the bone lesions of nose and palate already described (page 363). The chief primary lesions are indicated in the table on the opposite page.

In this table all lesions, whether unilateral or bilateral, periosteal or medullary, productive or gummatous, are grouped together.

Bilateral Lesions.—Though bilateral, symmetrical, periosteal tenderness is preëminently an early symptom; bilateral

OCCURRENCE OF BONE LESIONS[1]

Year	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16–26	Over 26	2	3	Total
Tibia	9	13	19	10	7	5	5	3	3	3	2	2	2	2	2	6	1	25	88	119
Parietal	0	4	4	6	2	1	4	0	1	1	1	1	1	0	0	3	0	6	10	35
Frontal	1	2	4	5	2	1	0	1	0	0	1	1	1	0	1	1	1	7	11	29
Sternum	0	1	1	3	0	1	2	0	0	0	0	0	0	0	1	2	1	0	4	12
Clavicle	0	0	1	1	0	1	1	0	0	0	0	3	1	0	1	1	0	1	3	11
Ulna	0	0	0	4	1	2	1	0	0	0	0	0	0	1	0	0	0	1	0	10
Femur	1	0	0	2	1	0	0	0	1	0	1	1	0	0	0	0	0	2	3	9
Ribs	0	2	1	0	0	1	0	1	0	0	0	0	1	1	0	0	0	2	5	9
Lower jaw	0	0	0	3	1	1	0	0	0	0	1	1	0	0	0	0	1	1	3	9
Metacarpals	1	0	2	0	1	1	0	1	0	0	1	1	0	0	0	0	0	0	3	8
Fingers	0	1	3	0	0	0	1	0	0	1	0	0	0	0	0	1	0	1	6	8
Fibula[4]	0	0	1	1	0	0	0	0	0	1	0	0	0	1	0	0	0	1	3	5
Other bones[5]	1	2	3	0	2	0	4	2	1	1	0	1	0	1	1	2	0	5	22	26
Total	13	25	39	35	17	14	18	8	6	7	7	11	6	6	6	16	4	52	161	290

[1] Exclusive of nose and palate (p. 350), and joints (p. 445).

[2] Year not stated.

[3] Unique lesions (no other bones involved).

[4] The X-ray shows involvement of the fibula in a surprisingly large number of cases.

[5] Superior maxilla, calcaneum, humerus and temporal thrice; occipital, patella, ilium, radius, and metatarsus twice; scapula, sacrum, malar, and tarsus once.

graver lesions are distributed relatively late. Of 37 recorded bilateral lesions (31 tibial), 11 are of unknown date, 9 occurred in the first three years, 8 in the next three, 3 in the ninth, and 1 in the tenth, twelfth, thirteenth, fifteenth, and twenty-first years.

Multiple Lesions.—Though diffuse lesions are not uncommon, multiple, discrete lesions of one bone are rare.[1] But the 290[2] lesions in the table occurred in 224 cases, there being 63 in which two or more bones were involved (exclusive of the 37 bilateral, symmetrical lesions).[3]

ETIOLOGY

The connection between trauma and syphilis is best illustrated by lesions of bone. One sees gumma follow a contusion, the kick of a horse, the application of a set of false teeth, and other equally obvious examples of cause and effect. And the impression founded upon such observations is fortified by estimation of the relative frequency of involvement of the various bones. With the tibia, the bone most subject to trauma, easily first, and such subcutaneous bones as the parietals, frontals, sternum, clavicles, etc., following, there is doubtless some connection between the position of a bone, subjecting it to the possibility of trauma, and the occurrence of syphilis in it. Add to this the fact that the lesions are chiefly periosteal, that they usually occur upon the surface of the bone most exposed to trauma (e. g., anterior surface of the tibia, exterior surface of the cranial bones) and the picture is complete. But in most instances the direct connection between trauma and syphilis cannot be established, and surely of the many bruised shins few become syphilitic.

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[1] Excepting the cranial bones.

[2] Counting bilateral lesions as one.

[3] The distribution of unique lesions is shown in the table.

Mercury injures the bones only inasmuch as very severe salivation may destroy portions of the alveolar processes of maxilla or mandible.

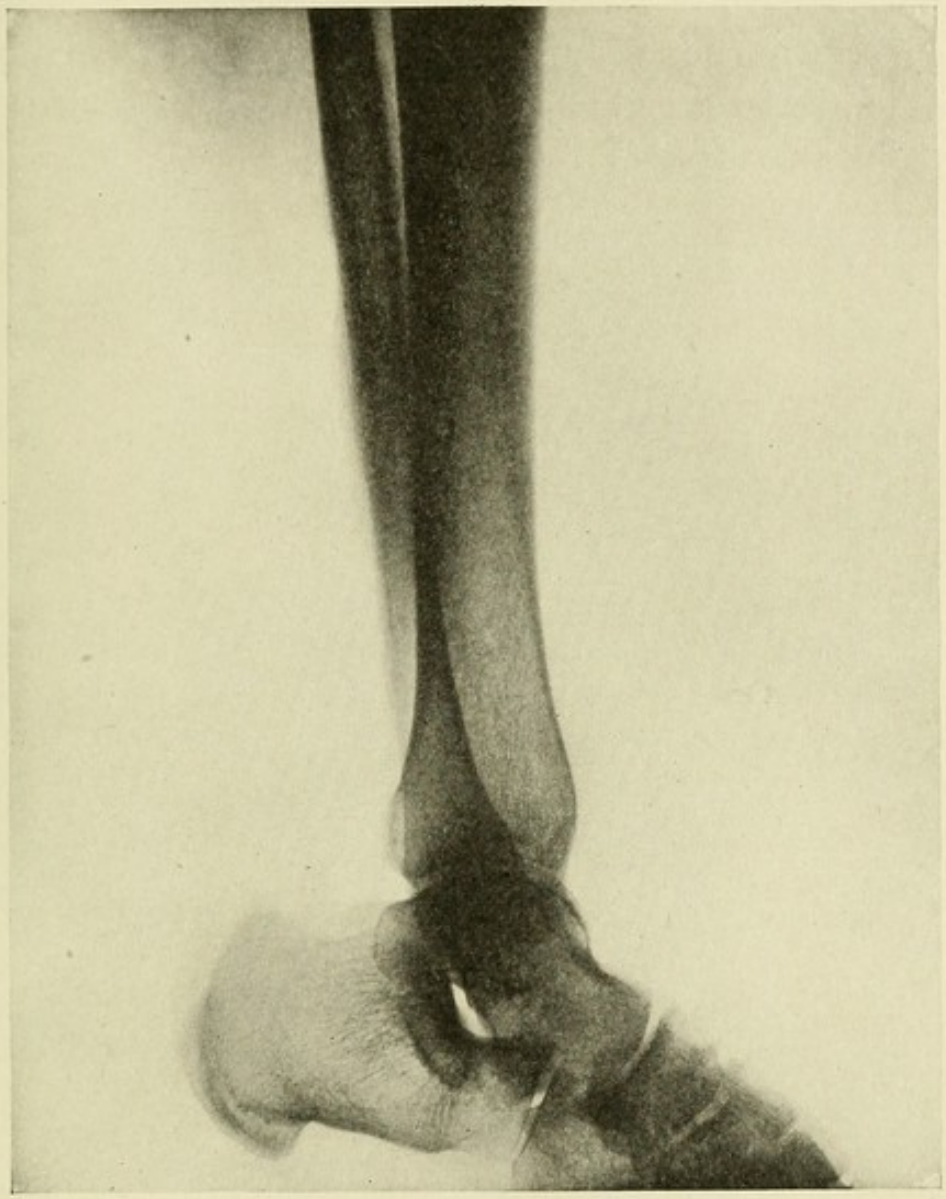

FIG. 52.—SYPHILITIC PERIOSTITIS (HEREDITARY) OF SHAFTS OF TIBIA AND FIBULA. Note gradual transition from healthy bone at malleolus to indefinite osteosclerosis above. Note narrowing of medullary canal. (Case of Dr. Jaeger. Skiagraph by Dr. Cole.)

PATHOLOGY

The lesions of bone syphilis, in order of their frequency, are:

Osteoperiostitis.

Gummatous osteoperiostitis.

Osteomyelitis.

Osteoperiostitis.—The lesion begins with exudation into the deeper layers of the periosteum, thence it extends inward, invading the bone, and to a less degree outward, invading the overlying soft tissues.

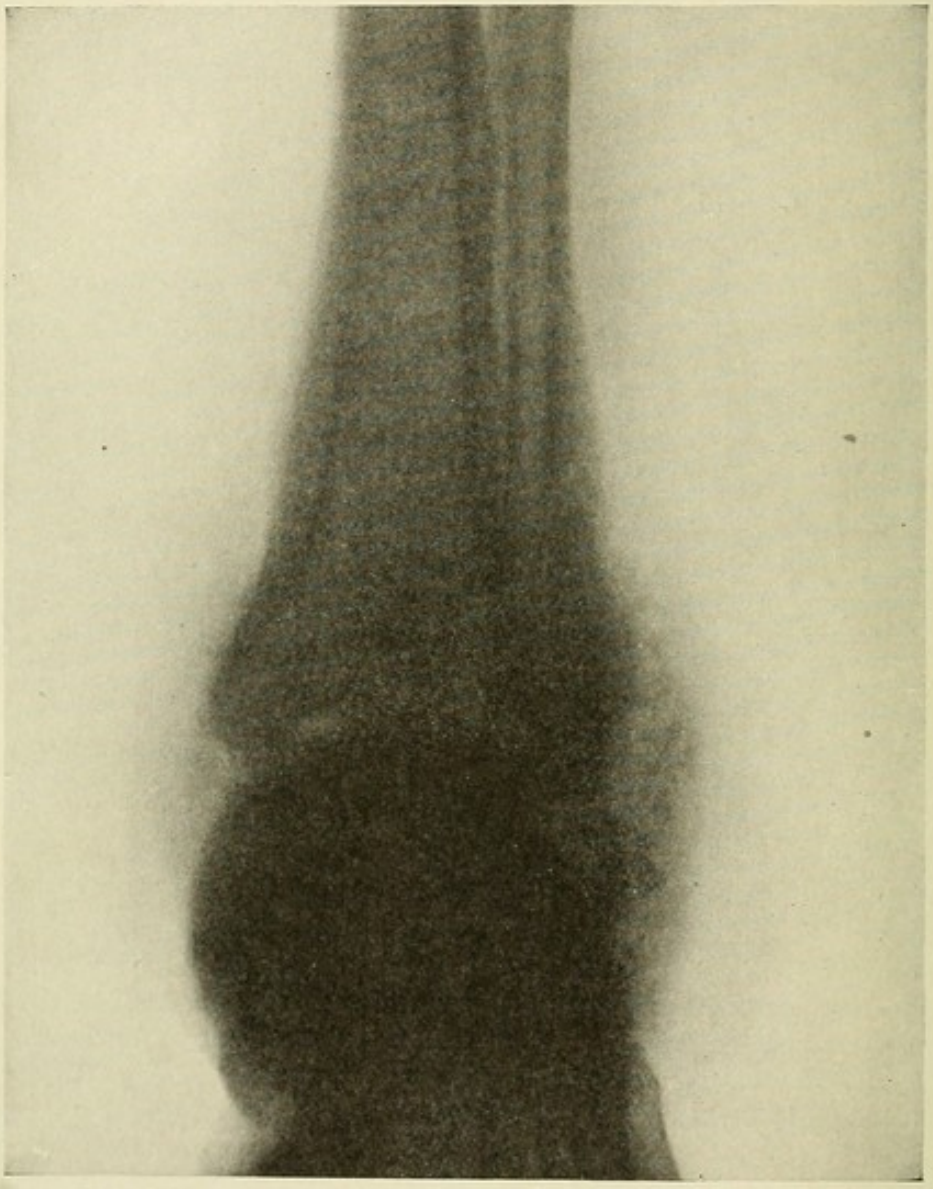

FIG. 53.—OSTEOPERIOSTITIS (HEREDITARY) OF CARPUS AND TO A LESS DEGREE OF RADIUS AND ULNA. The carpus is utterly blurred, but in radius and ulna the diffuse irregular thickening of the periosteum with osteosclerosis (ulna) is typical. (Case of Dr. Jaeger. Skiagraph by Dr. Cole.)

The invasion of the bone takes place by way of the vessels: the Haversian canals are filled with small, round cells;

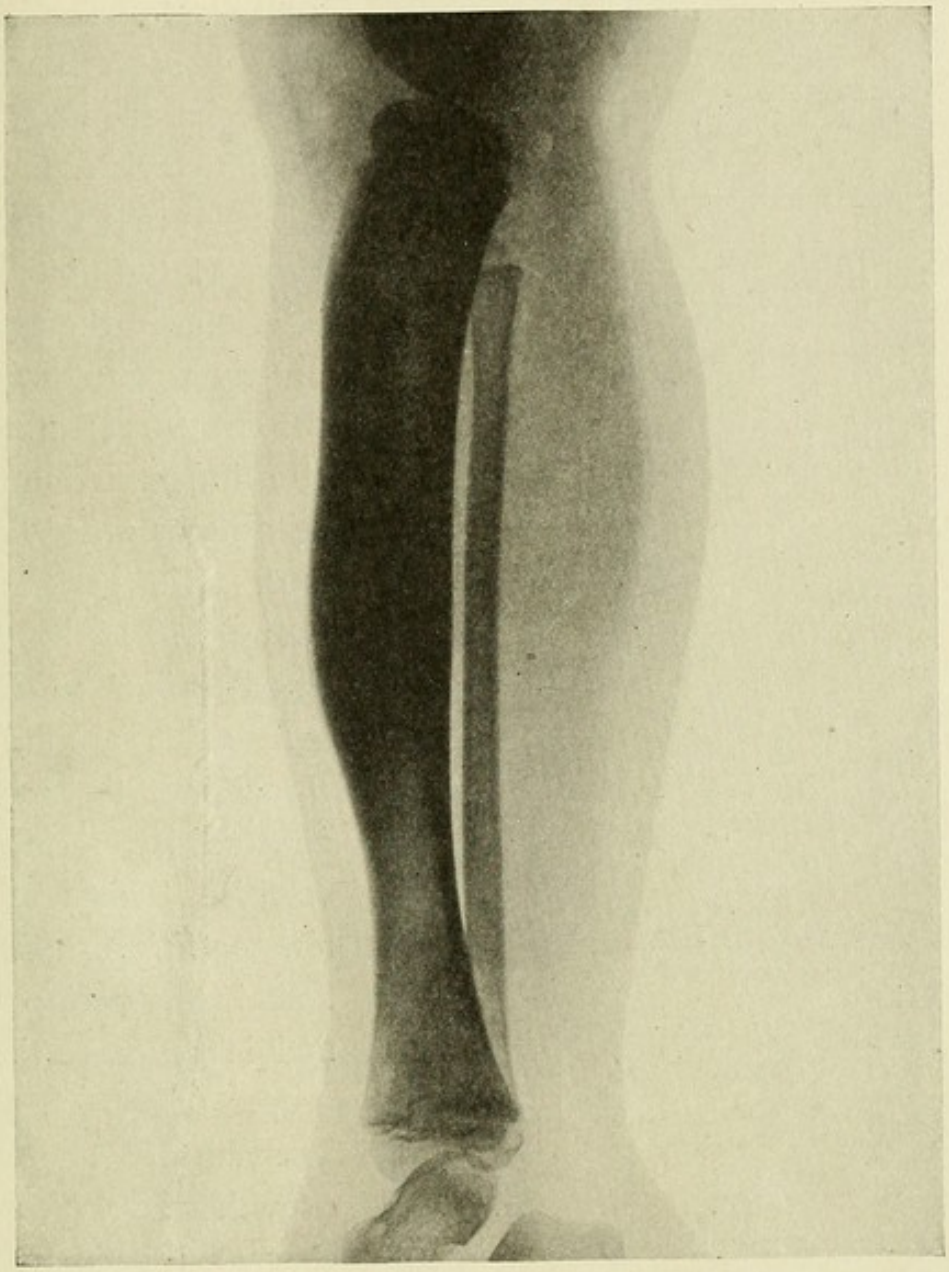

FIG. 54.—SABER TIBIA OF HEREDITARY SYPHILIS. Diffuse osteosclerosis of shaft of tibia projecting outward and also encroaching upon the medullary canal. This lesion is quiescent and does not show the light thick periosteal shadow as an active lesion (Figs. 52, 53) does. (Case of Dr. Jaeger. Skiagraph by Dr. Cole.)

the intervening layers of bone are eroded, and may disappear through absorption (rarefying osteitis) or necrosis.

These processes may be diffused over a considerable area

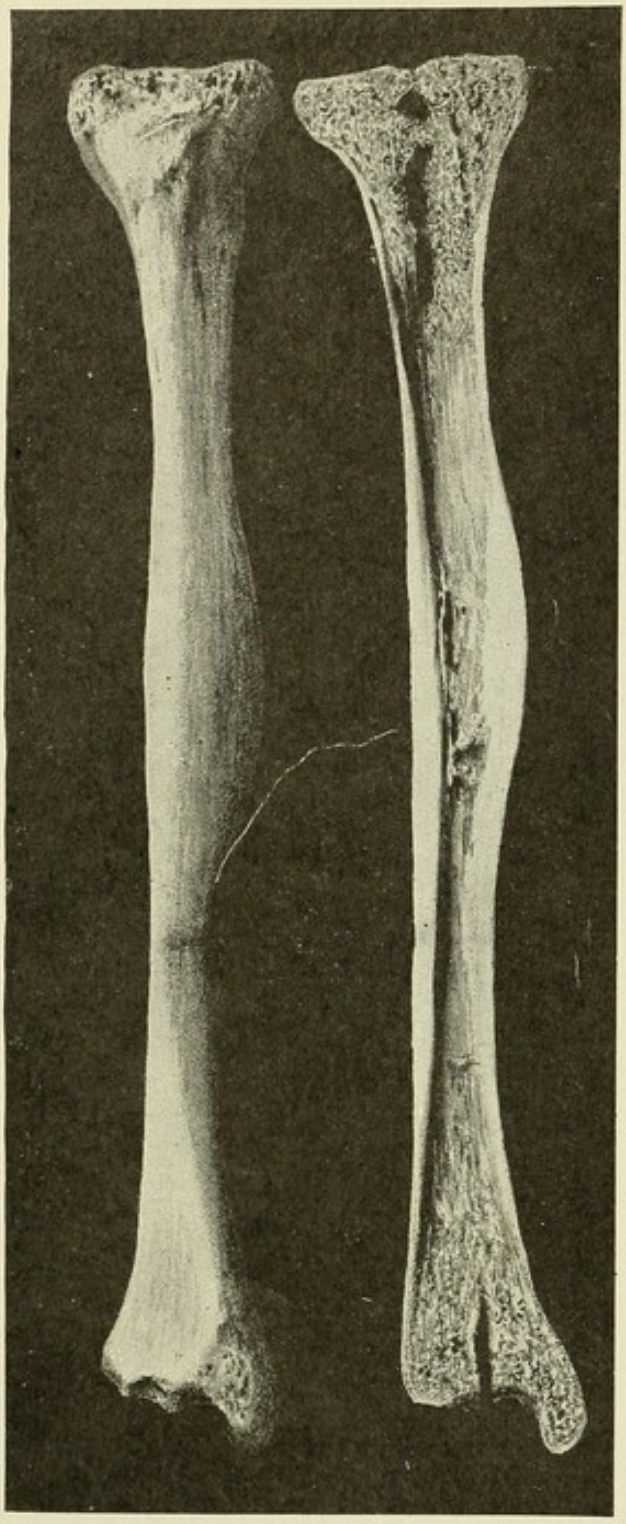

FIG. 55.—TIBIAL NODE OF ACQUIRED SYPHILIS. Compare with Fig. 44. (Townsend, in "Morrow's System.")

or quite sharply localized (Figs. 52 to 55). In my experience, the diffused process is commonest at the inner end of the clavicle, the localized process in the shaft of the tibia.

When, in the course of time or through the effect of treatment, the inflammation diminishes or tends to heal, this tendency shows itself by the production of new bone either around or throughout the region affected by rarefying osteitis. This *productive osteitis* originates both in the periosteum and in the bone tissue proper (Figs. 52, 53). It results in an overgrowth of very dense, eburnated bone tissue, which projects from the surface of the bone in the form of a node [1] (exostosis), and if severe often obliterates,

[1] A very small node is called an osteophyte, a very diffuse node a hyperostosis.

or at least constricts, the medullary canal, as shown by skiagraph.

When the process is diffuse, the intermingling of rarefying and productive osteitis quite destroys the regularity of the bony outline and throws an X-ray shadow characteristic of syphilis.

Prognosis.—The inflammation may be checked (1) in the early stages before any notable change in the bone has occurred, in which case there remains no trace; or (2) in the stage of localized or diffuse osteitis, in which case there remain an exostosis or an irregular hyperostosis to attest its passage; or (3) it may progress, rapidly or slowly, to—

Gummatous Osteoperiostitis.—This process is a gummatous degeneration occurring in an area of rarefying osteitis. When the gumma once begins, it softens rapidy, soon involves the overlying tissues, and, if the bone is near the surface of the body, involves and breaks through the skin, just as does the subcutaneous gumma.

Meanwhile productive osteitis occurs in a cup shape, surrounding the gumma (Fig. 56).

Bone gumma may be single or multiple, and may be associated with diffuse osteoperiostitis.

Prognosis.—The gumma may be cured either (1) at its inception before the superficial tissues have been invaded, in which case there results a depressed area in the bone surrounded by an irregular ridge, but not adherent to the skin; or (2) after the overlying and skin tissues have been involved, in which case the skin (or scar, if the skin has been destroyed by ulceration) is adherent over the depressed bone, and the loss of tissue in the soft parts accentuates the depth of the depression and the height of the surrounding ridge of eburnated new bone.

Osteomyelitis.—Syphilitic osteomyelitis is extremely rare in acquired syphilis. Certain early osteocopic pains are

apparently due to exudative lesions within the bone, but this has not been proven. *Gummatous osteomyelitis* produces the

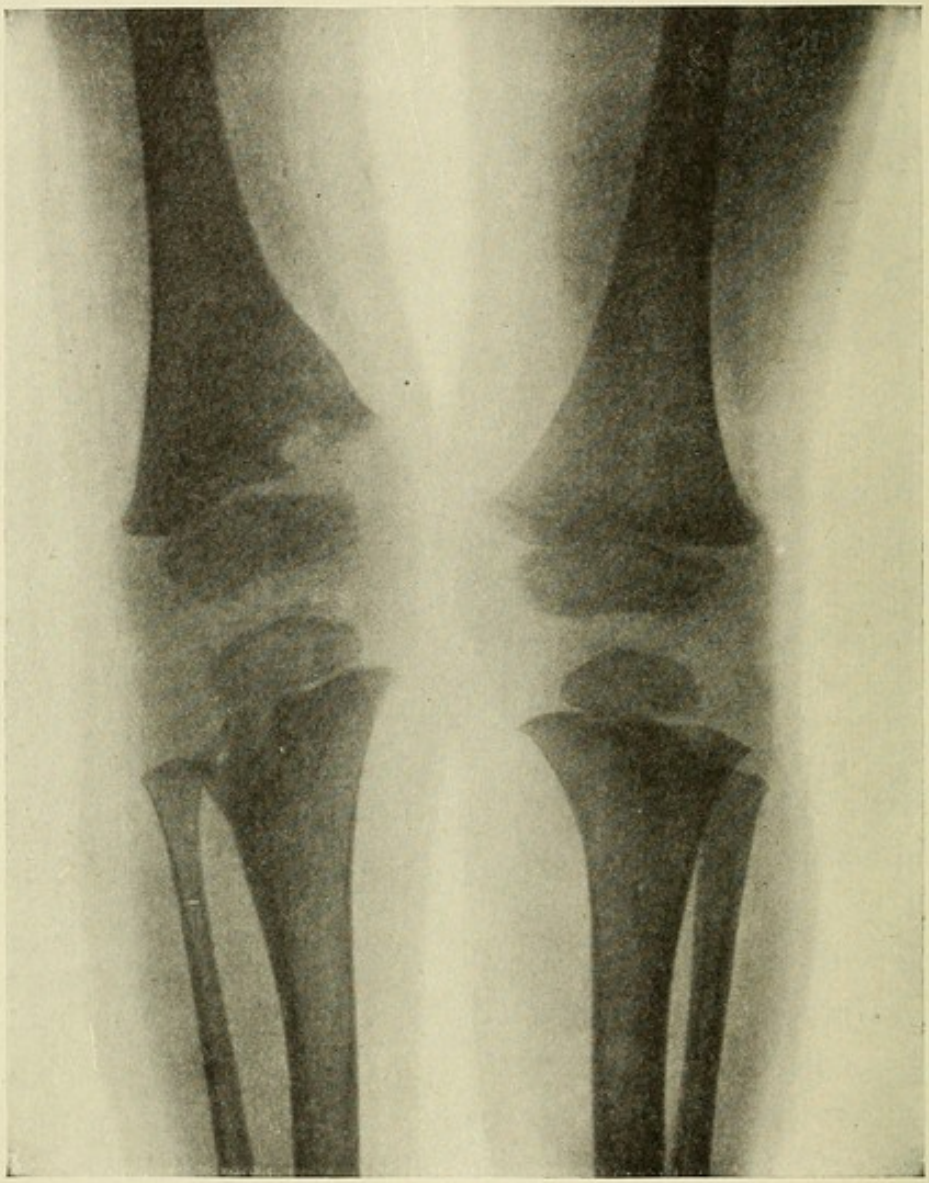

FIG. 56.—GUMMA OF INNER CONDYLE OF FEMUR AND OUTER CONDYLE OF TIBIA (HEREDITARY SYPHILIS): ARTHRITIS. (Compare normal joint.) (Case of Dr. Jaeger. Skiagraph by Dr. Cole.)

same lesions, *mutatis mutandis*, as does gummatous osteo-periostitis. The strength of the bone may be so weakened as to permit " spontaneous " fracture.

SYMPTOMS

Medullary Lesions.—These are so uncommon that their symptoms have not been thoroughly studied, except in reference to hereditary syphilis.

In acquired syphilis they show themselves under one of four clinical types:

1. *Osteocopic pains*, deep-seated, without periosteal swelling or tenderness, and with typical nocturnal exacerbations.

2. *"Spontaneous" fracture* may be the first evidence of medullary syphilis. After the event one may notice absence or diminution of crepitation when the fragments are brought in contact, and later a gummatous tumor enveloping them. If the diagnosis is not made and antisyphilitic treatment promptly instituted, union will not occur and the gumma will extend. Under mixed treatment, however, such fractures heal kindly, though usually with considerable permanent deformity due to absorption of bone.

3. *Non-union of a traumatic fracture* may be caused by syphilis. Of the thousands of syphilitics whose bones are broken, very few show anything abnormal in the process of repair. But, exceptionally, one meets a case of fracture about which there is nothing unusual, except that the patient has had syphilis, and the fracture fails to unite. No large gumma develops at the point of injury; but if the bone ends are cut down upon they are found eburnated and eroded. A skiagraph should show typical rarefaction and eburnation.

4. *The gumma may eat through the bone* and extend from its surface as though it were a periosteal lesion (see below), without causing severe pain or spontaneous fracture.

Periosteal Lesions.—The *onset* of all osteoperiosteal lesions is the same whether the process is to terminate with or without gummatous degeneration.

The onset is marked by pain, tenderness, and swelling.

The *pain* is usually quite severe, with marked nocturnal exacerbations less by day and relieved by exercise. The *tenderness* is localized and marked. The *swelling*, though not always recognizable in the milder lesions, is the best index of prognosis. Marked and rapidly progressive swelling shows active disease with tendency to gummatous change, while slight or stationary swelling shows that central degeneration is not threatening—that the lesion tends to follow the milder productive type.

Following upon such an onset, the disease establishes itself either as a productive osteoperiostitis or a destructive gumma. Besides these two types, there is a "mixed" type due to gummatous degeneration in an area of osteoperiostitis.

Osteoperiostitis.—The lesion as a rule develops very slowly. When once established, this shows itself as a thickening of the bone. It is usually situated near the extremity of a long bone (tibia, clavicle, ulna), and is diffuse, irregular, insensitive, and without subjective symptoms. Such a deformity simulates nothing else than an exuberant callus, following fracture, from which it may be readily distinguished by the history. The patient usually attributes the pain at the onset to some imaginary injury, unless it is so slight as not to have attracted his notice; in which case he states that he has just discovered the bony growth, which, as a matter of fact, has been silently growing for months.

But almost every symptom of such a typical case may vary. The lesion may be situated upon the skull or near the center of a long bone; it may be circumscribed, sensitive, tender, and even accompanied by reddening and edema of the superficial tissues, as in gumma.

Periosteal Gumma.—This lesion usually follows an acute course in marked contrast to the chronicity of productive osteoperiostitis. The onset is usually sharp, exceptionally subacute, with little pain and tenderness.

The *swelling*, which may be obscured by the deep situation of the bone (spine, femur), is at first a flattened, hard, circumscribed, tender swelling attached to the bone. As it grows, it becomes softer; the tissues over it first adhere, then become edematous, and finally involved in the gumma. In its subcutaneous situations (tibia, cranial bones) periosteal gumma passes through precisely the same clinical phases as does subcutaneous gumma (page 327), except that it is from the beginning adherent to and in some measure incorporated in the bone.

When the bone is deep-seated, the gumma usually reaches the stage of softening and considerable involvement of the overlying soft parts before it is diagnosed.

Destruction of the bone by gumma occurs in two ways. In part the bone is actually eaten away and absorbed; in part its circulation is cut off, and the fragment is thrown off as a sequestrum. Hence at the bottom of an ulcerated bone gumma there may be fragments of dead bone. These are usually few and small; large fragments, requiring surgical judgment as to the time and surgical skill as to the manner of their removal, are extremely rare.

A more important result of bone destruction is the complete absorption of a phalanx by a severe *syphilitic dactylitis.* (Fig. 58). Whether the phalanx is absorbed by rarefying osteitis or necrosed and cast off as a sequestrum, the result is the same; with healing the soft parts contract and almost completely obliterate the phalanx, leaving a shortened, distorted finger.

Bone syphilis in children, whether acquired or hereditary, has a peculiar affinity for the epiphyseal cartilages of the long bones and the cranial sutures. The resultant deformities (pseudo-paralysis, shortening or lengthening of the long bones, natiform skull, etc.) are discussed with the lesions of hereditary syphilis (page 518).

Complications.—True suppuration in a syphilitic lesion of bone is very rare, although the syphilitic bone lesion may be so acute as to cause a slight rise of temperature which, with the redness, swelling, and tenderness, may well give the impression of suppuration.

DIAGNOSIS

The nocturnal pains (if present), the characteristic course of the disease, whether productive or gummatous, and the other evidences of syphilis usually establish the diagnosis. In doubtful cases the X-ray confirms or rejects this diagnosis, by showing an area of rarefaction surrounded by an area of condensation or irregularly intermingled spots of the two, typical of syphilis (Figs. 52 to 55).

Differential Diagnosis.—Bone syphilis may be mistaken for suppurative periostitis or osteomyelitis, tuberculosis, neoplasm, and exuberant callus. The X-ray must be used to diagnose obscure cases.

Suppurative Bone Lesions.—Though an active periosteal gumma may show all the local signs of suppuration, there is little or no fever, little leukocytosis, and the characteristic night pain to guide one. If an X-ray is not accessible, exploratory puncture or incision withdraws a sero-sanious fluid immediately suggestive of syphilis, while the boggy fluctuation is undiminished.

Tuberculosis.—Syphilis of joints is often mistaken for tuberculosis, syphilis of bone hardly ever; their clinical characteristics are quite distinct. The lesions of tuberculosis are situated in the epiphysis rather than in the shaft; the pain of tuberculosis does not show *severe* nocturnal exacerbation, and is relieved by traction while that of syphilis is not.

If a large gumma is mistaken for cold abscess, puncture or incision promptly rectifies the error.

NEOPLASM.—The X-ray is by far the best diagnostic aid. Without it one may have to depend upon exploration.

CALLUS.—As already noted, irregular masses of old osteo-periostitis may simulate exuberant callus, a history of injury being imagined to fit the case. History and X-ray establish the diagnosis.

TREATMENT AND PROGNOSIS

Treatment.—The treatment of bone syphilis shows the iodids to the best advantage. It is upon them that we chiefly depend, although it is impossible to state in exact terms how much iodid will be required to control any given lesion. The most active gumma may yield promptly to a very small dose, while a very sluggish and apparently superficial periosteal thickening may require prolonged treatment by high doses. Such obstinate cases should be treated after the rules laid down for the treatment of nervous syphilis by interrupted courses, alternating mixed treatment with hygiene.

The use of mercury in the treatment of bone syphilis sometimes seems superfluous, yet it is surely of great assistance in preventing relapses and in eradicating the last remainder of the syphiloma.

The *local treatment* of bone syphilis is relatively unimportant. The application of a mercurial ointment or plaster to a lesion that has not already ulcerated through the skin somewhat retards its growth; but one should beware lest the ointment irritate the skin and so reduce its vitality as to encourage its destruction by the gumma.

After ulceration has occurred it is necessary to keep the lesion clean, and for this purpose a powder consisting of one part of calomel to two of talcum is useful, though for dispensary patients applications of blue ointment are commonly employed.

If there is considerable cellulitis or any tendency to sup-

puration, these must be combated by the usual wet dressings employed in surgery.

If the ulcer is old or extensive, it should be carefully probed for dead bone; but the surgeon must resist as far as possible, the temptation to remove such dead bone by operation with the idea of hastening healing, for demarcation occurs very slowly, and, if the removal of a semidetached piece of bone is attempted, this may result either in leaving behind bone which will subsequently necrose or in stirring up the lesion to renewed activity. The best plan is to push systemic treatment and not to attempt the removal of sequestra until they are freely movable. The only exception to this rule is the case in which a large sequestrum encourages suppuration, especially if it be in a cranial bone. For such a case prompt operation is indicated for the removal of the sequestrum.[1]

Prognosis.—The prognosis depends almost entirely upon the treatment. Bone lesions, however shocking in appearance, are marvelously amenable to medication with the iodids. But if the lesion is untreated, it is impossible to foretell in the beginning how far it will go or exactly what character it will assume; whether it will be diffuse or circumscribed, productive or destructive.

If treatment is begun at the onset, the lesion may be cured and practically no scar remain. If a productive periosteal lesion has already formed exostosis or hyperostosis before it is controlled by treatment, we may not expect to reduce these deformities any more than to dissolve the bones themselves.

Thus when a patient presents himself with extensive, irregular enlargement of a bone, and examination shows this enlargement to be insensitive while the X-ray reveals it to be composed of dense, eburnated bone, the patient should be put upon a mild course of mixed treatment in order to eliminate

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[1] Voss, *Dermat. Zeitschr.*, 1905, vol. xii.

any possible remaining active process within this bone and to minimize the probability of relapse; but he should not be encouraged to expect from any treatment the reduction of this permanent deformity.

The only stage at which bone syphilis demands immediate heroic treatment is when a gumma is developing rapidly and threatening to soften or to break through the skin. Under such conditions iodid should be pushed rapidly in the hope of preventing the scar which will result if the skin is broken. But, even though such lesions are brought rapidly under control, the unbroken skin may be left thinned and adherent to the underlying bone and leave a scar almost as disfiguring as if ulceration had occurred.

LESIONS OF SPECIAL BONES

The preceding description applies to bone syphilis in general, but the lesions of certain special bones show peculiar characteristics that merit some attention. We may note particularly the lesions of the cranial bones, of the fingers, of the inferior maxilla, of the sternum, and of the vertebral column.

Cranial Bones.—Syphilitic lesions of the cranial bones commonly affect the frontals and the parietals. They are often multiple, are usually gummatous in character, and, as a rule, begin in the periosteum covering the external table, though they may originate in the diploë or upon the internal table.

When projecting externally, these lesions show no very peculiar characteristics except their disfiguring character (great swellings upon the head), and the rapidity with which they eat into the bone, leaving it riddled and worm-eaten, as shown in Fig. 57.

When they project within the cranium, however, whether beginning on the internal surface or in the diploë, or having eaten through the whole bone from the external surface, they

promptly involve the meninges, and thence the brain, causing localized headache and epileptiform convulsions, according to the region of the brain involved.

If the lesion is entirely internal, there may be no external manifestation beyond a certain tenderness to pressure, and later,

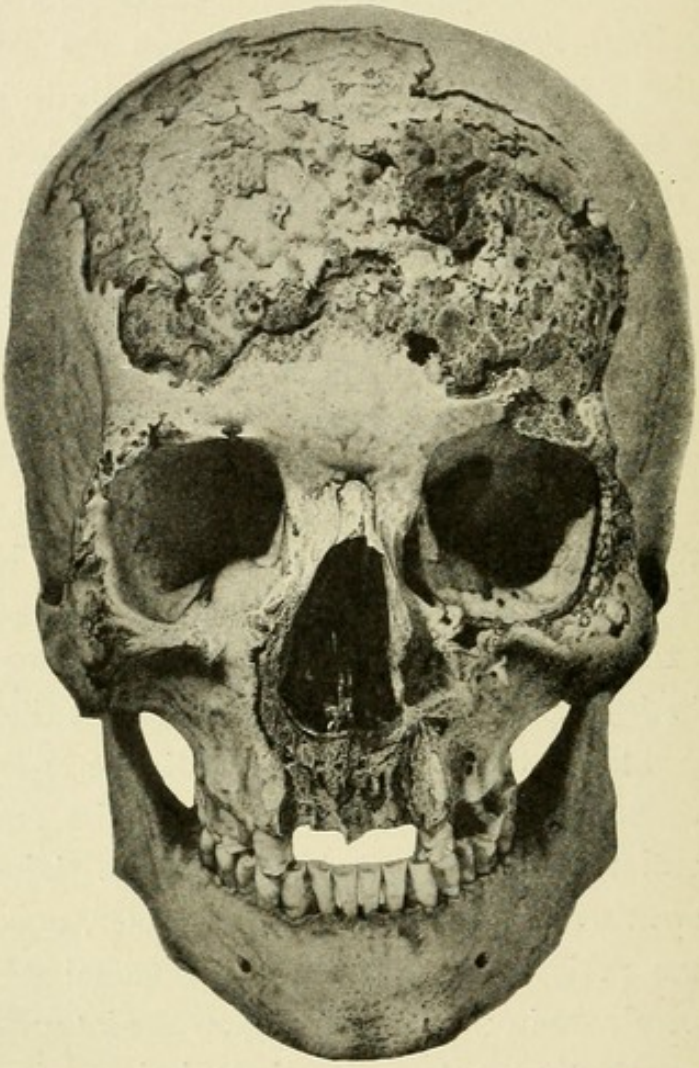

FIG. 57.—EXTENSIVE GUMMATOUS DESTRUCTION OF SKULL. (Lebert.)

as the bone becomes eaten away, a crackling of the outer shell, as this yields under the finger.

If, on the other hand, the bone has been eroded and the skin ulcerated, rapidly fatal, septic meningitis may result, though it is surprising how frequently even these cases resist infection.

Syphilitic Dactylitis.—Syphilis of the phalanges may attack one or more fingers or toes. It is usually single, and attacks the index or middle finger. The proximal phalanx is almost always the one attacked (Fig. 58). The toes are much less frequently affected than the fingers.

As Taylor has shown, the lesions of syphilitic dactylitis may begin in any of the tissues of the fingers, but it usually involves the whole of the phalanx, beginning as a periosteal lesion or as an osteomyelitis. Taylor states that, if the process begins in the soft parts, there is simply a chronic fusiform thickening of all the tissues of the finger, the bone finally becoming

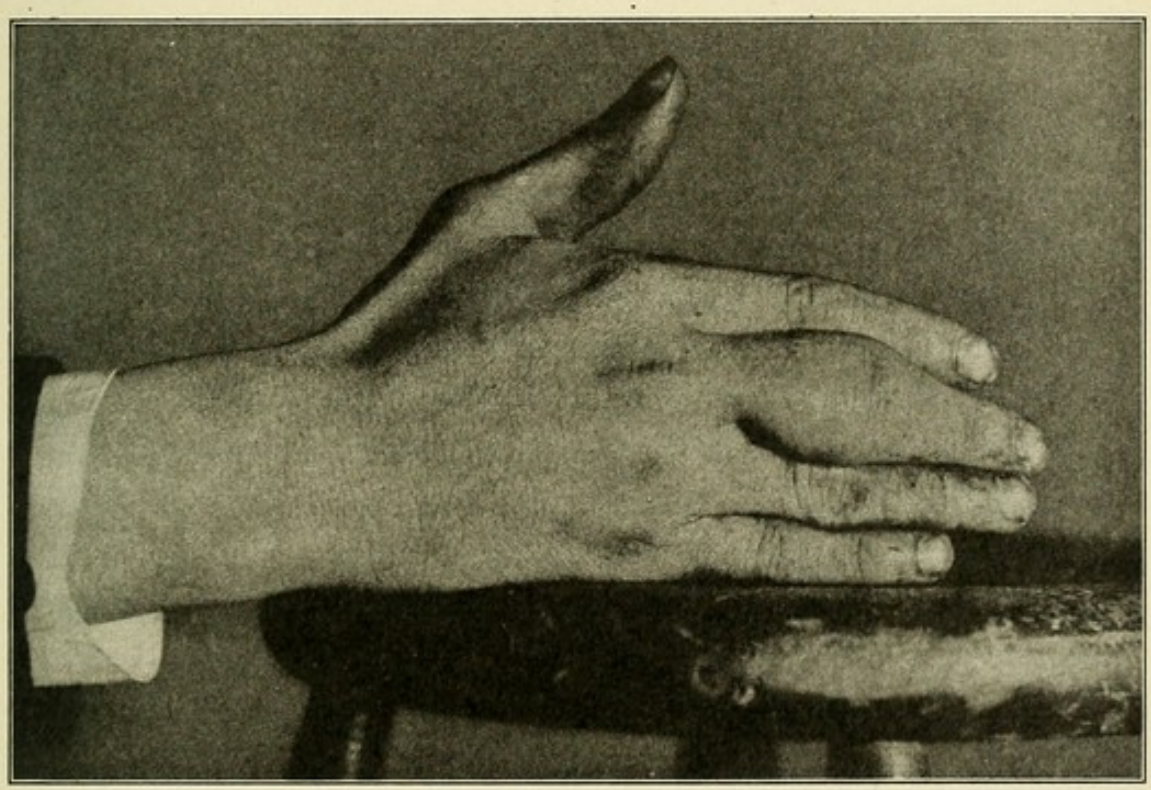

FIG. 58.—SYPHILITIC DACTYLITIS. (Piffard.)

involved; and this involvement of the bone beginning near the metacarpo-phalangeal joint.

In the more common form of dactylitis that begins in the bone, the external appearance is very much the same. The finger is swollen but not reddened, and the process exists a

long time before gummatous softening and ulceration occur. Indeed, in some cases, spontaneous resolution may terminate the inflammation.

Taylor states that the progress of syphilitic myelitis is much more rapidly destructive than that of periostitis.

All of these lesions are much more common in children than in adults, and therefore are usually seen in hereditary rather than in acquired syphilis.

On account of the youth of the patient and the relative painlessness of the inflammation the process is often neglected for many months, and, as a result, the phalanx may be greatly disfigured, either by destruction of the shaft of the bone or by implication of the joints at either extremity. But when seen early, it yields quite as readily as does any other form of bone syphilis.

Inferior Maxilla. —The lower jaw may be affected by osteoperiostitis or by gumma in any or all of its parts. But the common lesion is destructive of a portion of the alveolar border by acute gummatous inflammation. The process is similar to necrosis of the palate and nasal septum.

The lesion begins with a swelling around the root of one or more teeth. These soon become loose, and then in a few days or weeks a portion of the alveolar process is shed and the loosened teeth fall with it. Exceptionally, the inflammation may be checked with loss of little or no bone and with a resultant deformity in the gum, which may not seriously impair the vitality of the adjacent teeth; but, as a rule, even if attacked early, one may expect to lose at least one tooth.

Of the three cases of syphilis of the upper jaw which I have recorded, two were of this same nature, i. e., necrosis of the alveolar process; the third was a gumma in the antrum.

Sternum.—In early syphilis tenderness of the sternum, due to periostitis and excited by tapping upon that bone, is only less common than tenderness of the shins. But the later, more

serious lesions of the sternum are far less common. There may be osteoperiostitis or periosteal gumma, or gumma of the diploë.

The chief and most important peculiarity of these lesions is that when they begin on the internal surface of the bone, or extend thereto, they may invade the anterior mediastinum and cause various symptoms, such as pericardial pain, attacks of dyspnea, asthma, angina, or circulatory disturbances, on account of pressure or of involvement of the pericardium or pleura.

Vertebral Column.—Lesions of the vertebral column are extremely rare. They have been most often recorded in the cervical and in the dorsal region. Most of the cervical lesions are due to gumma of the posterior pharyngeal wall, whether primarily periosteal or submucous.

The lesions are almost always found in the bodies of the vertebra, though syphilis of the transverse and spinous processes has been recorded. The lesion assumes one of the following clinical types:

1. *Pain.*—Localized pain, often accompanied by sensitiveness and with the typical nocturnal exacerbation, is the commonest symptom, and may stand alone for many months.

2. *Syphilitic pseudo-Pott's Disease.*—Syphilis may imitate Pott's disease of the spine very closely, destroying the bodies of one or more vertebræ, causing pain, anchylosis, deformity, secondary cold abscess (gumma) formation, with exudation of fragments of bone and secondary invasion of the cord. Such lesions occur almost exclusively in children, and may be diagnosed from tuberculosis of the spine by the evidences of other syphilitic lesions in the child or in its parents; which evidence is fortified by the beneficial effect of antisyphilitic treatment.

CHAPTER XXIX

SYPHILIS OF JOINTS, MUSCLES, TENDONS AND APONEUROSES, BURSÆ AND TENDON SHEATHS

SYPHILIS OF THE ARTICULATIONS

SYPHILIS simulates every joint disease from rheumatism to tuberculosis. Its lesions may be classified under the following types:

Arthralgia.

Hydrarthrosis.

Pseudo-rheumatism.

Tertiary arthritis and osteoarthritis.

Deforming arthritis.

Syphilitic Arthralgia.—The special characteristics of syphilitic arthralgia are:

1. There is no discoverable lesion sufficient to account for the pain.

2. Nocturnal exacerbation and relief by exercise.

3. Frequent in larger joints (shoulder, knee, elbow), rare in smaller ones; if polyarticular, one joint is usually much more painful than any other.

4. Common with first onset of secondary symptoms; rare later.

5. Unaffected by mercury; promptly relieved by small doses of iodid.

Little need be added to this brief characterization unless it be the statement that the pain varies greatly in intensity and may not show the typical nocturnal exacerbation. Thus I have recently been treating a man who, in the third year of his dis-

ease, and while under systematic mercurial treatment, has persistently complained of slight and indescribable sensations about the left knee. No objective signs of disease could be elicited, and the discomfort continued for fully six months until he was put upon potassium iodid, gr. v, t.i.d. In two days the discomfort disappeared.

Hydrarthrosis.—Syphilitic hydrarthrosis consists of a painless joint effusion. It is quite rare. It is scarcely ever seen except in the knee. If slight, the patient may overlook it; even if marked, it is rapidly absorbed under iodic medication.

The absence of trauma and of all sensitiveness differentiates it from the ordinary traumatic hydrarthrosis. Moreover, it occurs almost always in the first months of syphilis and in connection with other lesions of the disease.

Syphilitic Pseudo-rheumatism.—"Secondary pseudo-rheumatic arthropathy" (Fournier) is extremely rare. It simulates acute articular rheumatism in its local symptoms (heat, swelling, pain, tenderness, redness), but it usually strikes only one joint, never more than two or three. The knee is the joint most often affected, the wrist, elbow, and ankle less frequently.

The notable distinction between syphilitic and true rheumatism consists in the absence of systemic disturbance or of generalization in the former. There is never much fever, usually none; no sweats; no concentrated urine; no cardiac implication. Moreover, the syphilitic lesion is an early one and yields rapidly to iodids.

Tertiary Syphilitic Arthritis.—Tertiary syphilitic arthritis is much more common than the secondary hydrops and rheumatism just described. My records show four cases of secondary hydrarthrosis of the knee during the first year and one in knee and one in elbow during the second, while the tertiary lesions are distributed as follows:[1]

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[1] The numbers in parentheses indicate numbers of cases when more than one.

Knee, 14 cases at one, four (2), five, six, seven (2), eleven, twenty-six, thirty-three years.

Elbow, 5 cases at four (2), five, six, fourteen years.

Shoulder, 2 cases at seven, twelve years.

Ankle, 2 cases at four, seven years.

In one case both knees were involved at seven years; in one ankle and elbow were involved at four years; in one elbow and knee at six years.

Pathology.—Tertiary syphilis of the joints usually originates in the capsule or ligaments, less often in the bones, rarely in the synovial membrane or subserous tissue.

In *the synovial membrane* the lesions are congestion and the production of warty excrescences, especially in the folds and pockets. The warty growths may become fibrous and form "joint mice" if neglected. There are subserous infiltrations, and perhaps gummata, which open into the joint. There is always considerable effusion. Long-neglected cases result in a stiff joint from adhesions of the inflamed serosa.

The cartilages are often the seat of gummata, which soften, discharge, and finally heal, leaving rounded, depressed scars quite characteristic of syphilis.

In *the capsule* there is general thickening with circumscribed areas of hard, fibrous infiltration. These are spoken of as gummata, but they rarely break down.

The bone involvement is usually slight. In five of the syphilitic knees there was marked involvement of the condyles of the tibia, in one the femur was implicated. The lesions are the usual osteoperiostitis or gumma (Fig. 56); a slight degree of the former is present in all severe cases.

Symptoms.—There is painless, usually gradual, enlargement of the joint due in some cases almost entirely to hydrarthrosis, in most to the periarticular thickening. The joint is merely stiff and clumsy. Examination reveals the thicken-

ing of the capsule with nodules in it, a certain amount of effusion, and perhaps some thickening of the ends of the bones.

If the papillary fringes get between the articular surfaces the familiar symptoms of the " joint mouse " result, i. e., sudden, excruciating pain followed by more or less acute synovitis.

The progress of the affection is extremely slow. If neglected it terminates in partial or complete fibrous ankylosis. Frauenthal [1] states that in neglected cases there is danger of secondary tubercular or pyogenic infection.

Diagnosis.—Syphilis of the knee-joint is often confounded with tuberculosis. The diagnosis can usually be established by the X-ray; but lacking this it must rest upon the absence, in syphilis, of redness, heat, fistulæ, and tenderness; the presence of other lesions or scars of syphilis and the effect of antisyphilitic medication.

Treatment.—While rest is advisable, it is surprising how well clinic cases do without it. The iodids are our chief reliance; but it is essential that repeated courses of treatment be given for months after the lesion is apparently cured; otherwise relapse may be expected.

Deforming Arthritis.—Trophic changes in the joints, especially the knee, occur in tabes, less often in hereditary syphilis. The lesion is only indirectly due to syphilis, and has no peculiarly syphilitic characteristics.

SYPHILIS OF THE MUSCLES

Apart from the muscle pains that may occur in early syphilis (page 263), syphilis of muscles is extremely rare. I have record of only two cases, one of torticollis (sterno-mastoid spasm), one of bicipital contracture.

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[1] *Med. Record*, 1906, May 26.

The lesions described are:

Contracture.

Interstitial myositis.

Gummatous myositis.

Myositis ossificans.

Syphilitic Contracture.—This singular condition consists in a gradual or sudden contracture of a muscle (usually the brachial biceps or triceps). It is painless so long as no attempt is made to overcome the contracture. It occurs almost exclusively in the first year of the disease. Examination reveals the body of the muscle to be in a semiflaccid condition, its tendon on the stretch, and the joint to which it is attached absolutely fixed by the muscular contracture. Any attempt to overcome this excites the most vivid pain, and is futile.

The progress of this condition is extremely chronic. It may get well spontaneously, but even under treatment it resolves very slowly. The ultimate outcome, however, is a perfect restoration of function.

The *treatment* is by mercury.

Interstitial Myositis.—Extremely rare. The condition is a diffuse infiltration beginning in the connective tissue between the bundles of muscle tissue and involving them secondarily. The greater part of a muscle is usually involved. There may be gumma. The muscle is swollen, dense, contractured, and slightly sensitive. The skin may be a little red, but it is not adherent nor is there subcutaneous edema.

Gumma.—The first sign of gumma in a muscle is slight subjective pain on motion in a spot which examination reveals to be a sensitive lump quite like an induration of chronic myositis.

But this lump grows and becomes a definite, hard tumor within the muscle. It may attain the size of an orange. There may be several gummata in a single muscle. Together with the gumma there may be interstitial myositis (sclero-gummatous type).

Finally it softens, invades the surrounding tissues, and bursts through the skin like a subcutaneous gumma. Exceptionally, instead of softening, it becomes sclerosed or ossified.

The muscle most frequently affected is the sterno-mastoid.

Myositis Ossificans.—Myositis ossificans may develop in a patient who has had syphilis, and, very exceptionally, an interstitial or gummatous myositis degenerates into a bony growth. But myositis ossificans is scarcely more frequent from syphilis than from any other sort of irritation, and postsyphilitic myositis ossificans does not differ in symptoms or treatment from that due to other causes; antisyphilitic treatment has no effect upon it.[1]

SYPHILIS OF TENDONS AND APONEUROSES

Tendons and aponeuroses are scarcely ever involved except by extension of a syphiloma from the surrounding parts. The occipito-frontal aponeurosis is said sometimes to be inflamed in early syphilis. I have record of one case of gumma of the tendo-Achillis in a patient with many other tertiary lesions.

SYPHILIS OF BURSÆ

Acute bursitis or chronic bursitis with effusion may complicate arthritis. It is extremely rare.

Gummatous bursitis is far more common. I have record of five cases, two of which were published in my father's monograph.[2] Including the cases therein collected by him there are

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[1] Perhaps some confusion has been injected into this subject by the fact that dense syphilitic myositis of the masseter feels very like bone, produces the same contraction as would myositis ossificans, and yet yields to antisyphilitic treatment.

[2] *Am. J. Med. Sci.*, 1876, p. 349.

6 double (about the knees), 13 single: 10 occurred in women, 9 in men. Three occurred in the second year, 4 between the fifth and eighth, and 1 each at fifteen and twenty-eight years

FIG. 59.—SYPHILITIC PREPATELLAR BURSITIS WITH INVOLVEMENT OF THE OVERLYING SKIN. (Case of Dr. Keyes. Photograph by Dr. Fox.)

after the chancre. In 14 cases the bursæ about the knee were affected, usually the prepatellar (Fig. 59).

The involvement of the bursæ is almost always primary. It causes a considerable boggy swelling (below the patella), over which the skin reddens, thickens into great ridges, and finally ulcerates.

The cure by iodid is rapid.

SYPHILIS OF THE TENDON SHEATHS

Fournier believes that many of the pains about the knees and elbow at the outbreak of secondary symptoms are due to tenosynovitis in the neighborhood of these joints. Acute pseudo-inflammatory tenosynovitis (with tenderness, edema) and chronic hydrops are rare, but less so than the parallel types of bursitis.

Gummatous tenosynovitis resembles gummatous bursitis, but is even less common (I have record of but two cases).

CHAPTER XXX

SYPHILIS OF THE AIR PASSAGES[1]

SYPHILIS OF THE LARYNX

THE larynx is not often sufficiently affected in early syphilis to produce symptoms,[2] though laryngoscopic examination reveals slight secondary lesions in many patients whose throats give them no inconvenience. Tertiary lesions, however, are fairly frequent and quite distressing.

Apart from the laryngeal paralyses (page 372), I have record of 34 cases of marked or prolonged syphilitic aphonia. Twenty of them were about equally distributed in onset between the first and the eighth year, 3 began in the twelfth, and 1 each in the eleventh, seventeenth, twenty-first, twenty-fourth, and thirtieth years of the disease.

SECONDARY LESIONS

The *causes* of secondary laryngeal syphilis are abuse of alcohol or tobacco or overuse of the voice.

Hence it is much more common among men than among women, among singers and public speakers than among those who simply sit and listen.

The secondary lesions are:

Erythema.

Mucous erosions and papules.

Condylomata.

Diffuse infiltration.

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[1] Reviewed by Dr. L. M. Hurd.

[2] Two cases of laryngeal chancre have been reported.

The specific characteristics of these is that of the same lesions occurring in the mouth (page 336).

Laryngeal erythema is much the most common. It usually occurs with the secondary outbreak (or within a few months thereafter). It may be diffuse (and mimic acute non-specific laryngitis) or mottled. The inflammation habitually involves the epiglottis, the false and the true vocal cords. A mottled erythema of these regions is almost pathognomonic of syphilis; it is the laryngeal roseola. But, contrary to what one might expect, this roseola is very persistent, and subsides spontaneously only after many weeks. This mottled erythema is much less common than the diffuse congestion.

Either with or without this general erythema, *erosions* or superficial ulcerations may appear in the larynx. Their seat of election is the true vocal cords. They are often multiple, and appear as glistening or opalescent lesions, set in an area of congestion, occupying a part or the whole of the cord.

Laryngeal papules, whether simple or eroded, are rare.

Condylomata are equally rare.

Diffuse infiltration of the larynx is one of those lesions that might be termed intermediate between secondary and tertiary syphilis.[1] It consists of a more or less diffuse redness and swelling usually confined to the upper portions of the larynx (but which may invade the true cords) and usually unilateral, or at least affecting one side of the larynx more than the other. The region involved (epiglottis, false cords, etc.) loses its sharp contour, and appears reddened, thickened, and bulging. The swelling is usually smooth, but may be broken by little pseudo-papules.

This lesion, which usually occurs after the first outbreak and may appear several years after the chancre, is slow to develop and, even under treatment, extremely slow to disap-

[1] Ulcerative laryngitis is another such. I prefer to call it tertiary.

pear. It almost always lasts more than a month, and may, if neglected, lead to permanent thickening and aphonia.

Symptoms.—*Aphonia is the cardinal symptom of laryngeal syphilis.* The vocal disturbance due to secondary lesions may be but a slight huskiness or a complete aphonia, accompanied by a dry, irritative cough and slight pain on deglutition (if the epiglottis is considerably involved).

These symptoms differ from those of acute non-specific laryngitis only by their chronicity. The hoarseness or aphonia does not get well in a few days, and this brings the patient to his physician.

Diagnosis.—The persistent hoarseness, combined with symptoms or history of recent infection with syphilis, suggests the diagnosis, which the laryngoscope verifies.

Treatment.—Since the hoarseness is likely to last several weeks at best, several months at worst, the treatment should be vigorous. Mercury effects a cure, and it is usually best to administer it by injection. Iodids and local treatment are useless.

Tertiary Lesions

The tertiary lesions of laryngeal syphilis are:

Ulceration.
Gumma.
Fibroid degeneration.
Perichondritis.
Paralysis.

Ulcerative Laryngitis.—This lesion resembles the ecthymatous syphilid; it may be secondary or tertiary, may or may not destroy tissue, may or may not require iodids for a cure.

Superficial syphilitic ulceration consists of one or more rounded or ovoidal clean-cut (but not punched-out) superficial ulcers. They occur most commonly upon the true cords, less often on the false cords or in the interarytenoid space (Whis-

tler). If numerous and situated upon the cords they may excite edema of the glottis. This I have not seen; but Fournier says he has several times known it to require tracheotomy.

When the ulcers heal, they usually leave permanent superficial scars.

Deep syphilitic ulceration, more common than the superficial type, is usually a malignant, destructive lesion. The ulcer is deep, punched out, single, or multiple, commonest upon the epiglottis and false cords. If neglected it spreads superficially, at the same time eating into the subjacent tissues. Perichondritis or edema of the glottis, acute or chronic, may supervene unless the case is brought promptly under control.

The adhesions resulting from the cicatrization of large ulcers are as characteristic as those that occur about the soft palate under similar circumstances. The epiglottis is adherent to the base of the tongue or to some part of the pharynx, while from below there are adhesions binding it to the false vocal cords and adjacent regions. Dislocation of various parts of the larynx may result from cicatricial contraction, or the whole organ may be stenosed; a web of adhesions may unite the vocal cords.

Gumma of the Larynx.—Gumma of the larynx is extremely rare unless one regards all deep syphilitic ulcerations as originating in unseen gummata.

Gumma is almost always circumscribed, single, and situated in the upper part of the larynx. It appears at first as a red nodule, which becomes yellow as it softens, then breaks and follows the course of deep syphilitic ulceration of the larynx.

Fibroid Degeneration.—Under this title J. N. Mackenzie [1] describes " a decided tendency to the gradual development of fibroid tissue in the structure of the larynx, which tends to diminish the lumen of the organ, not only by contraction of the new-formed tissue, but also by the production of large, dense,

[1] Morrow's "System," 1893, vol. ii, p. 342.

fibroid tumors, which are often mistaken for and described as gummy tumors, but which pathologically have nothing in common with them. These fibroid tumors appear as hard, nodular masses occupying the epiglottis, aryepiglottic folds, and other portions of the vestibule and subglottic region. Sometimes the greater portion of the organ is converted into a dense hypertrophic mass. Acute ulceration occurs, and is fraught with great danger from accompanying edema, and each succeeding attack of ulceration favors a greater deposit of fibrous tissue, and increases proportionately the gravity of the case. In this variety of laryngeal syphilis, which Whistler has especially insisted upon, no retrograde metamorphosis takes place; its processes are essentially progressive, and the caliber of the larynx becomes diminished sooner or later by an irregular nodular mass—half-hypertrophied tissue, half-cicatricial bands—which does not subside under internal or local treatment, and which, if extensive, demands tracheotomy.

"These fibroid tumors may be differentiated from gummata by their pale, grayish or whitish appearance, by the surrounding anemia of the mucous membrane, and by the absence of the peculiar yellowish submucous discoloration of the latter. The hard, dense sensation communicated to the probe contrasts forcibly, too, with the soft elastic feel of the gummy growth.

"This class of case is only seen in hospital or dispensary practice, and presents a long history of neglected laryngeal trouble with gradually increasing obstruction to respiration."

PERICHONDRITIS.—Perichondritis (usually of the cricoid or thyroid) may be primary, but habitually results from neglect of a syphilitic ulcer or gumma.

The onset is slow and obscure with symptoms of moderate aphonia and hoarseness and a diffuse, red swelling (or ulcer) over the cartilage affected. But as soon as necrosis of the cartilage begins, the suppuration about it causes acute edema, which may require intubation or tracheotomy.

Even if promptly taken in hand, perichondritis usually results in sequestration of part at least of the cartilage affected. This implies prolonged suppuration (possibly pyemia or septic pneumonia) and detachment of the necrosed fragment. This fragment is usually coughed up, but may remain caught in the larynx with fatal results, as in the case of Labbé:[1]

"The patient entered La Charité with laryngeal syphilis. On the sixth night after admission he suddenly died of suffocation before he could be relieved. Autopsy showed that the right arytenoid cartilage had fallen into the glottis, obstructing it completely."

Laryngeal Paralysis.—Laryngeal paralysis may be due to central lesion, to neuritis, or to pressure upon the nerve by enlarged glands. The lesion is doubtless usually a central one, though it is not often associated with other evidences of cerebral syphilis (page 372).

Paralysis of the larynx due to syphilis offers no pathognomonic features save its prompt reaction to mixed treatment. We need not, therefore, delay to describe it. It is imitated by ankylosis of the arytenoids.

Symptoms.—Tertiary laryngeal syphilis (paralysis apart) produces *aphonia* chronic in character, variable in intensity. This is *the classical syphilitic vox rauca.* To it may be added *pain*—in deglutition if the epiglottis is involved, in any effort to use the voice if there is perichondritis of cricoid or thyroid. This pain may be very intense, and has been known even to prohibit speech, and so to discourage swallowing that the patient would rather starve than suffer the pain of deglutition. Yet in the great majority of cases the pain is absent or negligible. Indeed, the classical *vox rauca* of syphilis is a hoarseness lasting for months or years without any pain whatever.

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[1] Quoted by Castex in Fournier's "Traité de la syphilis," 1906, vol. ii, Part II, p. 677.

Cough, expectoration, and *foul breath* are evidence of ulceration. *Asphyxiation* may be sudden by edema of the glottis or by impaction of a sequestrated cartilage, or it may be a gradual obstruction due to the projection of the diseased tissue into the larynx or to adhesions or cicatricial contractions forming as the active lesions heal.

Diagnosis.—*Secondary Syphilis.*—The secondary lesions of syphilis can scarcely be confused with anything except acute laryngitis. From this the duration of the congestion, if not its laryngoscopic peculiarities, soon differentiate it.

Tertiary Syphilis.—The lesion must be distinguished from tuberculosis and from malignant neoplasm.

The diagnosis, though usually easy enough, may be extremely difficult. Schnitzler, Luc, and Fournier maintain the possibility of tuberculosis in a syphilitic larynx.

The following table contrasts the chief distinguishing features of syphilis, tuberculosis, and malignant neoplasm:

SYPHILIS	TUBERCULOSIS	NEOPLASM
Number of Lesions.—Usually single.	Usually multiple.	Single.
Usual Situation.—Epiglottis.	Vocal cords.	False cords.
Hyperplasia.—Diffuse, dry, dark red or yellowish.	Moist, granular, pink, bathed in mucus; perhaps covered with ulcers or with inflamed glandular orifices, or with yellowish spots.	Dark, bleeding, vegetating.
Ulcers.—Deep, sloughing, eroding, perhaps disclosing bare cartilage; Usually single.	Superficial, granulating, multiple.	Rare; sloughing. Manifestly subordinate to the neoplasm.
Surrounding Tissue.—Red.	Very pale.	
Associated Lesions.—Syphilitic.	Tubercular.	
Pain.—Rare.	Usual.	Usual.
Pathognomonic Test.—Treatment.	Tubercle bacilli in sputum.	Biopsy.

While the chronic *vox rauca syphilitica*, lasting for years and unaccompanied by cough, pain, or expectoration, is very suggestive of syphilis, it is not absolutely pathognomonic.

Prognosis.—The prognosis of laryngeal syphilis is none too good. Even the secondary lesions may permanently impair the voice, especially in its singing quality.

To this danger the tertiary lesions add that of relapse (which is relatively frequent), acute edema of the glottis, deep cellulitis of the neck from mixed infection, chronic obstruction to respiration by adhesions or cicatricial contraction, if not by the growth itself, and possible acute suffocation by a detached sequestrum, or secondary inoculation with tuberculosis.

Treatment.—The general treatment should be vigorous, preferably by intramuscular injections of mercury and (for the tertiary lesions) large doses of iodids.

" The *local treatment* of the diffused laryngitis of secondary syphilis does not differ materially from that of simple, catarrhal laryngitis. Should ulceration occur, iodoform may be freely used. In the deeper form of ulceration this drug is of inestimable service, and is, in the writer's experience, superior to iodin and the nitrate of silver. Sprays of the bichlorid of mercury, or the local application of the yellow oxid in cosmolin, vaselin, or like substance, are also of considerable value. Before applying these remedies the ulcerated surface should be thoroughly cleansed by means of a detergent and disinfectant spray, for otherwise much of the good effect will be lost.

" Papillomatous growths may be dissipated by the local application of alcohol or chromic acid, or, if extensive, may be removed at once with the forceps. Membranous webs may be successfully divided with the galvano-cautery (Elsberg) or by cutting dilators, the best of which is that devised by Whistler.

" Whether any good can be accomplished by the division of adhesions must be determined by the peculiarities of the indi-

vidual case. Except when function can be restored, or serious dyspnea or dysphagia mitigated by the operation, it is better, as a rule, to let them severely alone.

"Serious interference with respiration from any complication calls for tracheotomy, and the early performance of the latter is especially to be advised when the larynx has undergone the fibroid degeneration described above. Systematic dilatation of the larynx is sometimes of value, but, as a rule, little can be expected of this line of treatment beyond temporary improvement, while it is much inferior to the cutting operation.

"Loosened necrotic plates of cartilage, in view of the dangerous complications to which they may give rise, should be removed, if practicable, by endolaryngeal operation or from without by exsection" (Mackenzie).

SYPHILIS OF TRACHEA, BRONCHI, AND LUNGS

Syphilis of the trachea and bronchi is associated clinically, on the one hand, with laryngeal syphilis, on the other with lung syphilis; but bronchial or peribronchial syphilitic lesions are entirely distinct from those beginning in the lung tissue.

But all of these lesions are extremely rare. (I have record of two cases of tracheal syphilis at fourteen and twenty-two years after chancre; one case of probable pulmonary syphilis at fifteen years.) Moreover, the clinical picture of disease of the lower respiratory organs due to syphilis is better considered as a whole, since, in many instances, lesions of one part of the respiratory tract overshadow equally important changes in another part; while in many cases it is impossible to prove during life that the lesion is syphilitic or to identify its exact site and character.

Tracheal and Bronchial Lesions.—Secondary roseola, catarrh, and mucous patches have been observed in the trachea,

and doubtless may occur in the bronchi as well. Clinically, they are but the extension of similar lesions in the larynx; they excite no characteristic symptoms and have no clinical importance.

Of the tertiary tracheal lesions, the majority occur in the lower third and extend into the bronchi. A certain minority occur at the upper third in connection with laryngeal disease, and but one or two instances of tertiary ulceration of the middle third (Mackenzie) have been noted.

These tertiary lesions are almost always diffuse infiltrations in and about the trachea with surface ulceration irregular in extent and depth. Individual cases of circumscribed tracheal gumma (Hanzel) and of diffuse, non-ulcerated, tracheal gumma (Raymond) have been recorded.

Diffuse, Ulcerated Syphiloma.—This, the type lesion of tracheo-bronchial syphilis, occurs almost exclusively in old, neglected cases of syphilis. The infiltration involves the whole wall of the canal, and is often very extensive. The ulcerations are usually multiple, circinate, and sharp-edged, like syphilitic ulcers elsewhere.

The lesion progresses slowly, and the ulceration may remain superficial for many months. The results of neglected ulcer, however, are (1) necrosis of the cartilages, (2) secondary infection of the deep structures of the neck, and (3) extension of the syphilitic process to the adjacent lymph nodes (syphilitic peribronchial adenitis), a very rare process, causing pressure either upon the bronchi themselves, or upon the great vessels, or the recurrent laryngeal nerves.

Treatment.—Mixed treatment must be vigorously administered. For the low lesions no local treatment is possible; for the high ones tracheotomy, and perhaps intubation with a specially long tube (Hurd [1]) may be required.

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[1] *The Laryngoscope*, 1905, February.

Pulmonary Lesions.—So rare and obscure is pulmonary syphilis that its very existence was doubted a generation ago. Since then congenital syphilis of the lung has been well defined, thanks chiefly to the work of Virchow, Cornil, Balzer and Grandhomme,[1] and Kokawa.[2] But the pulmonary lesions of acquired syphilis are so rare, simulate so closely those of tuberculosis, and occur usually at so late a date in the disease that their very existence has only recently been assured, and it is still far from clear how frequent they are or what may be the predominant clinical or pathological type of the disease.

The following summary of the lesions of pulmonary syphilis, hereditary and acquired, is founded chiefly upon the contributions of Bériel [3] and Milian.[4]

The essential lesion of pulmonary syphilis is an interstitial pneumonia, probably always beginning as a perivascular, exudative process. This process usually terminates in sclerosis, rarely in the formation of gumma. It is liable to the complications of mixed infection, and often produces dilatation of the bronchi. Indeed, Tripier and Bériel contend that most cases of bronchiectasis are due to unsuspected previous pulmonary syphilis.

Syphilitic Pneumonia.—The interstitial pneumonia caused by syphilis is seen as an uncomplicated process almost exclusively in cases of hereditary syphilis. This is the *white pneumonia of the newborn*, the first description of which is commonly attributed to Virchow.

The striking characteristics of this condition are thus described by that author:

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[1] *Rev. mens. d. mal. de l'enfance*, 1886, November, p. 485.

[2] *Arch. f. Derm. u. Syph.*, 1906, vol. lxxviii, pp. 69 and 319. Many other names might be mentioned but the two contributions referred to contain all that is material.

[3] "Syphilis des poumons," Paris, 1907.

[4] In Fournier's "Traité de la syphilis," 1906, vol. ii, Part II, p. 710.

"The lungs appeared so distended and light that the examiners believed they had to do with lungs that had already breathed. But what was their astonishment to find that fragments of this tissue sank in water. A more attentive examination showed that, in reality, there was not a bubble of air in the lungs; on the contrary, the cut section had rather the aspect of a pneumonia in the condition of white hepatization. Yet it was possible to insufflate the lungs."

Bériel recognizes three histological types of this pneumonia.

In the *adenomatous type* "the lesions are essentially a considerable thickening of the stroma and a peculiar condition of

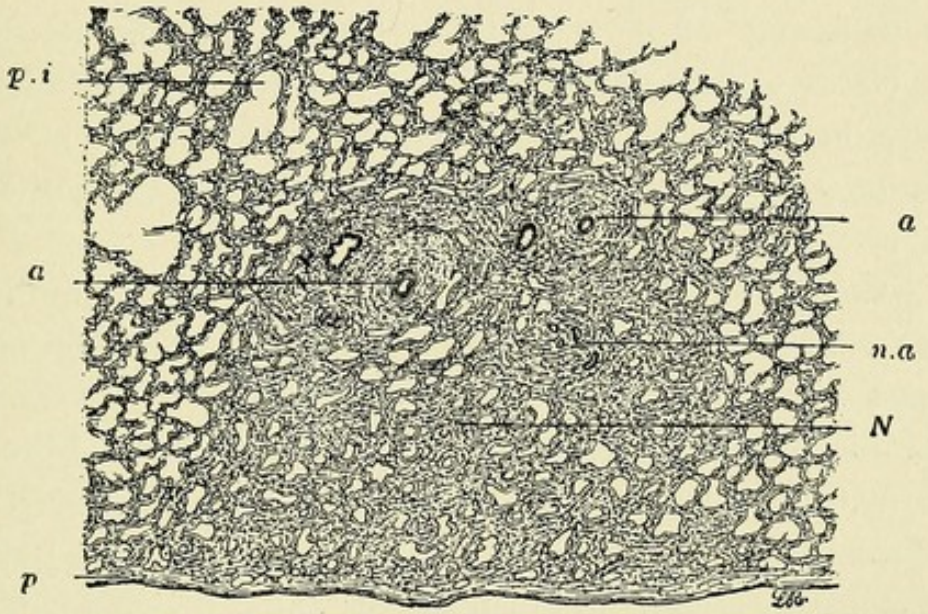

FIG. 60.—HEREDITARY SYPHILIS OF THE LUNG: AREA OF WHITE PNEUMONIA. *N*, nodule of interstitial pneumonia. *n. a*, adenomatous neoformation. *a*, periarteritis. *p. i*, lesser degree of interstitial pneumonia surrounding the nodules, with here and there a dilated alveolus. *p*, pleura. (Bériel.)

the epithelium, the cells of which become more or less cuboidal, so that the tissue presents a striking, adenomatous appearance. . . . It is rich in vessels and generally infiltrated with many cells. . . . In the alveoli are found a greater or less number of exudated cells. . . . The bronchi show the same infiltration, but are little affected, and their epithelium is intact. The ves-

sels may show a slight degree of endarteritis, but there is always a great thickening of the adventitia" (Fig. 60).

The second, or *desquamative, type* is that described by Virchow. The desquamation of cells into the alveoli is marked; the infiltration of the stroma is slight.

The third, or *interstitial pneumonia, type* shows considerable interstitial, subpleural, peribronchial and perivascular infiltration. There is here also a considerable desquamation. There is not the peculiar epithelial change seen in the adenomatous type.

These three types are habitually more or less intermingled in a given case, and are diffused over large areas if the patient is stillborn. If the infant has breathed the hepatization is lobular. Comparable lesions are met with in the adult, but only in small, irregular areas, and the typical picture of white, syphilitic pneumonia is exclusively seen in the fetus or infant.

Cicatricial Lesions.—Syphilitic sclerosis of the lung is spoken of as "soft" sclerosis or "hard" sclerosis, depending upon the density of the lesions, which density is, generally speaking, dependent upon the age of the process and the transformation of the exudate into organized fibrous tissue. This sclerosis is the common pulmonary lesion of acquired syphilis. It is usually irregular in distribution, and is believed to be more common in the lower than in the upper lobe, in the right than in the left lung.

Syphilitic sclerosis has no very peculiar characteristics. The tissue is hard, grayish white, or blackish, and very retractile. It may appear in radiating bands from a given central point or in nodular masses, often beneath the pleura; or, again, there may be a fibrous peribronchitis; but this condition is secondary to infiltration of the bronchi, and is only accidentally, as it were, a pulmonary lesion.

Microscopic examination of this tissue shows no peculiar-

ity, unless it be at its edges certain newly formed alveoli with cuboidal epithelium. These are considered by Tripier and Bériel as almost pathognomonic of syphilis.

Bronchiectasis.—Associated with the interstitial syphilitic changes in the adult lung, just described, one almost always finds bronchial dilatation of various shapes and sizes. In many instances the bronchiectasis is so marked as to be the chief lesion and produces cavities of considerable size, the presence of which can be determined during life. On section these cavities are often found full of pus, and may even be gangrenous; they therefore appear to be necrotic in type and similar to the cavities caused by tuberculosis. But histological examination shows an epithelial lining at least in some parts of the cavity; whence Tripier and Bériel infer that the cavities found in the syphilitic lung are always bronchiectatic, and ascribe them to the adenomatous tendency of syphilitic pneumonia already described.

Complications.—*Rupture of a gumma* into a bronchus is one of the classical features of pulmonary syphilis. But, although many cases of ulcerated or gangrenous bronchiectasis provoke a foul or putrid expectoration, the case reported by Jacquin is probably the only proven instance of ruptured gumma on record.

Acute processes due to *mixed infection*, and showing themselves as intercurrent bronchitis, or broncho-pneumonia, or inflammation or gangrene in a bronchiectatic cavity, are, on the other hand, important features of pulmonary syphilis.

Mixed infection by tuberculosis is alleged to be frequent; but so many of the classical cases of syphilis of the lung are manifestly nothing other than irregular tuberculosis, that the frequency with which the two diseases occur together has probably been overestimated. There is no reason why the syphilitic lung should not become tuberculous, however, and a few authentic instances of this mixed infection are recorded.

Symptoms of Tracheo-Bronchial Syphilis.—Although some authors recognize an *acute secondary bronchitis*, this has no other characteristics than that of occurring in the first year of the disease and disappearing after mercurial treatment. Such a condition is possible, though perhaps some of the recorded instances are coincidences.

The chief symptom of *tracheo-bronchial ulceration* is *inspiratory dyspnea* due to the contraction of the infiltrated air passages. Gehrhardt (Milian) states that dyspnea due to laryngeal obstruction causes the patient to throw back his head in the effort of breathing, while tracheal stenosis makes him throw it forward so as to relax the trachea.

Accompanying this dyspnea there is more or less cough and expectoration and the breath is more or less foul, depending upon the depth and extent of ulceration. Such fatal complications as edema of the glottis, inspiration pneumonia, and asphyxia by occlusion of one or both bronchi are exceptional. Infiltration of the surrounding tissues may cause cellulitis of the deep tissues of the neck as well as involvement of the nerves and vascular structures. Such complications, however, are extremely rare.

Symptoms of Hereditary Pulmonary Syphilis.—Usually there are no symptoms. The patient is either stillborn or dies within a few hours, and a white pneumonia is discovered among other visceral lesions after death. Gummata are extremely rare (Kokawa). If the infant lives a few days or weeks it is likely to die of acute broncho-pneumonia.

It is to be noted that the consolidation of syphilitic pneumonia is not very dense, and, although post-mortem examination may show consolidation throughout one or several lobes, ante-mortem examination usually fails to reveal any physical sign of it.

The diagnosis is founded upon the presence of other lesions

of syphilis and the presence of dyspnea and cyanosis without physical signs.

Symptoms of Pulmonary Syphilis in the Adult.—The symptoms of pulmonary syphilis in the adult are both obscure and irregular. There may be marked symptoms with slight pathological change, and practically no symptoms with extensive lesions. Thus the condition is very rarely diagnosed during life. It is usually associated with other visceral lesions of syphilis. It occurs late in the disease. Its symptoms may be overshadowed by those of tertiary laryngeal or tracheo-bronchial ulceration.

The usual clinical types are *the silent type,* in which the lesions (often gummatous) exist for years without causing any symptoms. As Bazin says: "The victims of pulmonary syphilis eat their food and walk about like people in good health; their expectoration is slight and grayish, their cough and dyspnea not marked." But the course of such a lesion may be interrupted by acute attacks of bronchitis or broncho-pneumonia which are liable to become chronic and to relapse; hence the *bronchial or broncho-pneumonic type* of the disease.

In the third place there are those cases in which the more or less slight pulmonary lesions are overshadowed by the dyspnea due to lesions in the upper air passages: *the tracheal type.*

Finally, there is the important *pseudo-tubercular type* due to interstitial pneumonia and bronchiectasis. The course of such lesions is chronic and interrupted by attacks of bronchitis or broncho-pneumonia. The symptoms are not very characteristic, though the *dyspnea* may be more marked than is accounted for by the physical signs. *The lesions are usually seated in the lower lobe* and on the right side. Pain, cough, expectoration, and cyanosis are quite variable. Hemoptasis is not common, though it does occur and may be repeated and severe. The physical signs are either those of an irregular pneumonia or of the bronchio-ectatic cavities. Pleural adhe-

sions and acute mixed infection may complicate the physical signs. Fever is absent and the pulse is slow except during these acute intercurrent attacks.

Diagnosis.—The diagnosis of *hereditary lung syphilis* is made on autopsy; that of *trachio-bronchial ulceration* depends upon the association of dyspnea and expectoration with other evidences of ancient syphilis; that of *pulmonary syphilis* in the adult depends upon evidences of pulmonary disease in connection with old and neglected syphilis; it is usually associated with syphilis of the liver.

Differential Diagnosis.—One must distinguish pulmonary syphilis from tuberculosis.

The lesions found by physical examination may resemble tuberculosis, but should be distinguished from it by the evidences of syphilis elsewhere, the absence of tubercular bacilli from the sputum, the site of the lesions—which are usually unilateral and seated in the lower rather than in the upper part of the lung and almost always on the right side, the relatively slow pulse and slight fever.

Two other striking features may help in the diagnosis: viz., marked dyspnea with no pulmonary lesion adequate to account for it, or marked pulmonary lesions of great chronicity with relatively slight fever or impairment of the patient's health.

Finally, the therapeutic test helps to clear up the diagnosis, though its findings are not absolute, and an ancient sclerosis or bronchiectasis cannot be affected by specific treatment.

Treatment.—The treatment for all these tertiary lesions in the adult should be "mixed." But, on account of the bronchial or laryngeal congestion which may result from the administration of iodids, these must be given with extreme caution, if at all; and the best rule is to submit the patient to a course of intramuscular mercurial injections before administering the iodid. If these fail, iodids may be given, but only in small doses, very gradually increased.

CHAPTER XXXI

SYPHILIS OF THE LIVER

THE preëruptive jaundice due to *secondary liver syphilis* has already been described (page 265). Exceptionally, this jaundice occurs or relapses at a later period [1] (within the first eighteen months). Although usually mild, a few fatal cases have been recorded.[2]

But the term "liver syphilis" is broadly applied, not to this transient secondary lesion, but to the tertiary, the sclero-gummatous lesions, which are the commonest and most obvious visceral manifestations of syphilis.

Occurrence.—Tertiary syphilis of the liver occurs in the later years of the disease. It is often not diagnosed during life; hence one cannot keep an accurate clinical record of its frequency. I have record of only 7 cases, occurring respectively at three, five, six, fifteen, sixteen (relapse at twenty-two), twenty, and thirty years after the chancre. Fournier found liver syphilis only 9 times among 4,400 tertiary lesions.

Alcohol, hardship, and neglect are obvious etiological factors. Hudelo states that 25 cases are seen in men to 10 in women.

Pathology.—The pathological changes wrought by syphilis upon the liver are almost precisely the same as those of the lung, but uncomplicated by the secondary infections and adenomatous changes that confuse the latter lesion.

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[1] Only once noted in my series.

[2] Acute parenchymatous hepatitis.

Hereditary Syphilis.—Syphilitic stillborn infants always show liver lesions (Hochsinger). Those that live usually escape.

The liver is increased in size, pale in color, but may show no other macroscopic change. Numerous small (miliary) gummata are sometimes found.

Microscopic examination reveals a characteristic, diffuse, perivascular, small-cell infiltration. The miliary gummata merely show a localized intensification of this process.

Hecker has described an acute necrosis without infiltration, and Schüppel an hereditary pyelophlebitis.

Secondary (Acquired) Syphilis.—The lesions are those of an acute, diffuse, congestive, or degenerative toxic hepatitis with but little perivascular exudation.

Tertiary (Acquired [1]) Syphilis.—Although it is customary and convenient to describe hepatic sclerosis and hepatic gumma as though these lesions often stood alone, and although actual instances of hepatic gumma without sclerosis or of syphilitic sclerosis without gumma have been reported, the fact remains that gumma and sclerosis are but manifestations of a single pathologic process, and almost always occur together. The clinical picture varies with the predominance of the one or the other, but the pathologic picture remains essentially the same.

The Gross Aspect.—The liver is usually large and deformed. It may or may not be adherent to the surrounding organs; but the *capsule* almost always shows more or less irregular whitish thickening. This thickening may be due to a diffuse productive perihepatitis or to the thick scars left by healed gummata.

The irregular shape of the liver is due to *bands of dense sclerotic tissue* traversing it irregularly, criss-crossing at vari-

[1] This type may occur in the later years of an inherited syphilis. It is well described by Funke, *Med. News*, 1905, vol. lxxxvii, p. 67.

ous points, radiating from certain others, and generally most numerous near the anterior surface of the liver in the region about the falciform ligament. The quantity and distribution of this sclerotic tissue is utterly irregular, and it is quite futile to attempt to classify the resultant deformity in any but the broadest terms: as a rule, the process is most marked near the anterior surface of the right lobe, whence the intersecting fibrous bands radiate in every direction, thinning out as they approach the hilum. This produces a generally lobulated appearance.

The gummata (cf. also p. 328) are found either as hard, yellowish nodules or as cheesy masses at the intersection of several bands of sclerosis or beneath (and involving) the capsule (Fig. 61). These gummata are rarely smaller than a pea, and may attain an enormous size, projecting noticeably from the surface of the enlarged viscus. They are usually quite numerous.

If this *sclero-gummatous* process is slight and healed, a few stellate scars on the capsule, whence some slight fibrous bands radiate within the organ, may be all the evidence remaining, while if pronounced it may result in marked atrophy of the affected lobe.

Such *exceptional conditions* as diffuse, general hypertrophy, generalized sclerotic atrophy, and miliary gummata (such as occur often in the hereditary type) need only be mentioned.

Microscopical Characteristics.—The cirrhosis is seen to be chiefly perilobular, though it sends into the lobules irregular, blunt bands of fibrous tissue. Here and there at the edges of the sclerotic bands are areas of more recent syphiloma, the usual perivascular cellular exudate in process of organization into fibrous tissue. Exceptionally, this exudate is massed into minute "miliary gummata," while, chiefly at centers of fibrous intersection, the large gummata are seen in various stages of degeneration (Fig. 61).

There is considerable infiltration of the hepatic artery and its branches, and of the portal system as well.

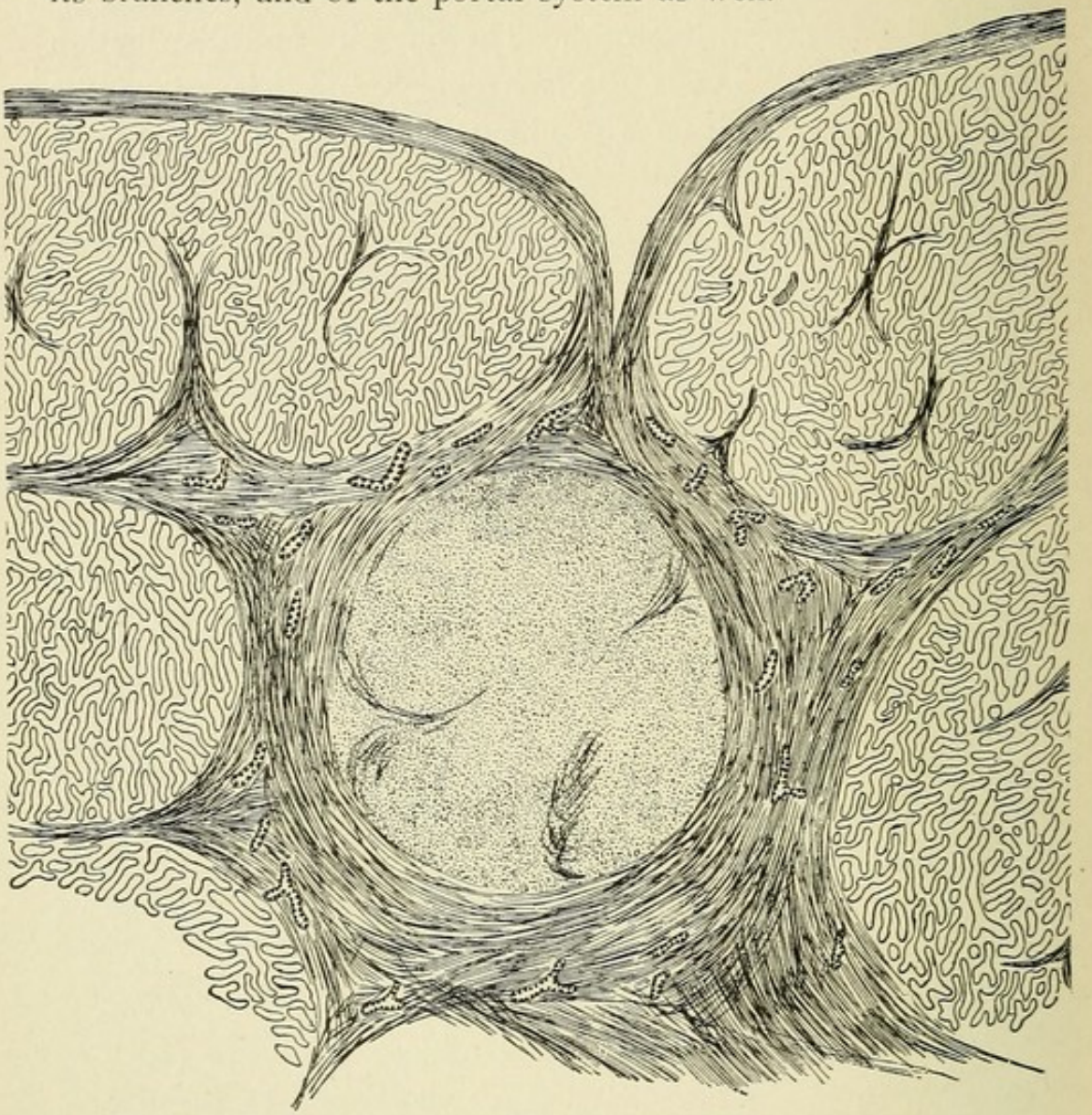

FIG. 61.—GUMMA OF LIVER. (Councilman, in "Morrow's System.")

Associated Lesions.—Suppuration of a liver gumma is extremely rare. Amyloid degeneration due to syphilis presents no peculiar characteristics (page 270); it occurs alone or in connection with sclero-gummatous hepatitis. Adhesion of the liver to the surrounding viscera is fairly common. Obstruction of the larger vessels or bile ducts by gumma or sclerosis is exceptional.

The liver lesions are usually associated with enlargement

of the spleen and chronic, interstitial nephritis. Other tertiary lesions (of skin, bones, etc.) are often to be found.

Symptoms.—The symptoms of hepatic syphilis—when it excites symptoms—are usually comparable to those of (non-syphilitic) cirrhosis or of neoplasm. In many instances, however, there are no local signs of disease, in which case there may or may not be a marked and progressive cachexia. Finally, there remains a small class of cases in which fever is the predominant symptom.

We may, therefore, classify the symptoms of hepatic syphilis according to the following types, arranged in what is probably their order of frequency; but recognizing that, whether in the liver or elsewhere, syphilis is bound by no absolute law of type:

1. Neoplastic type.
2. Cirrhotic type.
3. Cachectic type.
4. Febrile type.
5. Silent type.

Neoplastic Type.—The first symptom noted by the patient is usually pain or ascites, less often jaundice.

The *pain* often amounts to little more than an uneasiness in the region of the liver with slight tenderness. This pain is increased by exercise. It increases gradually during the first few months of the disease, but rarely attains any great intensity. Nocturnal exacerbation, so common with other types of syphilitic pain, is rather the exception here. After lasting a few months, the pain usually lessens little by little, and finally disappears.

This pain is ascribed to perihepatitis. In those exceptional instances in which the liver becomes adherent to the adjacent viscera the pain may be intense.

Ascites is a frequent symptom of syphilis of the liver. It increases slowly and recurs rapidly after paracentesis.

Jaundice is a rather rare symptom. When present it entails all the associated symptoms, such as clay-colored stools, bile-stained urine, itchy skin, etc.

Later in the disease such symptoms as *progressive cachexia* (loss of flesh and strength), *gastro-intestinal derangements* (loss of appetite, vomiting, intestinal fermentation, diarrhea), and various *secondary results of impeded portal circulation* (such as hemorrhoids, varicosities, edema) occur, as in other chronic hepatic diseases. The syphilis imprints upon them no special features, and they have no diagnostic significance.

Physical examination usually reveals *a large liver*, which may reach almost to the umbilicus. Upon the surface of the organ one may often feel one or more large, rounded projections, while similar projections from its free border may render this utterly irregular. These projections may even be pedunculated and movable. Moreover, a careful examination usually shows that one lobe or one part of a lobe is disproportionately enlarged.

The *spleen* is usually enlarged, and the *kidneys* the seat of chronic nephritis.

Diagnosis.—In certain cases the diagnosis is easy, either from the syphilitic history or the contrast between the large or multiple neoplasms manifestly growing from the surface of the liver with the excellent health of the patient. But often —exceedingly often in current surgical experience—syphilis is absolutely denied, and local conditions are so obscure as to permit grave doubt. If the tumor manifestly arises from the liver, it may be presumed to be syphilitic, for primary neoplasm of the liver is extremely rare; but if it is in the region of the gall-bladder, the probability is against syphilis and in favor of neoplasm, inflammation about the biliary passages, or tubercular peritonitis. Yet the following case, related by Cumston,[1] is an

[1] *Ann. Surg.*, 1903, vol. xxxii, p. 725.

excellent example of how every means of reaching a diagnosis —even exploratory operation—may fail:

Case XXXV.—Male, about forty years old. For several months he suffered pain in the right hypochondrium, and has been slightly jaundiced. Tongue furred; temperature normal; pulse 80. "A large, somewhat nodulated mass in the region of the gall-bladder; the border of the liver extending about three fingers' breadth below the costal margin."

Diagnosis, tumor of liver or biliary passages.

Laparotomy revealed slight ascites, a large liver, and a gall-bladder surrounded by adhesions, but which when freed proved normal and empty. A diagnosis of ulcer of the duodenum was therefore made, but the adhesions were so dense it was deemed inadvisable to interfere further.

After operation it was learned that the patient had had syphilis, and he was therefore treated by large doses of potassium iodid. Cure in three months, persisting two years.

In the same paper Dr. Cumston reports a number of instances in which he resorted to exploratory incision for a diagnosis. Such a measure is entirely proper if the condition appears to be operable (supposing it to be malignant), and not primary in the liver; but, if inoperable or apparently growing from the liver, a sharp test course of antisyphilitic medication should be given the preference.

Cirrhotic Type.—Less frequently the disease shows all the symptoms narrated in the preceding paragraph, except that there are no marked irregularities or tumors palpable upon the surface of the liver. Accordingly, the condition resembles non-syphilitic cirrhosis. The liver is usually moderately enlarged (or it may be contracted), and thus the condition simulates the hypertrophic (or the atrophic) form.

Diagnosis.—The presence of other syphilitic lesions, scars, or a syphilitic history are almost essentially for a differential diagnosis. The diagnosis by exclusion would not be unjustifiable if a case of cirrhosis not manifestly due to alcoholism or

any other of the usual causes were subjected to mixed treatment, and cured thereby.

Cachectic Type.—Not only may the characteristic irregularities of tumors upon the liver be lacking, but there may be also an entire absence of jaundice or ascites, of pain, and of marked change in the size of the liver. The patient suffers vague intestinal symptoms, and becomes gradually more and more cachectic without obvious cause. Very careful examination may disclose a slight enlargement of the liver and spleen, but even then there may be no direct evidence of hepatic disease. Such cases are habitually undiagnosed unless submitted to exploratory operation or until they reach the autopsy table, unless the presence of other lesions of syphilis shall lead to a course of mixed treatment.

Febrile Type.—The so-called fever of tertiary syphilis has already been described (page 269). In most of the reported instances this fever accompanies syphilis of the liver, and where manifest lesions of the liver were absent one may doubt whether autopsy might not have revealed some lesions of this organ. Though this fever is neither frequent nor characteristic in type, it is none the less important, since it is manifestly due to syphilis and usually to liver syphilis, and may be cured by antisyphilitic treatment.

Silent Type.—Post-mortem examination reveals active lesions or scars of hepatic syphilis in many cases that have shown no symptoms of such lesions during life. The precise frequency of such cases has not been estimated.

Diagnosis.—Hudelo lays down these two fundamental propositions:

"1. Every obscure cachectic condition in a patient known to be syphilitic suggests a visceral lesion, and in particular a lesion of the liver.

"2. Every chronic disease of the liver with irregular symptoms and of obscure origin should make us think of syphilis."

Prognosis.—The prognosis is bad in a sense, for liver syphilis occurs in relatively old and obstinate cases, and the patient is rarely willing or able to undergo the prolonged course of treatment necessary to assure a cure. On the other hand, it is surprising how well the worst cases sometimes do under treatment, so long as the other viscera—notably the kidneys—are not too gravely implicated.

Treatment.—A strict diet and the application of mercury by some other than the alimentary route are required to spare the digestive organs; iodid must be given freely. Surgical measures, such as those employed in certain cases by Dr. Cumston, for example, are not calculated to cure the disease, and but little good can be expected from them, unless by the removal of pedunculated masses of gummatous tissue (Koenig [1] and Cumston).

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[1] *Berl. klin. Woch.*, 1905, February 6.

CHAPTER XXXII

SYPHILIS OF THE GENITAL ORGANS

SYPHILIS OF THE TESTICLE

SYPHILIS of the testicle is a relatively common and characteristic lesion, which, like the other visceral lesions, is much more often found on autopsy than during life.

I have record of 67 cases, 10 of them bilateral. The following table shows the dates of onset:

4 to 7 months	3 cases
12 to 18 "	5 "
2 years	4[1] "
3 "	9[2] "
4 "	6 "
5 "	1 case
6 "	4 cases
7 "	3 "
8 "	1 case
9 "	2 cases
10 "	1 case
11 "	4 cases
12 "	3 "
15 "	2[1] "
26 "	1 case
31 "	1[1] "
Indefinite	17[3] cases
	67

It will be noted that, though three of the cases occurred between the fourth and the seventh month, the lesion is not common during the first year; though over half the cases occurred within the first four years.

[1] One bilateral. [2] Three bilateral. [3] Six bilateral.

Yet the appearance of syphilis in both testicles is no evidence of a recent syphilis, as shown by its occurrence once in the fifteenth and once in the thirty-first year.

Morbid Anatomy.—The French school, following Dron,[1] recognize a secondary epididymitis and a tertiary orchitis or epididymo-orchitis. The distinction cannot be clinically established. Of my three earliest cases one was distinctly an orchitis, and other authors (cf. Lang) have reported similar experiences.

Clinically it is safer to consider all syphilis of the testicle rather tertiary than secondary.

The Testicle.—The syphilitic testicle usually shows marked interstitial sclerosis, sometimes considerable gummatous infiltration. Thus the process is the familiar sclero-gummatous one; but as the inflammation is painless and usually progresses very slowly, it may never be discovered at all, or it may almost totally destroy the seminal tubules before the patient considers it worthy of medical care.

Active gummatous orchitis, however, may cause rather rapid enlargement and terminate by involvement of the overlying tissues and eruption through the skin, leaving a typical syphilitic ulcer, in the base of which the tubular structure of the testicle may be discerned.

The Epididymis.—The epididymis alone may be involved (secondary epididymitis of Dron), though more often the testicle is implicated as well. The lesion is usually confined to the globus major, which forms a hard, solid, infiltrated mass with a sharp edge. It caps the end of the testicle, separated from it by a distinct sulcus, so that the organ seems to be resting in a clam shell. Gummatous nodules are very rarely felt in the epididymis. This diffuse infiltration, sharp-edged, not nodular and not sensitive, is very characteristic of syphilis.

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[1] *Archiv. gén. de méd.*, 1863, vol. ii, pp. 513, 724.

The French speak of it as a "helmet crest"; the comparison with a clam shell is more familiar to our minds.

I have in one instance seen syphilis begin in the epididymis as a rounded nodule, the size of a marrow-fat pea, and to progress by the addition of other nodules in the epididymis and in the testicle itself. These gummata so closely resembled tubercles that the testicle was removed under a mistaken diagnosis. Such rounded nodules in the epididymis, however, are extremely rare.

The Tunica Vaginalis.—Hydrocele is almost always present, but the amount of fluid is, as a rule, not very great. Adhesive vaginalitis is found after the fluid has been resorbed in the course of a cure.

Symptoms.—The characteristics of the syphilitic testicle are painlessness and slow growth; as a rule but one testicle is involved. It does not attain a very great size; it does not ulcerate through the skin unless it has been neglected for a long time.

Examination reveals a testicle wooden in hardness. If the epididymis is involved, the sharp, clam-shell edge of the globus major (less often the globus minor) can usually be made out without drawing off the hydrocele fluid.

If there is orchitis, the testicle is either generally involved, evenly and densely hard, or else it is of uneven hardness, with projecting small gummata.

The vas deferens was involved in only one of my cases. The general health is not impaired, but, if both testicles are involved, sexual appetite and power are likely to be lost.

The course of the disease is infinitely slow; it terminates either in fungus or in atrophy.

Prognosis.—The prognosis is excellent. Whatever part of the parenchyma has not been destroyed by sclerosis will continue to functionate, and the testicle which has been syphilitic for years may still secrete spermatozoa. But the patient

should be warned that the result of treatment upon an enlarged testicle may be to cause such absorption of the syphilitic tissue as to reduce the gland far below its normal size, while any delay in instituting treatment will only make this atrophy more marked.

The hydrocele disappears with cure of the orchitis.

Diagnosis.—The diagnosis of syphilitic testicle is often easy from the appearance of the organ and the syphilitic history. Exceptionally, the onset of the disease is accompanied by mixed infection, so that for a time the testicle is tender and there is some little temperature.

This mixed infection is not obviously connected with gonorrhea, and usually leads to the diagnosis of tuberculosis; but in a short time the fever and tenderness disappear and the characteristic epididymitis or orchitis remains.

In the later stages the general irregular involvement of the whole gland may lead to a diagnosis of neoplasm; but here the general rule applies absolutely: No testicle should be removed for neoplasm until the patient has been given the benefit of a test course of mixed treatment, which test course should imply hypodermic medication.

The aspirating needle may do good service, both in withdrawing the hydrocele that obscures the outlines of the testicle and in distinguishing between a solid growth and hematocele.

Treatment.—The general treatment is along the usual lines. We may not expect to bring a badly disorganized testicle back to an entirely normal condition. Local treatment is of no value. The hydrocele requires no treatment. Ancient syphilis of the testicle often resists every form of treatment short of mercurial injections.

CHANCRE REDUX

Any syphilid, secondary or tertiary—papular, ulcerative, tubercular, gummatous, squamous, mucous—may occur upon

the penis. In this there is nothing extraordinary or important. But the fact that certain of these syphilids absolutely mimic true chancre is most important. Such sores are common. They are the foundation for many a theoretical "second attack" of syphilis. They have misled the best of syphilologists, and will continue to mislead so long as men dare make the diagnosis of chancre from the appearance of the sore alone; for between true chancre and eroded or ulcerated syphilitic tubercle or gumma of the penis it is impossible to distinguish without collateral evidence.

Hence the name and the significance of *chancre redux*.

Chancre redux is thus nothing more than a tubercular or gummatous syphilid upon the glans penis or the foreskin. It imitates true chancre in all its types; it may or may not be phagedenic. When it occurs in the first two or three years of the disease it usually springs from the scar of the chancre, but later it manifests no such predilection. It is usually single (though I have seen three appear almost simultaneously in the twenty-seventh year of the disease).

Occurrence.—Our records show 75 instances of chancre redux in 71 patients. The four relapsing cases occurred in the following years: one to three, eleven to twenty-seven, thirteen to seventeen, and fourteen to twenty-one. The date of onset was as follows:

First year	3 cases	Tenth year	3 cases
Second year	4 "	Eleventh to fourteenth year.	10 "
Third year	11 "	Sixteenth to seventeenth year	4 "
Fourth year	3 "	Twentieth to twenty-first year	2 "
Fifth year	7 "	Twenty-sixth to twenty-ninth year	4 "
Sixth year	8 "		
Seventh year	8 "		
Eighth year	1 case	Thirty-second year	1 case
Ninth year	1 "	Indefinite	5 cases

Thus half the lesions appeared between the third and the seventh years of the disease.

DIAGNOSIS.—To the description given above, the diagnostic table on page 246, and the instances of erroneous diagnosis on page 44, nothing need be added. We may summarize by stating that chancre redux usually lasts much longer than true chancre, is quite frequently phagedenic, is not associated with inguinal adenitis except accidentally, does not reveal the spirocheta in smears taken from its surface, and is not followed by any outbreak of secondary symptoms.

TREATMENT.—The treatment is that of the same lesions elsewhere on the body. The sore may be kept clean by calomel powder.

CHAPTER XXXIII

SYPHILIS OF THE CIRCULATORY AND LYMPHATIC SYSTEMS

THE syphilitic lesions of the capillaries that underlie the pathology of all syphilitic lesions (page 48) and that disease of the cerebral arteries which is the most important feature of syphilis of the nervous system (page 373), have already been described. Compared to these two the remaining lesions of the circulatory and lymphatic systems are relatively infrequent, though some of them (e. g., syphilis of the aorta) are extremely grave.

The subject may be subdivided as follows:

Syphilis of the heart.
- Secondary lesions.
- Tertiary lesions.
- Lesions of the coronary arteries.

Syphilis of the aorta.
- Aneurysm.

Syphilis of the arteries of the extremities.
- Aneurysm.

Syphilis of the veins.

Syphilis of the lymphatic system.

SYPHILIS OF THE HEART

Syphilis of the heart is both rare and obscure. The single instance of it on our records has been mentioned (page 5). This was supposed to be a syphilitic myocarditis; it improved

slowly under treatment and was finally cured. In all of its manifestations syphilis of the heart simulates other conditions, from which it can be distinguished only by the therapeutic test or by autopsy, the former a test unsatisfactory to the pathologist, the latter a proof of little avail for the patient.

Secondary Lesions

Fournier recognizes three clinical types of "secondary" heart lesions, manifested respectively by palpitations, tachycardia, and cardiac arrhythmia. That such conditions occur in syphilis cannot be doubted, but that they are due to syphilis it is almost impossible to prove; they do not kill, they get well under antisyphilitic treatment; they also get well without such treatment.

I have recently seen two cases in point; both developed spontaneously, and while under treatment a most annoying tachycardia. One case occurred in the first, the other in the third year of the disease. In both instances antisyphilitic medication was at once suspended, hygienic and tonic measures instituted, and after a few weeks the heart resumed its normal action, and the tachycardia has not recurred.

Doubtless all the cardiac irregularities occurring during early syphilis are attributable to neurosis (which may itself be caused by syphilitic toxemia) or to digestive disturbance, as these were, and may be cured by appropriate measures and without the aid of antisyphilitic remedies.

Tertiary Lesions

Sclero-gummatous Myocarditis.—In the heart, as in the other viscera, diffuse syphilitic inflammation and localized gumma always coexist, though clinically one or the other predominates. The areas of sclerosis (or interstitial myocarditis) follow the sclerosed capillaries of the coronary arteries. The gummata may be single or multiple, small or large.

Symptoms.—The myocarditis causes arrhythmia and dilatation. Gumma [1] causes sudden heart failure; it cannot be diagnosed during life.

Diagnosis.—Symptoms of myocarditis (arrhythmia and dilatation) in a known syphilitic demand a prompt "test" course to prove whether the myocarditis is syphilitic.

For gumma there is no diagnosis.

Pericarditis and Endocarditis.—These are post-mortem features of syphilitic myocarditis.

Bradycardia and Stokes-Adams Syndrome.—These are exceptionally due to syphilis. In a recently reported case the cause of heart block was found to be a gumma of the bundle of His.[2]

Lesions of the Coronary Arteries

Syphilitic lesions of the coronary arteries, though always associated with similar lesions in the aorta, and often with myocarditis, may clinically dominate the scene. The arteries are affected throughout their length, all the coats are involved (endoperiarteritis), the inflamed vessel rapidly becomes sclerosed (Deguy [3]).

The clinical manifestation of syphilis of the coronary arteries may be *sudden* death due to infarct, rupture of the heart, or aneurysm of the artery itself; but, as a rule, the symptom complex is that of angina pectoris.

Syphilitic Angina Pectoris.—"Syphilitic coronaritis with angina pectoris in persons thirty or thirty-five years of age (Henderson); it is as frequent in women as in men (Fraenkel)—it usually ends in sudden death—and antisyphilitic treatment is all-powerful [4] if the diagnosis is made in time" (Deuy).

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[1] Bruhns, *Berl. klin. Wochenschr.*, 1906, vol. xliii, p. 513.

[2] *Presse méd.*, 1907, xv, No. 8.

[3] Fournier's *Traité*, vol. ii, Part II, p. 773. An excellent essay on cardiac syphilis.

[4] Cf. Dieulafoy, *Presse méd.*, vol. xiv, No. 32.

SYPHILIS OF THE AORTA

Syphilis of the aorta is confined almost exclusively to the arch, where it produces patches of endoperiarteritis. These promptly become sclerotic and lead to aneurysm, small or large, single or multiple, or to disease of the coronary arteries. It is by these two lesions, therefore, that it is manifested clinically.

Aortic Aneurysm.—We cannot undertake to describe aneurysm of the aorta, but must confine ourselves to a discussion of the following pertinent questions:

Does syphilis cause aortic aneurysm?

What is the frequency of syphilitic aneurysm and what the date of its occurrence?

May syphilitic aneurysms be recognized as such?

Is antisyphilitic treatment of any value?

Does Syphilis Cause Aortic Aneurysm?—The statistics on this subject are confusing. The syphilographer does not encounter aortic aneurysm. Thus, I have seen a number of aneurysms in one way or another, yet my office records of syphilitic cases show only a single instance of this condition. On the other hand, when the patient is seen with aneurysm he usually confesses to ancient syphilis or shows scars of the disease. Étienne,[1] in a series of 240 cases of aneurysm (chiefly aortic), found 166, or sixty-nine per cent, syphilitic, and Malmsten found eighty per cent (in 101 cases).

Moreover, other causes for aneurysm sink into relative insignificance. Thus Étienne notes alcoholism only 28 times, and in those northern countries where severe privation and chronic alcoholism are so much more common than in France, aneurysms are not unduly common.

Hence syphilis is often the apparent cause of aneurysm. It is not the sole cause, and the pathologist can very rarely

[1] *Ann. de dermat. et syph.*, 1897, vol. viii, No. 1.

demonstrate typical syphilitic tissue[1] in the affected vessel. Yet that syphilis is the dominating etiological factor in the production of aneurysm may be considered as proven. "If pathology, gross or microscopic, does not always, or even generally, show that an aneurysm is or is not syphilitic; if specific treatment is often useless; it is nevertheless true that syphilitic arteritis is eminently calculated to prepare the vessel for aneurysmal dilatation. It would be most surprising if it did not produce it" (Darier[2]).

Frequency and Date of Incidence.—From the evidence it may be concluded that aortic and other aneurysms are relatively rare and late results of syphilis, otherwise the syphilographer would see more of them. Yet Étienne has collected 72 cases, of which 10 occurred in the first five years, 18 in the second, 10 in the third, 14 in the fourth, 13 in the fifth, and 6 from twenty-five to forty years after the initial lesion. But the current clinical experience in this country is that the syphilis is at least ten years old before the appearance of aneurysm.

Diagnosis.—Let us frankly admit that in any given case it is impossible to state during life whether the aneurysm is syphilitic or not.

Treatment.—There is no evidence that mercury is of the slightest service in the treatment of aneurysm. *Potassium iodid* is unmistakably serviceable in certain cases; yet even so enthusiastic a supporter of the syphilitic etiology as Étienne confesses that "Iodid acts upon aneurysms quite as it does upon other lesions due to arteriosclerosis."

SYPHILIS OF THE ARTERIES OF THE EXTREMITIES

Syphilitic lesions of the arteries of the extremities rarely cause symptoms. The clinical types are two, depending upon

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[1] Cf. von Duhring, *Deutsche med. Wochenschr.*, 1905, vol. xxx, No. 51.

[2] "Syphilis arterielle," Paris, 1904.

whether the artery is weakened so as to permit aneurysmal dilatation or thickened so as to obstruct the circulation partially or completely. Thus we have—

Aneurysm, or Arterial Obstruction

Aneurysm.—The remarks made concerning aortic aneurysm apply equally here.

Arterial Obstruction.—Depending upon the degree of obstruction, the symptoms follow one of three types, viz.:

Intermittent Claudication.—There is pain or paresthesia and a tendency to cramps of the muscles of the affected limb, increased by exercise and alleviated or entirely relieved by rest.

Reynaud's Disease.—That symmetrical gangrene of the extremities may be due to syphilis has recently been suggested by Klotz.[1]

Gangrene.—Gangrene occurs from total occlusion of the artery. When due to syphilis it is usually preceded by the pain and paresthesia of intermittent claudication.

Diagnosis.—The test of treatment may sometimes prove the syphilitic nature of the lesions, just as the history may suggest it; but, as a rule, the lesion is sclerotic when attacked, and no longer amenable to antisyphilitic medication.

SYPHILIS OF THE VEINS

Acute phlebitis of the superficial veins of the lower extremities is one of the rarest complications of early syphilis (cf. Mendel[2]). It is not clear that the lesion is truly syphilitic. It runs the usual course.

Sclero-gummatous Phlebitis.—This, too, is extremely rare (cf. Vaquez[3]). It may be sclerotic, gummatous, or

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[1] Internat. Dermat. Congress at New York, 1907.

[2] *Arch. gén. de méd.*, 1894, March.

[3] Fournier's "Traité," vol. ii, Part II, p. 890.

sclero-gummatous. When the lesion affects a superficial vein it appears as a gumma or as a mere hard fusiform nodule in the wall of the vein. If a deep vein (portal, cava, jugular) is affected, the symptoms are those of pressure on adjoining structures or obstruction of the circulation (edema, ascites, etc.).

SYPHILIS OF THE LYMPHATICS

Apart from the early lymphangitis and lymphadenitis that characterize the primary lesion and the early period of the disease (pages 238, 261), *gumma of the lymph nodes* is the only lesion to arrest attention. It is not extremely uncommon, and the following cases, which I have recently published,[1] sufficiently describe its symptoms and diagnostic features:

The first patient, a man forty-eight years of age, with no previous history of syphilis, complained of a hard, irregular tumor of the cervical lymph nodes below and behind the angle of the jaw. Examination revealed an enlarged, ulcerated tonsil directly connected with the growth. A few weeks of vigorous, antisyphilitic treatment failing to have any effect, it was promptly removed, and proved to be a mixed-celled sarcoma of the tonsil.

A few weeks later I saw a patient in the third year of his syphilis, who had been quite constantly under treatment, and had, upon the left side of his neck, a swelling of the lymph nodes, at first sight almost identical with that of the previous case. Neither was particularly painful, and their degree of hardness was about the same. But in this latter case there was no involvement of the tonsil; there was a large submental lymph node which rapidly broke down and discharged a watery pus, and on the opposite side of the neck there was slight enlargement of the corresponding node. The history of syphilis, the bilateral lesion, the rapid growth and ulcera-

[1] "Bryant and Buck's Surgery," vol. ii.

tion of the submental lymph node made the diagnosis of syphilis, which was confirmed by the success of treatment.

At the time when I was treating this latter case I had under my care a third man, an alcoholic, who, although in the first year of his syphilis, had very rebellious, diffuse, tubercular syphilids. While these were disappearing under treatment, there suddenly appeared a large, tender swelling of the inguinal lymph nodes, most marked upon the left side. Examination of the preputial cavity revealed a number of mucous patches and a disgusting state of filth. Cleanliness and an antiseptic powder, together with the general treatment of syphilis, which was being vigorously pushed, promptly cured the mucous patches, and the swollen nodes in due time subsided.

The chain of causes in this instance was characteristic. First, the dirt under the prepuce caused the mucous patches by its irritation; then the mucous patches emitted an acrid secretion, which caused balanitis, of which the secondary adenitis in the groin was the first sign; and, finally, cleansing of the dirt and a little antisepsis were the means of cure, the mercurial treatment not being changed in any way.

CHAPTER XXXIV

SYPHILIS OF VARIOUS VISCERA

SYPHILIS OF THE DIGESTIVE ORGANS

SYPHILIS, though it often strikes the liver, the mouth, and the pharynx, usually spares the rest of the digestive tract and its associated glands.

Salivary Glands, Pancreas.—Gumma and diffuse syphilitic sclerosis may occur in any of the salivary glands [1] or in the pancreas,[2] but these lesions are pathological curiosities. That syphiloma of the pancreas may cause glycosuria is possible. Lancereaux states that such glycosuria assumes little clinical importance. (It is noted six times in our cases—at six, fifteen, twenty-four (twice), and twenty-five years from the chancre, and once after an unrecorded interval.)

Esophagus. — Very rare. Gastou [3] distinguishes three clinical types:

"1. *The latent type* shows no characteristic symptoms. The course is rapid. There is at first slight interference with deglutition, then grave dysphagia, generally in a case of malignant or grave syphilis. Then rapidly ensue weakness, emaciation, cachexia, and death. . . .

"2. *Dysphagic or guttural type.* In cases of this type there are pharyngeal lesions (gumma, infiltration, ulceration) near the orifice of the esophagus. Swallowing causes painful spasm and regurgitation.

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[1] Chatin in Fournier's "Traité," vol. ii, Part II, p. 559.

[2] Bensaude, *ibid.*, p. 566.

[3] *Ibid.*, p. 465.

"3. *Stricture.* This is the least rare and the most typical lesion. It begins with dysphagia . . . but without vomiting or pressure symptoms. . . . The passage of a bougie is obstructed at the level of the first ring of the trachea or at the termination of the esophagus. . . . The passage of the instrument gives no relief to the patient. The obstruction may be relieved by mixed treatment or by gastrostomy."

SYPHILIS OF THE STOMACH

Syphilis of the stomach is almost as rare as syphilis of the esophagus. In our records it does not appear. But a few autopsies and clinical histories prove that gummatous infiltration and ulceration may occur,[1] and that this condition is distinguishable during life from the conditions which it simulates only by the fact that it resists all other treatments, but may be rapidly and brilliantly relieved by mercury and iodids.

Clinically, Morgan distinguishes four types, imitating respectively chronic gastritis, ulcer, stenosis, and carcinoma. Hence these diseases must be added to those in which the possibility of syphilis must be considered, and for the cure of which—if all else fail—mixed treatment should be given on the bare chance that it may cure.

INTESTINAL SYPHILIS

Syphilis of the intestine (except the rectum), like syphilis of the stomach, imitates more familiar conditions (such as diarrhea, tumor, stricture), from which it may be distinguished during life only by the therapeutic test. The one case of suspected intestinal syphilis on our list has already been cited (page 100).

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[1] Gerry Morgan, *Am. Med.*, 1906, vol. xii, p. 123.

Gaucher alleges syphilis as a probable cause of appendicitis—at least, of 32 Frenchmen who had had appendicitis before coming to him, 8 gave a history of acquired syphilis, and 21 of hereditary syphilis.

SYPHILIS OF THE RECTUM

That syphilis of the rectum assumes so important a place in the mind of the general practitioner is due to the insistence of Professor Fournier, rather than to its frequency or clinical importance.

Apart from the chancres and secondary lesions that may encroach upon the anus and the lower inch of the bowel, we may recognize:

1. *Gumma.*—Most exceptional.

2. *Ulcers.*—Very rare, usually confined to the ultimate inch or two of the bowel, sometimes associated with similar lesions higher up.

3. Ano-rectal Syphiloma.—This lesion, though said to be much more common than gumma or ulceration, is, nevertheless, very rare. We have record of but one case.

The lesion consists of a "hyperplastic infiltration of the ano-rectal wall" (Fournier). The infiltration may be confined to the rectum, in which case it affects the region within or just above the internal sphincter. *It is never found higher* up the bowel. It consists in a diffuse cylindrical rigidity of the rectal wall (a stricture), an inch or two in breadth, admitting the finger (perhaps with some difficulty), and not ulcerated upon its surface.

It may, on the other hand, be ano-rectal or exclusively anal. At the anus it bulges out in a hard, indurated, pseudo-condylomatous mass which may undergo secondary erosion.

The progress of the lesion is slow. Though it may be

checked by being seen early, it usually terminates in a permanent cicatricial degeneration; it becomes a true stricture.

4. Syphilitic Rectal Stricture.—The loose denomination of every rectal stricture in a person who has had syphilis, as a syphilitic stricture, is utterly unwarranted. Surgeons who permit themselves this liberty find that most of their so-called syphilitic rectal strictures occur in women, and at a depth of from two to four inches up the bowel, while Fournier—than whom no one could be more enthusiastic on this subject—has seen eleven strictures in men to three in women, and finds them *all in the lower two inches of the rectum.* The strictures higher up are properly attributable to such causes as pelvic cellulitis, retroversion, trauma in parturition—all peculiar to women and bearing no relation to syphilis.

These strictures are as rare as the syphiloma that causes them.

The treatment by mercury and iodid should be tried, in the hope that what appears to be cicatricial tissue may still contain enough active syphiloma to be favorably influenced by these remedies. If these fail, dilate or incise, and give no very optimistic prognosis, for recontraction is the rule.

SYPHILIS OF THE URINARY ORGANS

Syphilis of the Kidney.—Syphilis of the kidney, though abundantly proven to exist, is still in the category of pathological curiosities.

Reference to Karvonen's [1] classic work reveals evidence that every form of nephritis may result from syphilis.

In the earlier stages of the disease there may be some slight *congestion* evinced only by a transitory albuminuria, or the lesions may be much graver, and even amount to an

[1] *Zeitschr. f. Derm.*, 1898.

acute, diffuse nephritis, in which either parenchymatous or interstitial lesions predominate. In the later years of the disease *chronic diffuse nephritis* and *amyloid kidney* occur; exceptionally there is sclerosis, or gumma, or *sclero-gumma.* These last conditions, though characteristic of syphilis to the pathologist, rarely offer a clinical picture permitting a diagnosis. Finally, it is alleged that *hemoglobinuria* occurs oftenest in syphilitic patients, and certain brilliant cures of this condition by mercury or iodids are alleged as proof of its syphilitic origin.

We may group all these lesions under two heads: the one medical, including albuminuria, hemoglobinuria, diffuse nephritis, acute or chronic, whether predominantly parenchymatous or interstitial, amyloid kidney, and sclerosis of the kidney; under the other, or surgical head, we may place gumma of the kidney. Of the former class it may be said that, with the exception of certain sclerotic cases, they possess no pathological characteristics to stamp them as exclusively syphilitic or to differentiate them from similar conditions due to other causes.[1] Clinically speaking, these medical conditions are often attributed to syphilis solely because they are discovered in a syphilitic subject. But when we consider the frequency of nephritis, especially chronic nephritis, in every walk of life, and add the special liability to nephritis which must exist in patients who die avowedly syphilitic, especially in those hospital cases that reach the post-mortem table, it is small wonder that so-called syphilitic nephritis (i. e., nephritis in a syphilitic patient), as noted by the pathologist, is extremely common.

Take, for example, Spiess's figures, compiled from autopsies made between 1866 and 1875 at the Charity Hospital in

[1] Syphilis of the renal arteries can sometimes be distinguished as the underlying cause of chronic nephritis; but it is by no means easy in the kidney to distinguish syphilitic arteritis from arterial sclerosis due to other cause.

Berlin. Among 220 syphilitics he found the kidneys sound in only 62 cases. In the remaining patients he found:

Amyloid kidney	42	times
Parenchymatous nephritis	21	"
Sclerosis of the kidney	18	"
Interstitial nephritis	16	"
Atrophy of the kidney	11	"
Sclero-gummatous kidney	7	"
Various inflammations only partially attributable to syphilis	16	"

But the clinician very rarely comes upon nephritis attributable to syphilis. One would expect acute nephritis to be a common complication of so virulent an infection as early syphilis sometimes is; yet it is surprisingly rare. I do not remember to have seen any symptoms of nephritis in the early stages of syphilis beyond the appearance in the urine of a little albumin and a few casts. In the later years of the disease, however, nephritis is quite common; but it is almost always of that latent, chronic type which shows itself only by the presence of albumin and casts, a slightly decreased output of solids, and no active subjective symptoms. The difficulty which constantly arises in the clinical management of such cases is to decide whether the nephritis is due to the disease, to its treatment by mercury or iodids, or to outside causes. I am strongly inclined to attribute the chronic nephritis I see in the later years of syphilis to overtreatment and alcoholism rather than to the disease itself.

Reference has already been made to the toxic effect of mercury upon the kidney and to the manifest danger of exhibiting it in high doses if the kidneys are not sound. On the other hand, the treatment necessary to the cure of the average case of syphilis does not the least damage to the kidneys; and if twenty-five per cent of all cases of syphilis die with chronic nephritis (Karvonen) this is probably attrib-

utable only in some measure and indirectly (through arterial sclerosis) to the disease, and often to the excesses, the deprivations, and the other nocive circumstances which have had a share in terminating the patient's life.

Diagnosis.—Syphilis of the kidney may be suspected if a patient develops albuminuria or symptoms of nephritis during the course of his syphilis. As Mauriac (quoted by Berdal) says: "Once it is proven that the (nephritic) patient has had syphilis, we must estimate this as a factor of prime importance; not that it (syphilis) is the most important cause of Bright's disease, but because it gives us hope and a therapeutic indication. . . . The insignificance, the fewness of the early manifestations, and the long period which has elapsed without the appearance of any syphilitic accidents, none of these things impair the force of this precept, and we must respect it even in those cases in which the action of syphilis upon the kidney appears problematic and subordinated to graver and more proximate pathogenic influences."

The test of treatment may be applied to confirm or refute the suggestion. But the usual regulations of diet, etc., should not be neglected, and clinically only those cases may be considered proven of syphilitic origin in which a cure is effected by antisyphilitic treatment alone, after the strictest milk diet and attention to other therapeutic precautions have failed. Such cases are as rare as white blackbirds.

Treatment.—In spite of the recognized irritating effect of mercury upon the kidney, the few cures of syphilitic nephritis that have been obtained are attributable to mercurial treatment; but the mercury must be administered cautiously, its effect upon the urine being checked by almost daily urinalysis. It is preferable to spare the digestion, and not to risk the sudden explosion of mercurial poisoning which is always possible when mercury is given either by fumigation or by inunction. The injection of insoluble salts is also too violent, and

the best method of treatment is by the injection of soluble salts, beginning at about half the usual dose and increasing carefully and slowly. If some two weeks of such treatment produce no effect, iodid may be administered in doses not higher than gr. xx or xxx (2 gm.) a day for three weeks; and if at the end of this time nothing has been achieved, the theory that syphilis is the cause of the nephritis may be dropped—at least it is fairly proven that antisyphilitic treatment will do no good.

Sclero-gummatous Nephritis.—This type of lesion we have dubbed "surgical" because its symptoms (if it produce any) suggest surgical disease of the kidney, either neoplasm or tuberculosis. The lesion is usually confined to one kidney; the gummata may be large and single or smaller and multiple. They begin usually in the cortex, and, after growing to a considerable size, burst within the pelvis of the kidney and discharge through the urinary organs.

The sclerotic process, like that in the liver, is usually widespread in the form of bands of scar tissue radiating from sclerotic or gummatous centers. The sclerosis may be slight and partial or general, and may be sufficiently widespread to cause atrophy of the organ; but the renal parenchyma between the areas of sclerosis is usually practically normal.

Gumma of the kidney does not occur before the fourth or fifth year of the disease, but may occur at any time thereafter. It is usually associated with other visceral syphilitic lesions.

Symptoms.—Sclerosis of the kidney without gumma can only be diagnosed on autopsy. It gives no symptoms to differentiate it from other forms of chronic nephritis; nor does gumma give any symptoms until it has either grown to a considerable size or has burst into the pelvis and given rise to pyuria.

The following cases reported by Israel [1]—the one mistaken for tumor, the other for tuberculosis—sum up admirably the present status of our knowledge of gumma of the kidney:

Case XXXVI.—In October, 1888, Israel was consulted by a woman, twenty-three years old, on account of a tumor of the right kidney. She had been married four years and had borne three children, of whom the first died in twenty-three days and the second in one year.

In March, 1888, she had bilateral, syphilitic iritis. In June she was delivered of a healthy child. For four weeks she had suffered from frequent urination and abdominal and pelvic pains, and, for two weeks, from a sharp pain in the right side, without cough. The urine was normal. The right kidney was large, hard, sensitive, and immovable. The urine was increased in volume, of low specific gravity, and contained a few leucocytes and bacteria, once a hyaline cast, and sometimes albumin and sometimes not. Continued pressure on the kidney markedly increased the epithelial sediment and caused slight bleeding. Repeated experiments of this kind produced a slight but definite decrease in the volume of the tumor.

A course of iodids proved futile, and on January 22d nephrectomy was performed. The fatty capsule was found edematous, scarred, and adherent. The kidney was found apparently normal in its upper two thirds, while the lower end was occupied by a hard growth. It was therefore removed. Section of the kidney showed syphilitic and interstitial nephritis and hyperplastic perinephritis. After operation the patient gained forty pounds, and bore a healthy child within a year, while the urine continued to show albumin from time to time for five years in spite of iodids. Since then the patient has been well and has borne several children.

Case XXXVII.—The patient, a man, thirty years old, had lost two brothers of phthisis. He had had a chancre many years before, and had been treated with mercury. In May, 1889, he began to have continuous pain in the left side. Six months later he began to have short, sharp pains every hour or so. Then a redness appeared over the tenth rib in the axillary line, and three

[1] "Chirurgie der Nieren."

weeks later a supposed abscess of the spleen was incised. Fourteen months later, in December, 1890, the patient consulted Israel, who found a diffuse, adherent growth in the region of the kidney, in front of which the spleen could be made out, distinct and movable, while a fistula led between the eleventh and twelfth ribs into the tumor. The urine was purulent and albuminous; tubercle bacilli could not be found in it.

January 3, 1890.—*Nephrectomy for Tuberculous Kidney.*—The fistula was followed down after resection of the eleventh and twelfth ribs, and the kidney isolated with great difficulty from the dense, perinephritic scar. The wound healed extremely slowly, but the patient gained weight and was apparently well in 1900. Investigation showed gummatous degeneration of the kidney.

In either case the diagnosis of gumma might have been made had there been other prominent lesions of syphilis; and in the former case one cannot but feel that perhaps a course of mercurial injections would have succeeded where the iodids failed. But it is doubtful whether any better treatment could have been devised for either than the removal of the kidney. To say that every case of pseudo-tuberculous kidney which shows no typical bacilli in the urine, and in which the history suggests tuberculosis rather than syphilis, should be submitted to a test course of treatment is to enunciate a platitude that would very rarely yield any good results.

SYPHILIS OF THE PELVIC ORGANS

Syphilis of the Bladder.—The known cases of syphilis of the bladder are three, two of them published by Margouliès and one by Le Fur;[1] each of the three was manifestly syphilitic. Two of them showed sloughing ulcers near the ureteral orifices; the third, three small tumors in the same region; all three were promptly cured by antisyphilitic treatment.

[1] *Guyon's Ann.*, 1902, vol. xx, p. 385.

Paralysis of the bladder due to spinal syphilis need only be mentioned.

Syphilis of the Prostate and Seminal Vesicles.—Noguès [1] relates several suggestive cases, but nothing convincing.

Pelvic Cellulitis.—Fournier [2] has reported the following case:

Case XXXVIII.—"Last December I saw, in consultation, a patient, thirty-four years of age, said to have a tumor of the pelvis, which had been diagnosed as an inoperable sarcoma by three members of the Faculty. I was asked if there was not some possibility that this lesion was syphilitic in origin, though the patient denied all syphilitic antecedents.

"I found the patient pale, thin, debilitated, almost cachectic. For several months he had suffered a vague discomfort in the pelvis, with symptoms of progressively increasing vesico-rectal compression. . . . Recto-abdominal palpation revealed a voluminous infiltration lining the pelvic cavity and extending out of it in front and to the left side. . . . The tumor seemed to be reflected over the bladder while the rectum was free below, but was surrounded higher up by the pelvic infiltrate, and permitted the passage of a finger only with difficulty and pain. . . .

"No personal history or evidence of syphilis; but in the family a singular mortality. Of the patient's fifteen brothers and sisters, twelve died, most of them in infancy. . . . No stigmata whatever of hereditary syphilis. I asked that an ophthalmoscopic examination be made as soon as possible. Then—and here begins the picturesque part of the narration—only a few hours had passed after this consultation, when a gentleman called upon me, none other than the elder brother (thirty-six years old) of the patient. 'I come for two reasons,' said this gentleman. 'I bring you the information that you may feel almost certain my father was syphilitic; moreover, I myself am anxious, seeing the malady of my brother. I therefore beg you to examine me and to tell me whether I have anything to fear.' You may picture the haste with which I took advantage of this unforeseen

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[1] Noguès in Fournier, vol. ii, Part II, p. 667.

[2] *Bull. de l'acad. de méd.*, 1902, October 21.

circumstance. I examined this gentleman and searched him with the most scrupulous care for evidences of specific heredity; but upon him, as upon his brother, I found nothing—absolutely nothing to awaken any suspicion. The examination of his eyes remained. I sent him to the same ophthalmologist (Dr. Antonelli) who was to examine his brother. The following day I received from Dr. Antonelli a note, which I may sum up as follows: '(1) In your first patient (with the pelvic lesion) nothing but slight stigmata of the fundus—suspicion but not proof of hereditary syphilis. (2) On the other hand, his older brother shows positive stigmata of syphilis (chorio-retinitis).'"

We may sum up briefly the remainder of the narration. A course of mixed treatment showed marked results in ten days, and after two months the whole infiltrate had been absorbed; the patient had gained eight kilos, and was entirely well.

Syphilis of the Ovaries and Uterus.—Lesions of the internal genitals of the female come in the class of rare and obscure visceral lesions, sclerotic and gummatous, of which there are a few autopsy findings and a number of alleged cures by mixed treatment (e. g., of metrorrhagia [1]).

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[1] *Semaine méd.*, 1907, vol. xxvii, No. 8.

CHAPTER XXXV

HEREDITARY SYPHILIS IN UTERO AND IN INFANCY[1]

CHAPTER VI, on Syphilitic Inheritance, is an introduction to the study of hereditary syphilis, the details of which need not be repeated; we may add, however, the following figures derived from the statistics of authors who accept the theory of paternal heredity (as indeed almost everyone does).

Hochsinger reports 72 families in which the father alone was known to be syphilitic; thirty-five per cent of them had no stillbirths or abortions. To the remaining sixty-five per cent there there were born 110 children who were dead at birth or died soon thereafter, while 197 lived.

Fournier reports that *paternal syphilitic heredity* causes sixty-seven per cent of the children to be syphilitic and twenty-eight per cent to die; *maternal heredity* produces eighty-four per cent syphilitic and sixty-eight per cent deaths, while *mixed heredity* (both parents syphilitic) produces ninety-two per cent syphilitic children with 68.5 deaths.

Le Pileur reports a hospital experience of 567 pregnancies of syphilitic women with 174 abortions.

Thus the most striking effect of hereditary syphilis is its polymortality among infected children, while the dangers of

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[1] Reviewed by Dr. John Howland. The most important recent works upon this subject are E. Fournier, "l'Hérédo-syphilis"; and Hochsinger, in *Handbuch d. Kinderheilkunde*; Pfaundler and Schlossmann, 1906, vol. i, p. 894.

infection to the child, already dwelt upon in Chapter VI, are thus stated by Fournier:

"1. The danger of syphilitic inheritance varies greatly with the age of the disease in the parent, but shows a maximum—and that a marked one—corresponding to the first three years of infection.

"2. The maximum of this maximum corresponds approximately to the first year.

"3. Beyond the first three years of the disease there is a decreasing danger, but infinitely less marked.

"4. Syphilitic heredity has occurred in the sixth, the eighth, the tenth, the twelfth years, even the fifteenth, and perhaps the seventeenth, the eighteenth, and the twentieth, which appears to be the extreme limit."

The important features of hereditary syphilis may be subdivided as follows (Hochsinger):

1. Fetal syphilis.
2. Infantile syphilis (first three to six months).
3. Relapses in infancy (until the fifth year).
4. Late hereditary syphilis.

The importance of these stages decreases throughout the series. Thus fetal syphilis is fatal to about one half the infected children. Infantile syphilis kills fully half as many more in the first three months of extra-uterine life. The later relapses in infancy are less frequent, less severe, and rarely fatal, while the so-called "late" or "delayed" hereditary syphilis, which occurs after the fifth or sixth year, differs in no respect from tertiary syphilis in the adult. But the adult who has suffered hereditary syphilis in infancy bears upon his body certain evidences of the storm that has passed. These form a fifth division:

5. Stigmata of hereditary syphilis.

FETAL SYPHILIS

Pathology.—The pathology of fetal syphilis differs from the same process in the adult chiefly in its hyperacuteness. In the adult the disease localizes itself here and there in small areas and shows a special affinity to the skin and mucous membranes, and later to the nervous system, the underlying process being always specific arterial disease. But in the fetus the chief localizations are in the viscera, and secondly in the bones; the skin is immune until just before or just after birth. Moreover, the hyperacuteness of the process is shown in its diffusion throughout an organ, contrasting strikingly with the circumscribed visceral involvement in the adult.

The viscera most frequently and most markedly affected are, in order of importance, the lungs, the liver, the kidney, and the spleen.

"The type of congenital syphilis is a diffuse cell proliferation arising from the perivascular connective tissue of the smallest vessels, so that the solitary syphiloma is very rare in the fetus and the infant.

"The infiltration is exclusively a diffuse one arising from the smallest vessels in the organ, with a special tendency to later contraction and obliteration of these vessels. The perivascular proliferation begins in a sleevelike way in the adventitia and extends outward into the connective tissue, less often into the inner wall of the artery, thus obliterating it. In all the diseased organs one finds here and there dense cell accumulations visible to the naked eye and spoken of as 'miliary syphilomata' or 'gummata.'

"In the affected organs we find checked development of the parenchyma, abnormal development of the Malpighian bodies, persistent epithelial rests and canals, cystic development of the cortex of the kidney, etc. *Thus the hyperplasia of the connective tissue goes hand in hand with a hypoplasia*

of the parenchyma. These changes can often not be made out macroscopically, and we note only an increase in volume and density of the liver and spleen. The liver always shows considerable interstitial infiltration, the connection of which with the vascular system is very marked. One often finds pinhead, yellowish masses of necrotic liver cells surrounded by areas of marked congestion. This is an anemic necrosis. Similar conditions are seen in the kidney, the suprarenal and the epiphyseal line of the bones; indurative thickening of spleen and pancreas; lack of development of the kidney cortex with rudimentary Malpighian bodies and canal systems; in the lungs, pseudo-sarcoma, desquamation of the epithelia, pneumonia alba; . . . in the central nervous system, testicle and epididymis, similar diffuse perivascular infiltrations and hypoplasia of the parenchyma" (Hochsinger).

The placenta usually shows changes similar to those in the fetal viscera. Both the maternal and fetal portions of the placenta show the diffuse perivascular infiltration, so that the placenta is more voluminous and heavier than normal (it may reach one fourth or one third the weight of the child); the cord is also hard and considerably enlarged.

The distribution of spirochetæ in these various lesions has already been described. Marked changes of the viscera do not appear until after the fourth month of pregnancy.

Symptoms.—The fetus affected with syphilitic visceral lesions may come to one of three ends, as follows:

1. It may be aborted or stillborn.
2. It may be born manifestly syphilitic.
3. It may be born apparently healthy.

1. *The death of the fetus in utero* is often caused by hydramnia, depending upon syphilitic phlebitis of the cord; or by inanition from overwhelming visceral disease. Abortion usually occurs between the fourth and the seventh month, and the fetus thus aborted is usually dead and macerated.

Monstrosities are said to be relatively frequent in syphilitic abortion because of the interference with placental circulation.

2. *The typical syphilitic infant* presents a picture of pseudo-senility—thin, feeble, marantic, with a dull, *café au lait* color, a wrinkled, loose skin, upon which, even at birth, there may be an eruption; with the snuffles, with a hoarse cry,

FIG. 62.—CONGENITAL SYPHILIS; WIZENED FACE: MACULO-PAPULAR ERUPTION WITH EROSION OF LIPS AND LARGE PUSTULES ON BROW: BILATERAL INTERSTITIAL KERATITIS. (Musée St. Louis.)

with sunken and inflamed eyes, and perhaps with pseudo-paralysis or some lesion of the nervous system, the unfortunate infant forms a horrible picture of the ravages of hereditary syphilis (Fig. 62).

Among the whole number of syphilitic infants relatively few of those born alive present so marked a picture of disease. Not only may the infant be born with no actual syphilitic lesions apparent, but he may be in fairly good condition.

The skin is likely to be lacking in pinkness and fullness and the infant rather under weight. Thus Tarnier, among 52 syphilitic children, found 14 of normal weight (above 3,250 gm.) and 38 below it (Hochsinger).

3. *But the child with syphilitic lesions of the viscera may be born seemingly in good health.* Its weight may be ample, its syphilitic symptoms nil, its viscera and bones and skin apparently normal. Yet lesions of the disease develop shortly after birth, and, if these prove fatal, autopsy reveals extensive visceral involvement.

Jacquet mentions a healthy looking child "on whom were found two or three dark-red squamous spots on the knee, two similar ones above the eyebrow, and very slight labial fissure—and that was all; but the infant died nevertheless, and we found at autopsy abundant specific visceral lesions" (Berdal).

The direct evidence of the syphilitic visceral lesions is the enlargement of spleen and liver or of pseudo-paralysis from osteochondritis.

INFANTILE SYPHILIS

Although the lesions presented by the infant with hereditary syphilis are due to precisely the same protozoal cause as those in the adult, they are markedly dissimilar. Thus we have seen that, in the fetus, the viscera are the chief points of attack. In the infant the disease first strikes the mucous membrane of the nose, producing snuffles, and then shows itself by lesions upon the skin; but these skin lesions are not the same in character as those of early syphilis in the adult, nor are they by any means constant.

Intermingled with these symptoms, or succeeding them, there may be evidences of bone or visceral disease, or the little patient may show his malady simply by his thinness, his slow gain in weight, and his restlessness.

Taking up the early symptoms in order of clinical importance, we shall discuss:

Inflammation of the nose.
Inflammation of the skin.
Inflammation of the bones, joints, and muscles.
Inflammation of the viscera.
Mixed "infection" (pyogenic or tubercular).
Syphilitic dystrophies.

INFLAMMATION OF THE NOSE

"Snuffles" is the earliest and most constant symptom of hereditary syphilis, and one of the least characteristic.[1] The symptom is not quite that of snuffles in an older child, for at first there is little or no running from the nose, but only an obstruction to nasal respiration which makes the child breathe in a characteristic snuffly way and gravely interferes with nursing.

Hochsinger has tabulated 65 cases, of which 38 began at birth, 15 in the first month and 12 later than the first month.

The inflammation begins as hypertrophy of the nasal mucous membrane, at first a dry congestion beginning in the lower part of the nose, which soon goes on to ulceration and causes a profuse mucous discharge, often tinged with blood; epistaxis is most exceptional. The discharge is acrid, and excoriates the upper lip. Perforation of the septum is rare in infancy; but as a result of this coryza the development of the nasal bones is impaired or arrested and the bridge of the nose left sunken in a characteristic manner (later relapses of hereditary syphilis of the nose commonly cause perforation). Hence result the various nasal deformities so suggestive of syphilis.

[1] For it may be due to many causes.

The first clinical evidence of this nasal disease is *restlessness*, due to the fact that the little patient's sleep is constantly interfered with by the difficulty of breathing through its stopped-up nose. The *snuffly breathing* soon follows, and the infant, being unable to breathe through its nose, may be *almost totally unable to nurse*; its attempts at suckling and swallow-

FIG. 63.—SUNKEN NOSE (EN LORGNETTE) FROM DESTRUCTION OF THE CARTILAGE.

FIG. 64.—SUNKEN NOSE FROM DESTRUCTION OF THE BONY SEPTUM.

ing being frustrated by the rapidly recurring need of breathing, so that the process becomes a disheartening series of gasps, chokings, and regurgitations which result in the ingestion of little or no nourishment. Thus this relatively insignificant lesion may be the chief cause of a grave impairment of nutrition.

INFLAMMATION OF THE SKIN

So sensitive is the skin of little infants, especially if they be ill-nourished, to all manner of irritative eruptions, and so likely are the syphilitic skin manifestations to appear most markedly upon irritated regions, that it is often difficult to distinguish what part of the skin lesion is wholly syphilitic and what part simply irritative.

Hence the striking characteristics of the first skin lesions of hereditary syphilis, viz.:

1. Polymorphism.
2. Regional distribution.
3. Confluence.

THE LESION.—The first secondary lesion to appear in acquired syphilis is usually the general macular syphilid. But in hereditary syphilis the first (external) lesion to appear is usually not upon the skin (but in the nose), and when the skin lesions do appear they are neither generalized nor macular. The distribution is regional (see below), the lesion papulo-macular.

The papular eruption of hereditary syphilis is quite comparable to that of acquired syphilis (page 287), except that it is commonly intermingled with lesions of a macular character, and is itself often confluent.

Most of the lesions are papular or maculo-papular, while an occasional macule may be seen, though no general macular eruption is encountered.

POLYMORPHISM.—On account of the tenderness of the skin this papulo-macular eruption very readily takes on secondary changes. In dry regions the lesions tend to become squamous, or, if the eruption is severe, bullous or pustular;[1]

[1] Jacquet recognizes a special type of syphilid, the acneiform; it is quite rare. The lesion is a very small, umbilicated papule covered with a crust. The eruption tends to become confluent and to cover large areas.

while in moist regions the lesions are often confluent, erosive, and ulcerated.

Regional Distribution.—The eruption usually appears about the genitals, the buttocks, the flexor surfaces of the thighs, upon the palms and soles, and upon the lips and chin. The scalp, arms, and legs are somewhat less frequently affected, while the upper part of the trunk above the line of the diaper is relatively immune, doubtless because it is the dryest and thickest skin upon the infant's body.

Confluence.—The marked tendency to confluence shown by the early skin lesions of hereditary syphilis is due to the tenderness of the skin and the difficulty in keeping it clean; hence the confluent lesions are usually seen under the diaper, in various moist creases of the body, and about the mouth.

It is convenient clinically to distinguish five types of lesions:

1. Pemphigus.
2. Circumscribed maculo-papular syphilid.
3. Diffuse maculo-papular syphilid.
4. Lesions of special regions.
5. Tubercular or gummatous syphilid.

PEMPHIGUS

The earliest, one of the rarest, and the most ominous skin lesion of hereditary syphilis is pemphigus, or the bullous syphilid. It appears before birth or in the first week of extra-uterine life, less often in the two or three weeks following. Its typical situation is upon the palms and soles, whence it may spread over the rest of the body.

The eruption consists of large, copper-colored papules, upon which are set flaccid blebs containing serum and pus or blood. As these rupture they leave an eroded surface; or, if the ulceration is deep, greenish scabs take the place of the

ruptured bullæ. When this eruption occurs before the second week of extra-uterine life it is an accepted sign that the child will die.

DIAGNOSIS.—It may be readily distinguished from the pemphigus neonatorum, which never occurs before the second week, and usually much later, is often epidemic, occurs upon otherwise healthy children, and only most exceptionally appears upon the palms and soles.

CIRCUMSCRIBED MACULO-PAPULAR SYPHILID

This is the common and typical eruption of early hereditary syphilis. It consists of rounded, oval, or irregularly shaped spots, usually a dark red (though sometimes light) in color. Some of these spots are macular, others papular, others papulo-macular, while others still are annular, the periphery of the affected area being elevated higher than its center; in diameter they average 1 cm., though they vary greatly. Their surface is tense, with a satin gloss, and may be covered with little scales or surrounded by a scaly collar.

In moist creases of the infant's skin the papules become eroded, and may even become condylomatous; while, wherever there is much irritation (especially beneath the diaper and about the mouth), they tend to run together and to ulcerate. Pustulation is, in the child as in the adult, an evil omen.

DIFFUSE MACULO-PAPULAR SYPHILID

The diffuse maculo-papular syphilid, not so common as the circumscribed form, but often associated with it, never begins in intra-uterine life, usually appears within the first three months after birth, and may relapse during the first year. It occurs most frequently beneath the diaper, on the palms and soles, and on the face. It has the characteristics of the

circumscribed papular eruption, being due to confluence of a number of smaller papular lesions. When it occurs under the diaper it usually stops in front at the upper part of the thigh,

FIG. 65.—MACULO-PAPULAR SYPHILID IN HEREDITARY SYPHILIS. (Wilson.)

and extends behind over the flexor surfaces almost to the knees; while in the genital creases the skin is broken by eroded, ulcerated, or condylomatous areas.

On the palms and soles the eruption looks thicker, more tense and shiny, and yellowish. Desquamation of large patches of thick epidermis is common here. Upon the face the diffuse maculo-papular syphilid is usually confined to the region about the mouth, covering the upper lip, the chin, and sometimes extending down on to the neck.

LESIONS OF SPECIAL REGIONS

Erosions of the Lips.—Eroded mucous papules are very common upon the vermilion border of the lips, and are promptly fissured and transformed into deep-red, oozing cracks, surrounded or covered by a little crust. They are usually multiple, and are commonest at the angle of the mouth. They are extremely painful, and interfere gravely with suckling. As they heal they leave marked radiating linear scars upon the lips, which in later years form one of the most striking and reliable stigmata of hereditary syphilis.

Ano-genital Lesions.—These are usually associated with dry or scaly papules elsewhere in the body, and are themselves confluent, erosive, or ulcerated, rarely condylomatous, and still more rarely pustular. But in the relapses of early childhood condylomata are very common.

Onychia and Paronychia.—The typical nail of the syphilitic infant is raised high in the center and depressed laterally, as though it had been pinched by a forceps (Holt). It resembles a claw rather than a nail. This clawlike nail is quite common, and strongly suggestive of syphilis. The condition usually affects all the nails.

Such nails are often undermined by a species of paronychia, so that they are readily detached.

Lesions of the Scalp.—Inasmuch as the infant loses its first hair a few days after birth, one cannot say that syphilitic defluvium at this time is common. Certainly the moth-eaten

poll is quite uncommon in infancy, and more extensive baldness (which has the same characteristics as in the adult) very rare.

The papular eruption upon the scalp, instead of forming small, disseminated, crusted lesions, as in the adult, may produce an extensive incrustation closely simulating eczema; but the syphilitic lesion is distinguished by the fact that removal of the scabs leaves an intact, though infiltrated area beneath it, while removal of an eczematous crust leaves the Malpighian rete exposed.

TUBERCULAR AND GUMMATOUS SYPHILID

It is not always possible to distinguish between a furuncle and a syphilitic tubercle or gumma; indeed, these lesions are classed as " furuncular syphilids " by Taylor, while their syphilitic nature is entirely denied by Jacquet; they are, however, accepted by most authorities, and Carpenter [1] states that among 364 syphilitic eruptions seen by him during the first year of life there were 70 cutaneous gummata, 34 of which were associated with other lesions of the skin.

Among these 70 cases, 4 occurred in the first month, 21 in the second and third, 14 in the fourth and fifth, 11 in the sixth and seventh, and only 15 in the remaining five months. The lesions are those of syphilitic tubercle and subcutaneous gumma; but they have an especial tendency to become pustular, and so remain for a considerable time before ulcerating. They come out in successive crops, and disappear under mercurial treatment.

LESIONS OF MUCOUS MEMBRANE

Excepting the nose, the lips, and the anus, lesions of the mucous membrane are extremely rare. The ulcers that occur

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[1] "The Syphilis of Children," London, 1901.

in the mouth are alleged by many authors to be aphthous, not truly syphilitic.

Aphonia is frequent, however, and is apparently due to infiltrative and ulcerative laryngeal lesions similar to those seen in the adult. The familiar "hoarse cry" of the syphilitic infant is quite as suggestive and far more common than the hoarse voice of the syphilitic adult.

The intestine may be inflamed, and may show *post mortem* a diffuse thickening of the mucous membrane with ulceration.

INFLAMMATION OF THE BONES, THE JOINTS, AND THE MUSCLES

While any of the lesions of bone that occur in the adult may also occur in hereditary syphilis, the two striking and distinctive characteristics of bone syphilis in the infant are osteochondritis and periostitis.

The former begins before birth, the latter shortly after birth; a difference explained by Hochsinger as due to the fact that syphilis attacks the most actively functionating region, which is the epiphyseal junction in intra-uterine life and the periosteum thereafter. Both types are in their milder manifestations extremely common.

Osteochondritis.—Mild syphilitic osteochondritis, causing slight distortion and enlargement of the bone at the epiphyseal line, is very common. Its lesions can be accurately studied only by the X-ray.

The knee and the elbow are most commonly and most markedly affected; and the lesion consists in a syphilitic infiltration of the diaphysis at its junction with the epiphysis, which results in a thickening of the bone at this point and a marked irregularity at this line of junction, as is well shown by the accompanying radiograph (Fig. 66). Very rarely this lesion progresses so far that the granulomatous tissue, disin-

tegrating the epiphyseal cartilage, produces an epiphyseolysis. This rare lesion, appropriately called "syphilitic pseudo-paral-

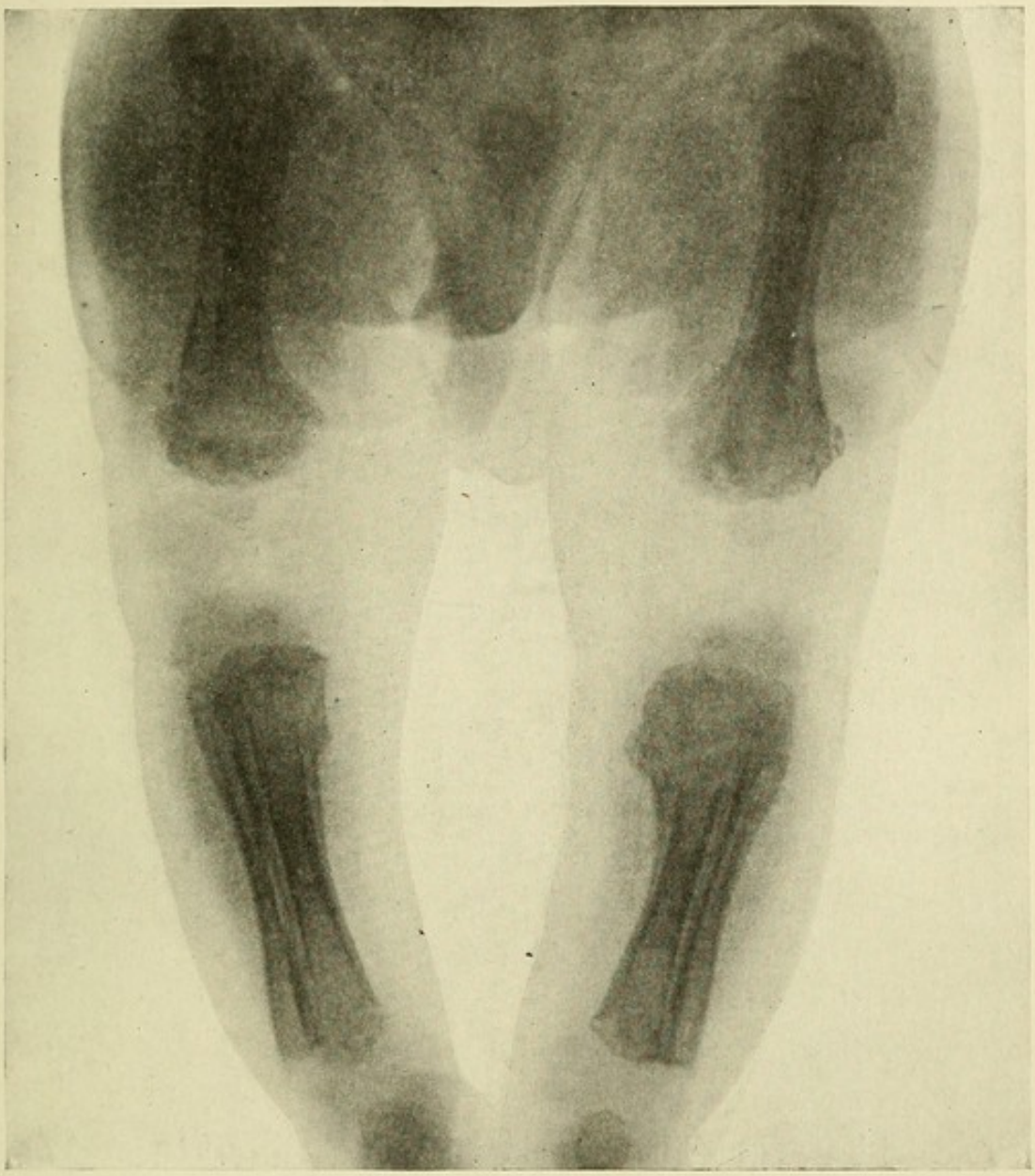

FIG. 66.—OSTEO-CHONDRITIS (PSEUDO-PARALYSIS) OF BOTH KNEES. Note the marked thickening and irregularity of the lower ends of the femoral and upper ends of the tibial diaphyses (compare normal knee in Fig. 56). (Case of Dr. La Fetra. Skiagraph by Dr. Caldwell.)

ysis of the newborn," usually affects the elbow and is congenital; it may be polyarticular.

"Its essential character is an inertia, an impotence of the

limbs, which may be partial or general, incomplete or absolute. . . . For an accurate study of the state of the child and of the degree of its disability, it must be held up by the axillæ while its limbs are being examined. When the affection is marked the child's limbs seem dislocated and hang like bell-clappers, limp and inert. Pinch the skin and the infant's muscles contract sharply, but its limbs are little moved by this contraction; yet they offer no resistance to any movement imparted to them by the observer. . . . Sometimes one feels crepitation, and in certain infants the limbs are increased in size in the region of the joints; fluctuation may even be made out" (Parrott).

This epiphyseolysis occurs only when there is marked visceral disease, and is an evil omen.

Periostitis.—The periosteal lesions of early syphilis are usually most marked upon the skull, though similar lesions may affect any bone.

The lesions of syphilis in the skull are, in order of frequency—infiltration and softening between the tables and in the sutures; diffuse periostitis of the frontal or parietal bosses, causing an enlargement which, if frontal, produces the so-called "Olympian" brow; if lateral, a considerable widening of the transverse diameter of the skull (natiform skull). Less frequent is the rarefying ostitis with tendency to gummatous degeneration. Finally, the skull may be affected secondarily by hydrocephalus.

American authorities consider it unsafe to accept cranial deformities as evidence of hereditary syphilis unless supported by other typical signs. Parrott's nodes and cranio-tabes, though sometimes spoken of as syphilitic, are actually of rachitic origin.

Dactylitis.—The dactylitis previously described (page 441) is far more common in hereditary than in acquired syphilis.

Ribs.—Circumscribed nodes are said by Hochsinger to be common in the lateral thoracic region at the level of the fourth or the fifth rib.

Arthritis.—At this early age the joints are very rarely involved, except as a complication of severe osteochondritis.

Myositis.—Syphilitic inflammation of the muscles usually affects the biceps or the sterno-mastoid, originates in periostitis, and causes a fixed cramp of the muscle (cf. page 448). Hochsinger states that the so-called muscle gummata are usually hematomata.

INFLAMMATION OF THE VISCERA

Lymph Nodes.—Lymphadenitis is not typical of hereditary syphilis. It is common when there is a multiplicity of syphilitic lesions; marked epitrochlear adenitis is of some diagnostic importance. The presence of other enlarged nodes is, as a rule, directly attributable to adjacent lesions.

Testicle.—Syphilitic sclerosis of the testicle is extremely common in stillborn children. It is rare in those that survive.

Inasmuch as the size of the testicle in infancy varies between wide limits while syphilis of the organ often does not greatly alter its size, the clinical manifestation of the lesion is usually an *increase in hardness*, which may be masked by hydrocele. Testis and epididymis may be involved separately or together. The lesion may be unilateral or bilateral. Hydrocele is quite common; fungus is rare. The lesion may pass undiscovered. It usually leads to atrophy of the testicle, and great stress is laid by certain authors upon the existence of this atrophy in adult years as an evidence of hereditary syphilis.

Diagnosis.—Inasmuch as tuberculosis of the testicle may occur shortly after birth (Dreschfield has recorded a congenital case), and produce lesions macroscopically similar to those of

syphilis, the diagnosis is not always possible; but of the two diseases syphilis is by far the more common.

Liver and Spleen.—Hereditary syphilis of the liver and spleen are so intimately associated that it seems proper to consider them as one. Syphilitic changes may be found in these organs in many instances *post mortem*, although no evidence of such disease was apparent during life. The pathological changes have already been described (pages 470, 506).

Clinically, the manifestation of these lesions consists in an enlargement of the affected organs. This enlargement is noted in thirty per cent to forty per cent of cases in the first three months of life; and in almost every instance enlargement of the spleen implies enlargement of the liver, and *vice versa*.[1] The enlargement, though usually slight, may be considerable, and, exceptionally, one may identify thickening of the edge and irregularity of the outline of the liver, as in acquired syphilis. Syphilitic jaundice is extremely rare in early infancy. Ascites does not occur until a later age.

Lungs.—The pathology of pulmonary syphilis has been described elsewhere (page 462). These lesions are so commonly fatal that they can scarcely be said to give any clinical manifestations.

Nervous System.—*Restlessness and sleeplessness* are striking symptoms of early hereditary syphilis, and have been referred to meningeal irritation, but are more probably due to nasal obstruction. Though these early cases may show *post-mortem* lesions throughout the nervous system, they show during life no evidence of peripheral neuritis or of syphilis

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[1] Hochsinger found that splenic enlargement accompanied hepatic enlargement in forty-four out of forty-six cases. He also states that only three per cent infants showing enlargement of the spleen were not syphilitic. Carpenter states that when the spleen is enlarged the liver is also enlarged in four fifths of the cases.

of the cord; while symptoms of syphilitic meningitis other than restlessness, sleeplessness, and convulsions are extremely rare. The only exception is *hydrocephalus*. This, whether acute or chronic, internal or external, is often syphilitic, and develops with the first outbreak of symptoms (in the first or second month). So common is this association of hydrocephalus and syphilis, and so hopeless is the former condition when not syphilitic, that it is most excellent practice to treat every infant with hydrocephalus as though it were syphilitic.

Eye.—Lesions of the eye are extremely rare at this early date. Iritis, parenchymatous keratitis, and optic neuritis have been noted, while it is highly probable that many cases of choroiditis begin very early in life.

Ear.—Extension of the nasal catarrh to the middle ear, resulting in acute otitis media, is not uncommon. Paralysis of the nerve or inflammation of the labyrinth usually occurs at a later date.

MIXED INFECTION

As a frequent and grave complication of early hereditary syphilis it shows itself chiefly in four types:

1. Mixed infection of the skin.
2. Mixed infection of the respiratory tract.
3. Mixed infection of septic type.
4. Tuberculosis and rachitis.

Skin.—Mixed infection of the skin shows itself in the frequency of pustular and ulcerating lesions, while pseudo-furunculosis is due in most instances to pyogenic bacteria.

Respiratory Tract.—The discharges of syphilitic coryza swarm with spirochetæ and with all manner of bacteria. Hence the inflammation may spread to the ear, causing otitis media, or to the lung, causing broncho-pneumonia. This latter complication accounts for many deaths.

Sepsis.—The petechiæ and other hemorrhagic skin lesions that occur in certain malignant cases of early hereditary syphilis are, doubtless, evidences of acute septicemia.

Tuberculosis and Rachitis.—The relation between rachitis and syphilis has long since been determined. Parrott believed and taught that every rachitic child was syphilitic; but subsequent investigation has clearly shown that this is not the case, that syphilis is only one among many potential, debilitating causes of rickets.

The same is true of tuberculosis. Sergent [1] has recently collected much evidence in the matter, and, with a wealth of authoritative quotations, enforces his conclusions that " la syphilisation du père a préparé le terrain pour la tuberculisation du fils "—syphilis in the father prepares the soil for tuberculosis in the son."

This aphoristic statement expresses the actual condition not quite accurately. It is necessary to distinguish as follows:

1. Syphilis in the father, if severe or if allied with other debilitating influences, such as drink, deprivation, or debauch, prepares the father's own soil for tuberculosis.

2. Syphilis transmitted from parent to child—hereditary syphilis—exercises the same predisposing influence magnified by the difficulty of eradicating the disease from the vulnerable tissues of the newborn.

3. But the non-syphilitic child of a syphilitic parent is not predisposed to tuberculosis, unless by such privation or neglect as would so predispose it in any event.

Although tuberculosis and rachitis are frequently attendant upon syphilis a little later in life, they scarcely figure in the first few months.

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[1] "Syphilis et tuberculose," Paris, 1907.

SYPHILITIC DYSTROPHIES

The interference with nutrition due to hereditary syphilis shows itself in two ways: First, and most frequently, by killing the child within a few days of its birth; secondly, by impairing its nutrition, but not killing it.

The first evidence of impaired nutrition may be the puny, decrepit state of the child at birth; or the child may be born apparently healthy, and promptly lose weight and strength in spite of a good digestion and an abundant supply of mother's milk. Such infants, though they may survive for a time, are carried off either by the cachexia of the disease itself or by such complications as convulsions, diarrhea, or broncho-pneumonia during the first year of their lives.

From these various causes something like nine out of ten bottle-fed or hospital babies fail to survive, though in private practice hygiene, human milk, and intelligent medical care cut down the mortality considerably.

But beyond this obviously syphilitic toxemia there is a set of vague conditions, such as go to produce congenital deformities, e. g., club-foot, harelip, etc., or to arrest development causing the child to attain puberty very late, to have its sex characteristics but slightly developed, and to remain childish both in mind and body. Idiocy and epilepsy are said to be exceptionally frequent in otherwise apparently healthy children of syphilitic parents.

In how much these conditions are due to a real syphilis of the child and in how much to an inherited tendency from the parent (similar to that of alcoholism, for instance) it is difficult to say; but it is perhaps safer to assume that in most instances the child is actually syphilitic, and does harbor or has harbored the spirocheta. For there seems no adequate reason to suppose that syphilis of the parent, unless it is gravely impairing the parent's own nutrition, should have any very obvi-

ous effect upon the nutrition of the sperm cell and of the infant. It would seem quite probable, however, that many cases of impaired nutrition attributed to syphilis should be attributed to a concomitant parental alcoholism or to other deteriorating forces.

CHAPTER XXXVI

HEREDITARY SYPHILIS; RELAPSES; LATE LESIONS; DIAGNOSIS; PROGNOSIS; TREATMENT[1]

RELAPSES IN EARLY CHILDHOOD

After the first outbreak of hereditary syphilis in the first three months of the disease the affection may remain in abeyance or may relapse. Relapses are peculiarly liable to occur in the first and second year of life, also from the sixth to the eighth year, and from the time of puberty until the twenty-fifth year. These periods are, of course, vague and undefined. There may be no intervals between relapses and there may be years.

Relapses of early childhood commonly occur in the first two years, but may continue for several years longer. In these relapses the skin manifestations, while remaining the same as those already described, are likely to be less diffuse, less polymorphous, and rather more tertiary in type; copper colored, or tubercular, or furuncular. Condylamata, however, remain frequent—even more frequent than during the first outbreak.

Of the lesions of the nervous system, hemiplegia and epilepsy are the most important, while chorio-retinitis and optic neuritis are quite common.[2]

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[1] Reviewed by Dr. John Howland.

[2] Among the visceral manifestations which Hochsinger mentions are the stridor thymicus caused by pressure of the enlarged thymus on the trachea, paroxysmal hemoglobinuria, hematoporphyriuria; and as a result of splenic syphilis, the anemia pseudo-leukemia infantum; this occurs at the end of the first and during the second year.

But the important feature of this period is the confusion of rachitis and syphilis. For, inasmuch as rachitis does not show itself until after the child is three months old, the splenic enlargement and bone deformity of early syphilis do not lend any color to the theory of rachitis until the first three months have passed. But since an enlarged spleen, manifest malnutrition, and very similar bone lesions may be the only manifestations of either disease, it is small wonder that they should be confused with each other. Indeed, in certain cases it may be impossible to decide whether the child is syphilitic or rachitic, or both syphilitic and rachitic, if its first outbreak of syphilitic symptoms has been so slight as to be overlooked (page 537).

LATE HEREDITARY SYPHILIS

From the sixth to the eighth year, and again at about the time of puberty, hereditary syphilis may relapse; relapses excepting at these times are infrequent.

The limit of duration of hereditary syphilis is quite as indefinite as that of acquired syphilis. It may surely last throughout the patient's lifetime, though after adult life is reached its lesions are extremely rare, and it may be clinically impossible to say whether these lesions are due to a hereditary or to an acquired disease.

The arbitrary limit of twenty-six years set by Fournier for the duration of hereditary syphilis is, in general, the extreme limit; indeed, lesions after puberty are extremely rare. Whether late hereditary syphilis may occur in an infant who has shown no early manifestations of the disease has long been a matter of controversy; and the question will never perhaps be more satisfactorily answered than by the statement that children showing lesions of late hereditary syphilis usually give a history of snuffles and of lesions in childhood; but yet this history may be wanting. Whether, in such event, the early symptoms are

actually skipped or simply overlooked, it is not possible to state. The former hypothesis is possible, but the latter is far more probable.

There is no more call to make a special class of those cases of late hereditary syphilis that give no history and show no evidence of early lesions than there would be to make a like distinction in acquired syphilis.

The active syphilitic lesions of late hereditary syphilis are, with but very few exceptions, quite the same as those that occur in late acquired syphilis. They are, in other words, the tertiary lesions of the disease occasionally intermingled, as in acquired syphilis, with certain late secondaries, especially those of the palms, the soles, and the tongue. Certain classes of lesions are, however, somewhat more common in late hereditary than in late acquired syphilis. Such are the various lesions of the eye, deafness from internal ear disease, perforation of the septum and palate, and diffuse periostitis of the long bones. The joints are relatively often affected, and the danger of mistaking a pseudo-tubercular syphilitic knee in the young for the tubercular "white swelling" must be borne in mind.

The chief exceptions in which late hereditary syphilis differs from late acquired syphilis are diffuse periostitis and parenchymatous keratitis. Besides these two lesions there are, however, many stigmata of early hereditary lesions, which will be taken up in the following section.

Diffuse Periostitis.—The periosteal changes of late hereditary syphilis are the same as those of acquired syphilis excepting in their distribution. The hereditary lesions, are, however, often much more diffuse than the acquired ones, sometimes extending over the whole length of a long bone. This diffuse process is most common in the tibia, to which it imparts a peculiar and pathognomonic form. The shaft of the bone is thickened in all its diameters, but especially antero-posteriorly;

so that, at first glance, the bone appears to be bowed forward; but palpation reveals that this bowing is simply a thickening around it of new bone without any deviation in the axis of

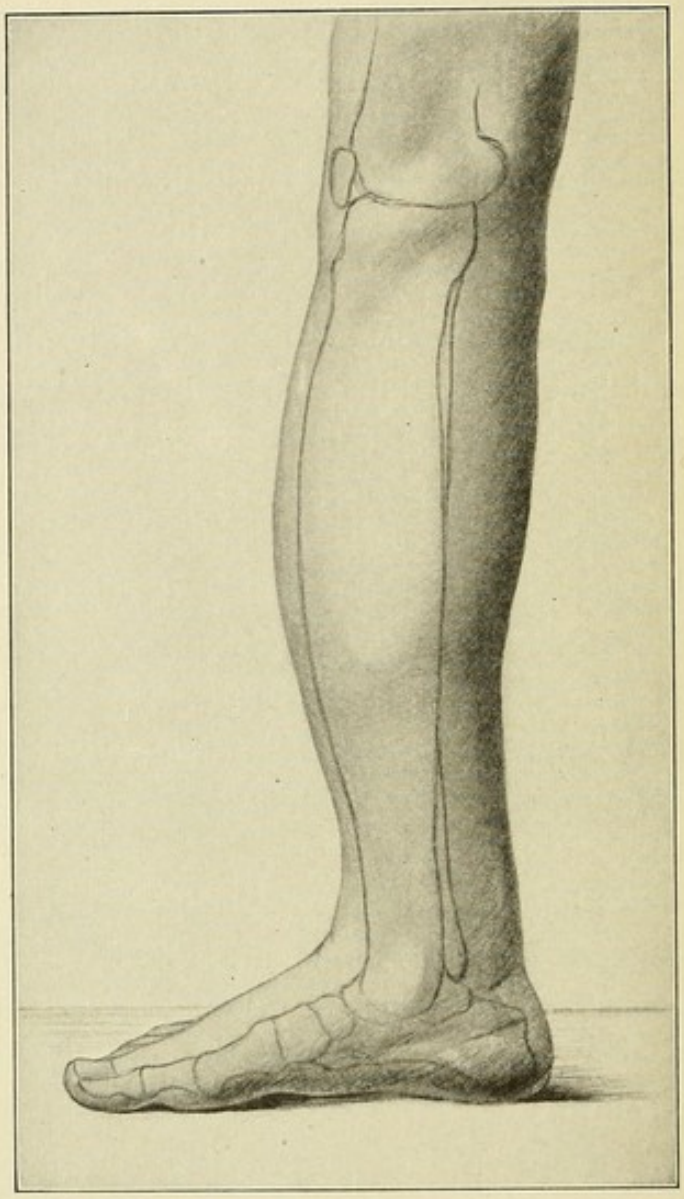

Fig. 67.—Saber Tibia (Diagrammatic).

the bone itself (in marked contrast to the bowed shins of rickets).

The new bone is fairly smooth, perfectly dense and insensitive, and so ensheathes the original bone as to completely conceal it. The anterior edge of the tibia is markedly rounded.

Its epiphyses, especially the upper one, may also be enlarged; and if there has been early disease at the epiphyseal junction, the limb may be shortened by premature ossification, or lengthened by excessive ossification. This bowed tibia is appropriately termed the " saber-blade " tibia (Figs. 54, 67) ; it is absolutely pathognomonic of syphilis.

Parenchymatous Keratitis.—Parenchymatous keratitis, though it may occur in the first months of extra-uterine life or even before birth (Parinaud), and as late as the thirty-sixth year (Huguenin), is common only between the eighth and the fifteenth year (Hutchinson). It is one of the frequent manifestations of hereditary syphilis, occurring very frequently (fifty-nine per cent, according to Huguenin). On the other hand, it is by no means pathognomonic of syphilis. Though Hutchinson places great weight upon it as a diagnostic sign, careful investigation reveals other evidences of syphilis in not much more than one half the cases (thirty-six per cent, according to Alexander; fifty-five per cent, according to Michel). Hence it is not of itself an absolute proof of the existence of syphilis.

Moreover, interstitial keratitis may occur in acquired syphilis. My father has seen two instances of this.

The lesion is almost always bilateral, although it begins first in one eye. It begins painlessly with a diffuse opacity (usually) in the center of the cornea. This opacity gradually increases in size, but does not ulcerate; and pericorneal congestion soon appears.

After some three weeks the whole cornea has become involved and looks like a piece of ground glass. The pericorneal injection has markedly increased, and the cornea itself may be so congested as to assume a pinkish hue. This is often the first sign of beginning resolution.

After six weeks or so from the beginning of the trouble the whole process begins to diminish. The congestion disap-

pears and the opacity is more or less resolved. It very rarely disappears completely, but usually leaves a disfiguring central scar large enough to impair if not to destroy vision.

The subjective symptoms are photophobia and obstruction of vision. The permanency of this obstruction cannot be foretold; but if a central scar remains it is, of course, permanent.

Such complications as iridokeratitis and opacities of the lens or other adhesions are fairly frequent.

Glaucomatous complications are rare.

TREATMENT.—The local treatment consists in protecting the eyes by smoked glasses and the iris by instillations of atropin (page 415).

Mercurial treatment should be given, though it is by no means proven that this has any beneficial effect. The tension of the eye must be carefully observed lest glaucoma intervene.

The application of hot compresses to the eye for a half hour (renewing them every two or three minutes), five or six times a day, is said to encourage healing.

STIGMATA OF HEREDITARY SYPHILIS

The stigmata of hereditary syphilis are the scars of syphilitic lesions or the deformities or peculiarities in development left by syphilitic lesions occurring in infancy or in youth. They are more or less pathognomonic of the disease, though few of them (only the radiating fissures of the lips and the typical saber tibia) are absolutely so. Yet many of them are presumptive evidence of the greatest importance.

Only these important stigmata need be described. Other developmental defects indirectly due to syphilis, such as rickets, idiocy, cleft palate, club-foot, etc., may be excited by so many other causes, known or unknown, that though we may in specific instances acknowledge their derivation from syphilis, we

may not infer from them that the patient is syphilitic, i. e., their importance as syphilitic stigmata is negligible.

Moreover, it is to be noted that most of these stigmata are only evidence that the patient has had syphilis in childhood or in infancy; whether acquired or hereditary they do not decide. Yet certain of them (especially the lip fissures and the dental deformities) derive from lesions of so early a date that they point to an hereditary taint with almost absolute certainty.

The chief stigmata of hereditary syphilis may be classed as follows:

1. Hutchinson's triad, consisting of dental, ocular, and auditory stigmata.
2. Bone stigmata.
3. Skin stigmata.
4. Visceral stigmata.
5. Constitutional stigmata.

Of these, the first three are of prime importance, the two last are rarer and less characteristic.

Excepting Hutchinson's triad, these stigmata have already been described in connection with the lesions that cause them. The chief bone stigmata are found in the skull (frontal or lateral bosses, asymmetry and irregular development, hydrocephalus), in the nose (sunken bridge), and in the long bones (diffuse periostitis, saber tibia, and such scars of epiphyseitis as epiphyseal enlargements, and arrested or excessive growth of the bone). The skin stigmata do not differ from those of acquired syphilis, excepting the important lip fissures. The only notable visceral stigma is atrophy of the testicle. The constitutional stigmata consist of a gray bloodless skin and a slowness of physical and intellectual development that may amount to "infantilism" or idiocy (page 525).

HUTCHINSON'S TRIAD

Most important among the stigmata of hereditary syphilis are those lesions grouped as a triad by Jonathan Hutchinson, Sr., from whom they very appropriately derive their title.

" Hutchinson's triad " is commonly taken to mean the association of—

1. Notched and pegged upper, central, permanent incisors.
2. Interstitial keratitis or the scar thereof, and
3. Nerve deafness.

But though these three are the most striking of the lesions in question, it is preferable to employ the term in a looser sense to cover all.

1. Dental stigmata.
2. Ocular stigmata, and
3. Aural stigmata.

Dental Stigmata.—The dental stigmata of hereditary syphilis are due to arrested development of the teeth in the process of formation. Permanent incisors and the sixth-year molars are the teeth most often affected because they are in process of formation during the first three months of extra-uterine life, i. e., at the very time when hereditary syphilis shows its greatest virulence.

Hereditary syphilis may, however, impart to the teeth almost any malformation in size, position, and shape. Fournier, who has made a special study of this matter, describes some ten varieties of syphilitic dental deformities. But we need not follow thus far, for these deformities are not in themselves in the least pathognomonic. They suggest syphilis only when they are associated with other more typical lesions. But there are two striking dental deformities, Hutchinson's teeth the more typical, Fournier's the more common.

Hutchinson's Teeth.—This deformity, though not peculiar to any tooth, is usually confined to the permanent, upper,

median incisors. It is not absolutely pathognomonic of syphilis (as Hutchinson thought), for several authors have reported cases of syphilitic teeth in non-syphilitic persons, and the ac-

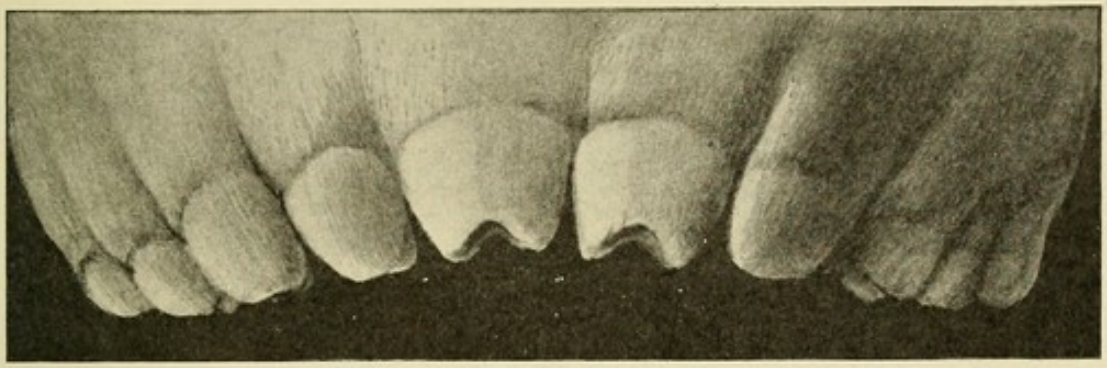

FIG. 68.—HUTCHINSON'S TEETH. (From a cast.)

companying illustration (Fig. 68) is drawn from a cast of such a case taken by my father.

Typical Hutchinson's incisors are abnormally small, pegged (i. e., tapering inward from above downward), and show a

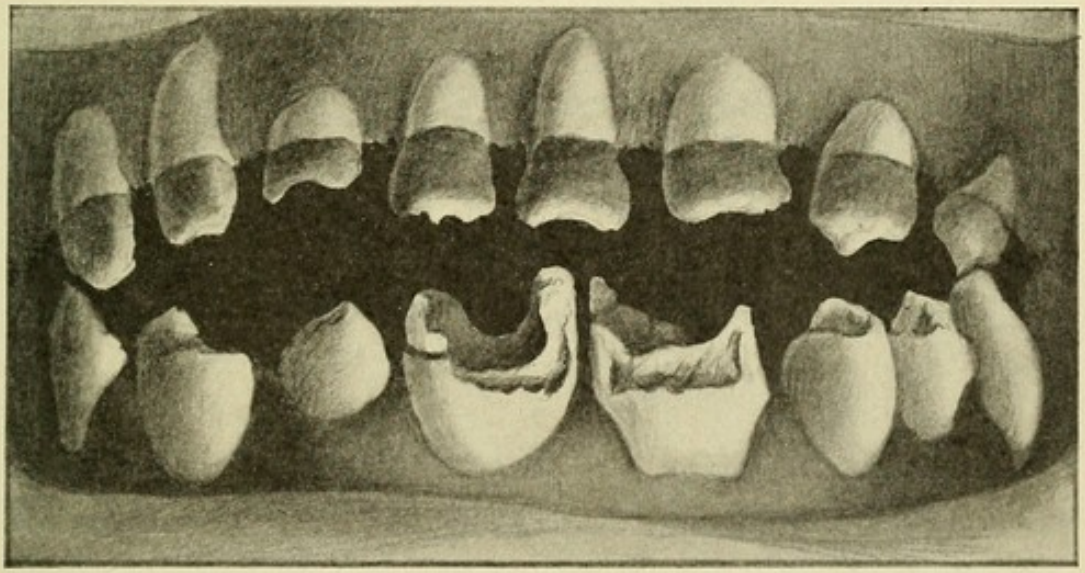

FIG. 69.—IRREGULAR AND DECAYED TEETH OCCURRING IN AN HEREDITARY SYPHILITIC, BUT IN NO WAY CHARACTERISTIC OF THE DISEASE.

peculiar erosion of the free border. This erosion forms an absolutely regular crescentic bevel of the anterior surface of the free border of the tooth. When the tooth erupts its free

border is quite rough, but this roughness rapidly wears away, leaving a typical Hutchinson's tooth, which retains its characteristics for some fifteen or twenty years. But by the time the patient reaches the age of twenty or twenty-five the bevel edge is all worn off. Hutchinson's teeth are almost always symmetrically bilateral, and affect the permanent, upper, central incisors, which are often somewhat displaced, usually converging toward each other.

Fournier's Teeth.—Fournier has described a less characteristic deformity of the sixth-year molar, which he says is even more common than Hutchinson's teeth.

The deformity consists in an erosion of the summit of the tooth. About three fourths of the tooth is entirely normal; the terminal fourth "is diminished in all its diameters, irregular, eaten, as though atrophied, and separated from the healthy part by a circular ridge." After five or ten years of use this eroded crown wears away, leaving a short tooth with a smooth top, to which Fournier attaches considerable diagnostic significance.

Ocular Stigmata.—Of these, the most striking, though by no means the most pathognomonic, is interstitial keratitis or the scars thereof. Iritic adhesions, though rarer, are almost equally suggestive. But the most typical stigmata are the white scars and the deposits of pigment left by syphilitic choroiditis (page 416). Ocular palsies are relatively rare in young syphilitics, but they, too, may leave traces suggestive of the disease.

Auditory Stigmata.—Deafness may result either from lesions of the middle ear or from those of the inner ear or of the nerve. Middle-ear deafness is only indirectly due to syphilis, and is much more often due to other causes.

But internal ear or "nerve" deafness (page 395) in children or young persons is almost exclusively due to syphilis.

DIAGNOSIS OF HEREDITARY SYPHILIS

In order to disclose a case of hereditary syphilis one may have to conduct a diagnosis along three lines:

1. Examination of the infant and study of its history.

2. Study of the history of previous or subsequent births or miscarriages, and

3. History of the father and mother in reference to antecedent syphilis.

Needless to say, this investigation must often be carried on with the greatest discretion and with the most delicate regard for the feelings of the parents as well as for their ignorance. Yet when one is satisfied that an infant is syphilitic and infectious one cannot compromise the issue, but must, for the safety of the community, declare to the parents that it is suffering from an infectious disease, though it may be discreet to withhold from the mother at least its "venereal" nature.

Diagnosis in Infancy.—During the first three months the symptoms most likely to be discovered are snuffles, erosions about the lips and anus, skin lesions, bone lesions—especially slight irregularities or enlargements at the ends of the diaphyses of the long bones, enlargement of the liver and spleen, and the pinched, clawlike nails; while *as early signs, restlessness and unaccountable loss of weight* are very suggestive of syphilis. Later, nodes on the long bones, condylomata, and perhaps tertiary lesions of the throat, with gummata or some one of the visceral lesions mentioned, may help fill out the picture. Of these lesions, the only ones likely to leave an imprint in later years on the parents' minds or the child's body are the bone lesions, the lip erosions, and perhaps a vague history of snuffles, feebleness, and a rash in infancy.

Examination of the Family Record.—Every syphilitic family may be depended upon to give a history of at least one miscarriage or one death in infancy. From a diagnostic point

of view, however, miscarriages, unless many times repeated, are not strong presumptive evidence, since they occur from all manner of causes; and it might equally be said that every family, whether syphilitic or not, gives a history of at least one miscarriage; with this difference, however, that the syphilitic family has the miscarriage at the head of the list.

Deaths in infancy are far more striking presumptive evidence, and a combination of several miscarriages, stillbirths, and deaths in infancy is peculiarly suggestive.

History of the Parents.—In about one quarter of all cases no syphilitic history can be obtained from the mother. In many cases no syphilitic history will be obtained from either parent. Ignorance or fear prompt the denial. In the case of the mother the investigation should be carried out solely with regard to the symptoms of the disease, though the father may be flatly questioned as to whether he ever had a chancre.

Diagnosis in Later Years.—In later years we have—besides the actual lesions of syphilis, which may be present upon the child's body, and the history of past syphilis in the child, in his brothers and sisters, or in his father and mother—a set of lesions known as the stigmata of hereditary syphilis, which may help materially in the diagnosis (page 532).

The presence of such conditions as adenoids, club-foot, or idiocy cannot be fairly attributable to syphilis, as many writers have attempted to do.

Rickets and tuberculosis, though common complications of hereditary syphilis, are by no means reliable stigmata.

Of the five types of important stigmata previously described, the radiating scars over the lips (resembling the radiating wrinkles seen in the aged) are alone accepted as pathognomonic by Hochsinger. Yet a typical saber-blade tibia, a pair of Hutchinson's teeth, or the scars of an interior or disseminated irido-choroiditis are almost equally convincing.

But one rarely has to depend upon one lesion. A careful

examination, if it reveals anything at all, usually reveals enough strongly suggestive stigmata and history to produce certainty in the mind of the examiner.

PROGNOSIS

The general prognosis of hereditary syphilis is best illustrated by the figures of Dr. James Nevins Hyde, who records 1,700 pregnancies, of which only 1,121 went to term, while of these, 916 died within the first year. In private practice one can do much better than this, though how much better is a matter of doubt.

As already stated, pemphigus, visceral disease, and osteochondritis are peculiarly ominous.

In infancy death occurs from convulsions, pneumonia, and digestive complications, and the surviving children are relatively hard to rear and subject to intercurrent disease.

The three important elements in prognosis are:

1. The more or less undeterminable condition of the viscera.

2. The food; for, as a general rule, breast-fed babies have a fair chance of surviving, while bottle-fed babies have a very small one.

3. Intelligent administration of mercury.

TREATMENT IN INFANCY

The treatment of hereditary syphilis is based upon the same rules as is that of acquired syphilis, with the important difference that our hygienic efforts are confined almost solely to the matter of diet, mother's milk being almost as important as doctor's mercury.

There is this further distinction to be made in the matter of mercurial treatment that, if the mother is properly treated

with mercury during her pregnancy, the hereditary syphilis should be prevented and the child be born at term uninfected.

Fournier would have us treat these apparently uninfected children as though they had syphilis, hoping thus to forestall an unsuspected latent disease. But other authorities do not follow this rule.

The duration of routine treatment in infancy should cover the first two years, just as in adult life. Certain practitioners are in the habit of renewing treatment at the period of second dentition and again at puberty; yet it seems wiser not to employ this routine practice, but simply to warn the parents that, if the child shows any deterioration in its health or local signs of syphilis, at these times, it should be treated. Indeed, a very brief treatment may suffice to quell the disease. Thus Hutchinson advises that treatment be stopped with the cessation of symptoms; and Hochsinger, who continues treatment only a fortnight thereafter, states that eighty-five per cent of the relapses seen by him from the fourth to the sixth month were in untreated cases. But it is the part of wisdom to clinch the advantage gained by a course similar to those employed in the adult and covering the first two years. In infancy, however, two rules apply:

1. Iodid of potassium is useless in the first year.

2. Mercury may be given by mouth or by inunction. Intramuscular injections have found little favor, though the employment of both the soluble and the insoluble salts has been urged.

The usual treatment consists in the application of the officinal ung. hydrargyri, diluted with two or three parts of vaselin (reducing it to one third or one quarter strength). This is laid on the belly band and renewed with it. No special precautions need be taken to prevent irritation of the skin, and the application may be continued daily throughout the treatment with only such intermissions as may be required by the appearance of diarrhea, dermatitis, or intercurrent disease.

Infants bear mercury very kindly, and do not become salivated.

For internal administration (if inunction proves inefficient or irritating) gray powder is usually employed, mixed with two or three parts of sugar. Beginning with a quarter or half a grain t.i.d., the dose is rapidly increased to a grain t.i.d. For a more rapid effect, Holt employs calomel, gr. $\frac{1}{10}$, q.i.d., until the bowels are touched.

Injections may be employed in about one tenth the adult dose, and this may be doubled at the time of second dentition, and trebled thereafter.

Local Treatment.—The baby's skin must be kept absolutely clean and dry, diapers frequently changed, and powder freely used. Erosions and condylomata are to be treated, as in the adult, by drying and calomel powder; but it is safer to dilute the calomel with two parts of starch or talcum powder.

The nasal congestion in snuffles may be somewhat reduced by anointing the inside of the nose with ammoniated mercury ointment. Since stomatitis does not occur in infancy, no especial care of the mouth is required; but erosions and fissures upon the lips may be touched once a week with the nitrate-of-silver stick.

It is safer, as a general rule, not to incise the furunculoid lesions, inasmuch as they are discrete and are often cheesy rather than suppurative; but when at the surface or already burst, they should be cleansed daily, or twice a day, with hydrogen peroxid diluted to one half strength.

TREATMENT OF RELAPSES AFTER THE SECOND YEAR

In childhood, and especially at the time of the second dentition, the outbreak of tertiary symptoms may require mixed treatment.

The iodid is then to be administered quite as for adults.

It is well borne, and the initial dose may be from three to five grains t.i.d.

Mercury may be given in from one third to one half the adult dose, but injections should be given at about one fifth strength.

It must not be forgotten that no amount of treatment will reduce an organized deposit of bone caused by diffuse periostitis, and that parenchymatous keratitis seems to run its course quite independently of antisyphilitic treatment, though this is habitually employed.

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