CHAPTER 9

THIRD-GENERATION SYPHILIS

For many years syphilologists have discussed the question of third-generation syphilis from at least two points of view. Firstly, as to whether it occurs at all, and secondly, if it does occur, whether it does so with sufficient frequency to be of any practical importance. With regard to the first problem, the majority of observers are of the opinion that third-generation syphilis can and does occur, although by a few its occurrence has been emphatically denied. A. Fournier in 1891 wrote: "Acquired syphilis may be transmitted to the offspring 18 to 20 years after the infection, and there is no valid reason why hereditary syphilis might not be transmitted by a woman of 18 to 20 years of age." He concluded by saying that while the transmission of syphilis to the third generation is logically possible, its occurrence had not yet been indisputably demonstrated. He made the conditions of proof very stringent: evidence of syphilis in the grandparent, evidence of early inherited syphilis in the parent and child (that is, the second and third generations), and also evidence that syphilis had not been acquired by either of the parents (second generation). Subsequent French authors regarded third-generation syphilis as a not infrequent occurrence, but the evidence adduced in support of their thesis was often unconvincing, a number of congenital malformations such as cleft-palate, harelip and hernia, as well as various dystrophies, being regarded as evidence of a syphilitic nature even though the S.W.R. was negative. Finger (1900) came to the conclusion that third-generation syphilis must be regarded as being theoretically possible, but that there had been no indisputable evidence of its occurrence. The conditions which he held to be essential for its demonstration were even more stringent than those of Fournier.

While these conditions might be considered necessary to demonstrate scientifically the presence of third-generation syphilis, the majority of observers would agree that it seems unnecessary, for example, to require the symptoms of syphilis in the second generation to occur soon after birth. As Igersheimer and others have pointed out, the presence of interstitial keratitis or the presence of the well-known stigmata of congenital syphilis, such as typical Hutchinsonian teeth, are in themselves, if not absolutely diagnostic of congenital syphilis in the individual, at least

highly presumptive of it. It is a well-known fact that the fathers of congenitally-syphilitic children, who presumably have introduced the infection into the family, in many cases either give no history of infection or may have a negative W.R., so that it is difficult to satisfy the postulate that syphilis in the second generation must be absolutely excluded.

The opinion of English authorities at the turn of the century was largely influenced by that of Jonathan Hutchinson who, in the last edition of his work "Syphilis" (1909), says that he is "absolutely incredulous as to the inheritance in the third generation." It should be pointed out, however, that later on in the same chapter he retreats somewhat from this position and states that in view of the recent investigations upon the treponema and of its presence in the ovary, third-generation transmission may be a possibility. It seems, therefore, that the discovery of the treponema and of the W.R., which were made since the previous edition of his work had been published, had led him to modify his strong view and apparently he had grafted his later view upon the more rigid one he had previously held, without properly integrating them. Hutchinson's eminence in the profession was so great and his knowledge of venereal diseases so extensive, that British medicine accepted his views almost without question. George Ogilvie (1897), in a long paper upon the subject, gave a critical survey of the reported cases, drawing attention to the weak points in several of them. In his opinion it was not essential that syphilis should be demonstrated in one or other grandparent as Fournier demanded, because it is undoubtedly possible to diagnose congenital syphilis in a parent by signs or symptoms, and evidence of syphilis in the grandparent is of corroborative rather than of absolute value, as Ogilvie remarks. With regard to Fournier's and Finger's second postulate, namely that acquired syphilis in the second generation must be excluded, Ogilvie writes: "It must be granted once and for all that absolutely to exclude acquired syphilis is impossible. Common sense and a sound valuation of the probable must step in where absolute proof is impossible." It must be remembered that these words of Ogilvie were written before the discovery of the treponema and of the Wassermann reaction, and while a positive reaction in the father might be taken as evidence of acquired syphilis, a negative reaction would have little value because, as is generally recognized, the fathers of syphilitic children, even with a history of a syphilitic infection, frequently have a negative W.R. Ogilvie sums up his review in the following words: "We are told that there is not the slightest evidence of third-generation transmission and, on the other hand, that there are undoubtedly cases in which syphilis was thus transmitted. Both statements are equally unwarranted. The evidence before us furnishes us, if not with absolute proof, still with reasonable probability that syphilis may descend to the third generation." Rietschel (1927), in Jadassohn's monograph, refers to a number of positive cases, but he states that hardly any of them satisfy the

conditions set down by Fournier and Finger. If all the recorded cases are critically examined, however, one cannot doubt, he says, "that syphilis can be transmitted to the third generation, and possibly even to further generations. Its occurrence, we may believe, is not so infrequent, but it cannot always be proved in actual practice."

American authorities do not appear to regard third-generation syphilis sympathetically, holding the view that practically none of the recorded cases satisfies the Fournier and Finger postulates. Stokes writes: "It is generally conceded that a tendency to constitutional inferiority appears in the children of parents who have severe forms of hereditary syphilis. Those who have inherited syphilis in mild form may, if sufficiently treated, give birth to healthy children." The Solomons say that while there is some divergence of opinion, the general tendency is to believe that syphilis is not transmissible beyond the second generation, from which they infer that a congenitally-syphilitic individual is probably never contagious by the time the age of marriage has been reached. On the other hand, Jeans and Cooke (1930) say that: "At present we can only speculate about the possibility of congenital syphilis in the father being transmitted to the third generation. The probability of a woman with congenital syphilis transmitting the infection to her offspring depends almost entirely upon the possible duration of the disease in such a woman. We know that a woman with acquired syphilis can give birth to syphilitic children for 20 or more years, and therefore there is no reason why the treponema should not remain transmissible for so long a time in the infected offspring." They add: "The chain of circumstantial evidence accompanying many of the reputed examples is such as to provide a high degree of probability for this type of transmission."

Kemp and Poole in their article on congenital neurosyphilis were of the opinion that 2 of the 20 mothers in one of their series were themselves congenital syphilitics, and that the family almost certainly represented third-generation syphilis. Another American writer, W. M. Sams, in reporting a case, expresses his belief in the existence of the condition, and in the points connected with the diagnosis he refers to the parentage of the child in question, which should be undoubted; he himself seems to have been the first to stress this point.

In connection with third-generation syphilis a problem sometimes arises as to whether the congenital mother can be reinfected by a syphilitic husband or not. A number of authors have described cases in which a congenital patient appeared to have acquired syphilis and this would seem to be undoubtedly possible. This aspect of infection was discussed by Tarnowsky at the International Congress of Medicine in Paris in 1900. He reported cases of acquired syphilis in congenital patients and suggested the term "syphilis binaria" for this type of case. A number of American writers have concerned themselves with the question of im-

munity and reinfections or super-infections in syphilis (Chesney, Halley and Wasserman, Urbach and Beerman and others).

My own experience having been mainly with congenitally-syphilitic children and their mothers, I have not had many opportunities of attesting the possibility of super-infection in a congenitally-syphilitic father or mother. My own view is that if a congenital mother is untreated and still has a positive W.R. she would not be liable to reinfection. If, on the other hand, as a result of treatment, or possibly by efflux of time, her serological reactions had become negative, I consider it would be possible for her to acquire a second infection. Similarly a congenital father, whose serological reactions were negative, might conceivably be susceptible to an acquired infection.

Personal observations

In 1927, in a paper read before the Medical Society for the Study of Venereal Diseases in London, histories were given of 8 families with syphilis in 3 generations and of 3 others in which the sequence was probable or possible. In the first category were included cases in which grandparent, parent (usually the mother) and child all had a positive W.R.; also cases in which the parent (that is, the second-generation mother) had obvious stigmata of congenital syphilis, such as typical Hutchinsonian teeth, rhagades or interstitial keratitis, from which one concluded that one or both grandparents were syphilitic. On further inquiry in most of these cases I was able to elicit the fact that the grandfather had died in a mental home of paralysis, which was subsequently confirmed to have been general paralysis.

I regard it of extreme importance when a history is taken of a patient, and particularly of a child, that the mother should be herself examined, and inquiry into her family history—parents, brothers and sisters—should be made.

In the second category, of "probable" syphilis in three generations, I included cases in which the mother was obviously a congenital syphilitic and the child might have had the disease in infancy, but owing to an imperfect history and absence of blood tests at the time, and a negative W.R. when the child was seen by me, I was unable to be certain of the diagnosis. If the child's early symptoms suggested that it might have suffered from the disease the case was called *probable*, whereas if the early symptoms were more vague it would be classified as *possible*. A similar grading would apply to stillborn children.

In the 5 years following 1927 my list of families increased from 8 to 16 with syphilis in three generations, from 1 to 6 in which the disease was *probable* in the third generation and from 2 to 7 in which it was *possibly* present; and in addition I had come across 2 families in which both parents were congenital syphilitics. This larger group of cases formed

the subject of a presidential address I delivered before the same society in 1933, and the considerable additions which were made during the 5 years I had been on the look-out for such cases would suggest that the condition is not so rare as is commonly thought. If such be the case, it may legitimately be asked why other authorities have not recorded similar experiences.

There are probably several answers to this question. First and foremost, I think, is the fact that life is so strenuous to-day that we do not allow ourselves time to do things thoroughly. For example, unless a mother were almost blind from corneal nebulae or had a saddle-shaped nose or rhagades which could be detected from the other end of the consulting-room, how many paediatric physicians or surgeons would take time to investigate such a mother and possibly inquire into her family history, when it was her child about whom she had come to seek advice? I have on many occasions seen mothers with obvious stigmata of congenital syphilis which had been missed, doubtless because the physician or surgeon had a definite and usually too large a number of patients to examine. Secondly, I had learned to detect finer diagnostic features, such as minor degrees of malformation in the incisors and 6-year-old molars, broadening of the forehead, with some widening of the bridge of the nose in the parent, which has prompted further inquiry into the personal and family history. In this way I was able to elicit information about earlier eye trouble in the mother herself or in one or more of her brothers and sisters, about her mother's obstetric history, or possibly her father's death from general paralysis, tabes, aneurysm or heart disease. Thirdly, there is the lack of appreciation by clinicians that one negative blood test on mother or child does not necessarily preclude syphilis. Fourthly, practically the whole of my experience was gained from patients of the hospital class amongst whom acquired syphilis in the father formerly received rather inadequate treatment, so that the chances of congenital children being born were great. Practitioners who treat syphilis in private doubtless get their patients to attend more regularly and oftener, so that such patients are less likely to transmit the disease to their offspring.

The conclusions which I drew from my observations recorded in 1933—though lacking in absolute proof—were that a congenitally-syphilitic mother, who had received no, or perhaps only very little, antisyphilitic treatment, might transmit a severe or florid congenital syphilis to her child, even though her husband were healthy. The explanation may be that she is a carrier of the treponema in her ovaries or other pelvic organs. On the other hand, if she has received some treatment, or even if she improves spontaneously, she may transmit only a mild form of the disease to her offspring, or the child may be only undersized or possibly the effect on the child may be the presence of petit mal, mental retardation or some other form of so-called "para-syphilis" or "occult" syphilis. This is a

possibility which, in my opinion, should be borne in mind. It must also be remembered that a congenitally-syphilitic mother, equally as well as a mother who has acquired the disease in the usual manner, may bear healthy children alternately with infected children. I made two further observations in connection with the children of congenitally-syphilitic mothers: (1) that in several of the families it was the second child who was apparently most markedly affected; and (2) that in several of the families the affected child showed involvement of the central nervous system. This might take the form of a typical neurosyphilis (clinical or latent) with a positive spinal fluid, or of an atypical case such as (i) D.C. (p. 291), who had a progressive hydrocephalus with a normal C.S.F.; (ii) L.W. (p. 289), who had a paretic fluid with granularity of the ventricular ependyma, yet a negative blood W.R.; (iii) K.T. and (iv) S.S., both of whom had relapsing cerebrospinal fluids. The former had slightly increased protein and cells with negative W.R., Kahn, Lange and globulin tests at $3^{5}/_{12}$ years, yet a strongly-positive W.R. with slightly increased protein, cells, Lange and globulin at $10^{10}/_{12}$ years, and a normal C.S.F. 2 years later; whereas the latter is referred to in Table 16, case 14, and Table 32, case 32 (pp. 276, 402), where it is seen that the C.S.F. relapsed twice before becoming negative eventually at the age of 5 years. Further observations upon these two points are needed before we can conclude that they are usual, and not accidental, occurrences. A congenital father appears to be much less likely than is a congenital mother to transmit the disease to his offspring, and it is noteworthy, as was pointed out in the chapter on neurosyphilis, that mothers with neurosyphilis are much more likely to transmit neurosyphilis to their offspring than are neurosyphilitic fathers. The interested reader is referred to the author's original paper for details of the families referred to, but a few of the more interesting families are recorded below:

Family 363. The grandmother had 15 pregnancies, including 5 miscarriages. Of 10 living children only 2 are now surviving, of whom the patient's mother is one. The grandmother's W.R., at the age of 86, was very strongly positive, but she had no symptoms or signs of the disease. Five years later, at 91, she was still hale and hearty and the W.R. was practically negative, but the Kahn was positive. The second-generation mother had I.K. from 8 to 17 years of age. She attended Moorfields Eye Hospital and was treated with pills. In 1911 her W.R. was nearly negative and in 1917 quite negative. She had typical Hutchinsonian teeth and corneal opacities, yet when she attended the Children's Hospital in 1909 and again in 1911 with her daughter (third-generation patient) her congenital syphilis passed unnoticed by the surgeon. The husband's father (second generation) is said to have had syphilis and to have been treated, but was not seen by me. Of 16 pregnancies only 2 children survived. The elder one, born 1909, had a rash at $3\frac{1}{2}$ months and was treated with mercury for 4 years, but at 8 years of age the W.R. was still positive and in spite of vigorous treatment by injections it did not become negative until the age of 22. Her sister, who was born 8 years later, had no symptoms and a negative W.R. until the age of 15.

This family shows syphilis in three generations, though it is impossible to say whether the mother's congenital syphilis was transmitted to her daughter or whether it was an example of super-infection, the second-generation mother having been infected by her husband. The family also illustrates the interesting point that an individual may have a strongly-positive W.R. at the age of 86 and appear to be perfectly well with no obvious signs or symptoms of syphilis, and that without treatment the W.R. could become negative 5 years later, even at that advanced age.

Family 376. The maternal grandmother (first generation) died of locomotor ataxy. The mother has typical facies, corneal opacities and positive W.R. (second generation). Father, no history of syphilis and W.R. negative. Married in 1910. Children:

1. 1911. Girl. No signs or symptoms of syphilis up to 12 years of age, but W.R. strongly positive at 11 and 12 years of age.
2. 1918. Male. Mentally defective and unable to sit up. W.R. strongly positive. Became negative in 1923 but was still very mental when last seen in that year. C.S.F. was not examined.
3. 1919. Abortion at 1½ months.
4. 1920. Female. No symptoms in infancy or during the first year. W.R. negative.
5. 1922. Female. Not seen or tested.

This family shows syphilis in three generations, the second-generation mother having very typical syphilitic facies and old interstitial keratitis (see Fig. 29). The first child had no signs or symptoms of the disease, but had a positive W.R., whereas the second child, born after a long interval, was very severely infected and mentally defective.

Family 109. The maternal grandmother (first generation) had no symptoms, but was found to have a strongly positive W.R. Mother (second generation) had no symptoms, but had a history of slight snuffles. The W.R. was very strongly positive during 3 years in spite of much treatment and was persistently positive after 3 more years, making 6 years in all. It was this fact which led me to inquire into the possibility of her having congenital syphilis. The father (second generation) admits no possibility of infection. His W.R. and Kahn were negative. Married 1920. Children:

1. 1922. Stillbirth at full term.
2. 1925. Male. Florid congenital syphilis at one month. W.R. strongly positive, but rapidly became negative.
3. 1927. Male. Born after mother's treatment. No signs or symptoms of the disease during the 3 years he was under observation.
4. 1931. Died in 10 days, ? cause. Patient was not seen by me.

Family 648. Maternal grandfather (first generation) died of G.P.I. (verified). He and his wife had 12 or 13 children and only 4 survived:

1. 1894. Male. Said to be well.
2. 1896. Male. Said to be well. Had I.K. aged 36. Married and has 2 healthy children.

3. 1897. Female (second-generation mother). No early history available. Has broad, bossed forehead and the right lower canine notched. Nearly all the other teeth are artificial. Married 1920 and when seen in September 1922 her blood was very strongly positive. Twelve years later she developed G.P.I. at the age of 36. Her husband could not be induced to come up for examination. Children:

(1) 1921. Male. Small baby—birth weight only 4 lb. No rash or snuffles. Had a double hernia and was under supervision for 6 or 9 months. Seen by me in 1932. Is small, but has no signs of congenital syphilis. W.R. and Kahn negative.

(2) 1927. Male. Healthy baby, but had a big head. At 3 years had slight jaundice and history of dark urine. Blood count, fragility tests, liver function tests, Van den Berghs reaction and C.S.F. were all examined at another hospital and said to be negative or normal. When seen at Great Ormond Street his blood gave a strongly positive W.R.

4. 1903. Female. Married and has 3 children, all said to be healthy.

This case is of interest because it brings out rather clearly the point I have already stressed, that by paying more attention to the parents of our young patients we can often obtain considerable help in diagnosis. This boy had attended hospital on many occasions for treatment, but the suggestive appearance of the mother had apparently not been noted. Owing to her brother recently having developed interstitial keratitis, which, be it noted, occurred at the age of 36 years, her family history had been gone into and the mother herself had already been acquainted with the result, but she had not imparted the information to the hospital physician under whose care her little boy had been. One may note further that it is again the second child who shows signs of congenital syphilis, though it is just possible that the older boy, who weighed only 4 lb. (1·8 kg.) at birth, might then have had mild symptoms of congenital syphilis although his W.R. and Kahn were negative by the time he came under our observation. (See also under "Congenital General Paralysis," p. 306.)

Family 422. This family is of interest because it is the only one in which a congenitally-syphilitic father appeared to convey an infection to one or two of his children without the wife ever showing any sign or symptom of syphilis. The paternal grandmother (first generation) had a strongly positive W.R. when seen by us at about 60 years of age. She had been married 8 years before the birth of her first child, who became the second-generation father. He is said to have been a healthy baby, but had chest trouble. Fourteen years later a girl was born who died of diphtheria at 2½ years of age. The paternal grandfather is said to have been healthy. He was a sailor in the mercantile marine, a calling which at that time was a fertile source of syphilis. He is said to have died at the age of 60 from pneumonia. The second-generation father had a big broad head rather suggestive of congenital syphilis. The teeth were bad but not Hutchinsonian. He gave no history of syphilis or of the possibility of having acquired it before marriage. His W.R. was strongly positive in 1921 and also on three occasions in 1922. Between these dates he was given 20 injections of sulfarsphenamine and mercury iodide pills. One year later the C.S.F. was negative

in all respects. In 1927 the S.W.R. was almost negative and in 1938 the W.R. and Kahn were quite negative. The mother has never shown any signs or symptoms of the disease, and her blood W.R. was negative on many occasions between 1921 and 1938.

This family has already been referred to in the chapter on "Transmission" (p. 51).

In the next two families both parents were congenitally syphilitic:

Family 542. The father was born in 1899. There is no available history of infancy and childhood, but at the age of 13 he was treated for his eyes, and apparently his sight had never been normal, though he was able to work until he was 21 years of age. At the age of 30 he was seen by Dr. (now Sir Francis) Walshe, of University College Hospital, who reported that "his right pupil was dilated and inactive to light; choroido-retinitis and patches of exudate in the lens were present. Knee and ankle jerks were not obtained; others were present." Dr. Archibald Gray noticed at the same time that the bridge of the nose was depressed and the central incisors typically Hutchinsonian. His W.R. was positive and he was given three injections of N.A.B. I examined him a few weeks later and found him to be obviously a congenital syphilitic and his W.R. was strongly positive. His father had died of pneumonia at 34 years of age and no history could be obtained about his mother except that she had remarried. The second-generation mother was born in 1900, snuffled in infancy and was said to have been almost blind from birth. She attended Moorfields Eye Hospital every week. In September 1928 at the University College Obstetric Hospital her W.R. was found to be negative. She was seen by Dr. Gray for the first time two months later when she was 8 months pregnant. He found opacities of both corneae, irregularity of the left pupil, a depressed nasal bridge and Hutchinsonian central incisors. Clinically the case was obviously one of congenital syphilis, but the W.R. taken at that time was negative, as it was also a week later after a provocative injection of N.A.B. I saw the patient 4 months later with her child. She was obviously a congenital syphilitic, although her W.R. was again negative. The child, born in December 1928, is said to have snuffled at birth, but had no rash. The cord blood was negative. When seen by me at $3\frac{1}{2}$ months, there were no symptoms and the W.R. and Kahn were negative. Eight months later, when he was about a year old, he was still free from symptoms of congenital syphilis. The mother was at that time again 4 months pregnant, but the subsequent history could not be ascertained because the family moved into the provinces.

This case is of interest inasmuch as both parents were undoubtedly congenital syphilitics and presumably both had been treated on account of eye trouble. This doubtless explains why the first child that was born to them gave a negative serological reaction. If it was infected at all (which is just possible, as it is said to have snuffled at birth), the infection must have been a very mild one.

Family 71. Both parents were congenital syphilitics, the father having been treated by me when a child; the mother was untreated. The paternal grandfather, born 1880, was a steward in the Mercantile Marine and he may have contracted syphilis on one of his voyages. In 1932 he was said to have been well.

The paternal grandmother was born in 1881 and married in 1903. Her W.R. in May 1915 was strongly positive. She had no symptoms between 1915 and 1932. Their family was as follows:

1. and 2. Stillbirths.

3. 1906. Male, "second-generation father." He snuffled in infancy and had periostitis of the tibia at 9 years for which he attended Great Ormond Street. His W.R. was then very strongly positive. It became negative as a result of treatment in 1918–19 and was still negative in 1927. In 1932 he appeared to be quite well.

4. 1909. Female. No symptoms in infancy or until puberty. In 1929 she is said to have had a "rheumatic heart." Her W.R. and Kahn were "doubtful."

5. 1911. Male. No infantile symptoms. 1915 W.R. weak positive. 1929 not strong, ? rheumatic.

6. 1913. Stillbirth.

7. 1915. Male. No infantile symptoms. Developed rheumatism at 4 years and died of valvular disease of the heart.

8. 1919. Male. No infantile symptoms. 1929 ? rheumatic. Enlarged glands in the groins. W.R. and Kahn negative 1929.

Second-generation mother born 1907. Her father died at 39 of "consumption" and her mother at 41 of ? cancer. At the age of 21 she was in a general hospital in London for tuberculous kidney, which was removed. It was then recognized that she had typical Hutchinsonian teeth and her W.R. was found to be positive. No antisyphilitic treatment was given or even suggested. In June 1930 she married patient No. 3 referred to above, who had been treated for, and was presumably cured of, his congenital syphilis. A baby was born in December 1931 who appeared healthy except for a left inguinal hernia. For this he was brought to Great Ormond Street Hospital, where the almoner, recognizing the name, upon inquiry found that the child's father had been originally my patient in 1915. Knowing that I should be interested to see this child she brought him to me. He was then 2 months old and showed no symptoms or signs of congenital syphilis. His mother's blood was examined and found to be strongly positive, but the child's blood was not examined until he was 6 months old. It was then strongly positive, so it was obvious the child was suffering from latent congenital syphilis as there were no symptoms of the disease present. X-rays of the long bones showed evidence of old periostitis, especially of the tibiae. In October of that year, at the age of 10 months, the W.R. was still strongly positive and the C.S.F. weakly positive.

This case is perhaps one of the most, if not the most, interesting in the series, because the father had been treated for congenital syphilis and he was apparently quite well at the time he married. The wife had never had any treatment and it would appear that her untreated congenital syphilis was transmitted to the child. The case also raises the important question whether, if a woman is accidentally discovered to have congenital syphilis, she should be advised that treatment was necessary for her, even though she may have come under observation for a totally unassociated condition, as this patient did.

In my opinion the patient's doctor should always be informed if congenital syphilis has been discovered in his or her patient, or if she has no

doctor then I think the patient herself, if of age, should be advised to have treatment. If under age, then the parents should be informed and recommended to have treatment given to their child.

Between 1933 and 1939 I came across 12 more similar families where there was syphilis in three generations or where the children of the third generation had probable or possible ("B" or "C") syphilitic manifestations. Two of these congenital mothers had children who were probably not syphilitic, but as they had had only one and two children respectively in 1939 further information about them, which unfortunately owing to the war was unobtainable, might have led to the detection of subsequent syphilitic children. Two other mothers, whose own mother suffered from tabes, had two children and one child respectively. The elder daughter had two children, the first being well and the second being possibly syphilitic (a "C" case). He had no infantile symptoms, but suffered with fits since a fall at the age of 9 months. His blood and spinal fluid both gave negative tests at the age of 5 years. The younger daughter had one child, who was brought to Great Ormond Street and seen by Dr. Reginald Lightwood, who diagnosed amyotonia congenita at the age of 8 months. The child's W.R. was then negative, but as the mother's S.W.R. was weakly positive, the child, in view of his clinical condition, might be regarded as a possible congenital syphilitic (a "C" case).

Among the families with syphilis in three generations encountered the signs and symptoms exhibited by the mother which prompted inquiry into the possible presence of congenital syphilis in her may be summarized as follows:

Typical or suggestive Hutchinsonian teeth, 15 cases.
Facies, including rhagades, squint and unequal pupils and pharyngeal scarring, 10 cases.
Persistently positive W.R., 6 cases.
Signs of old keratitis, 3 cases.
Suggestive history of the mother's mother or sibs, 6 cases.
Mother's own history of Raynaud's syndrome and of interstitial keratitis, 1 case, and
One died of congenital G.P.I.

It should be mentioned, too, that once suspicion had been aroused, more cases of interstitial keratitis were disclosed, but these had apparently almost entirely cleared up.

This summary illustrates the importance of recognizing the minor degrees of Hutchinsonian characteristics of the teeth and of bearing in mind the other stigmata and possible indications of congenital syphilis in mothers, which may thus give a possible clue to the nature of physical and mental defects, malformations and obscure conditions in their children.

TABLE 31

Details of 42 Families with probable Syphilis in Three Generations

Total number of families showing "probable" or "possible" syphilis in three generations	= 42	
Total pregnancies	= 157, including 3 twins	
Average per family	= 3·74	
Survivors	= 92 (? 94)	
Average per family	= 2·2	
Children:		
Well	= 45 (12 born after mother's treatment)	
Result unknown	= 9	
Congenitally syphilitic	= 19	
Congenitally neurosyphilitic	= 16 + 3 possibly neurosyphilitic	38 congenital syphilitics
"B" or "C" cases living	= 9[1]	
"B" or "C" cases died	= 21	
Died (unknown cause)	= 3	
Premature births or miscarriages	= 35 (most of these were probably syphilitic)	

[1] These include such lesions as epilepsy, mitral valve lesions, a bleeder and a case with amyotonia congenita.

The deaths may be summarized as follows:

Premature births or miscarriages (mostly syphilitic)	= 35
"B" and "C" cases, probably from syphilis	= 21
? cause	= 3
Arsenical dermatitis at 4 years	= 1
Empyema and syphilitic lung at 8½ years	= 1
Total	61

A few others died of conditions unassociated with syphilis.

Summary

My own view on third-generation syphilis is that during the 30 years that I was particularly interested in this disease, congenital syphilis in the mother played a not inconsiderable part. I think it is certainly possible for a woman with untreated congenital syphilis to have congenitally-syphilitic children by a healthy husband, and furthermore that if the children are not obviously infected by the treponema, their developing tissues and organs may be adversely affected by the maternal syphilis. In the last event the child's W.R. would be found negative and antisyphilitic treatment carried out after birth would be of no benefit to the patient. To this condition the name "para-syphilis" or "occult syphilis" might be given.

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The above list of references makes no claim to being exhaustive, but gives both sides of the picture.