HEREDITARY SYPHILIS

IN CONNECTION WITH CLINICAL PSYCHOLOGY AND PSYCHOPATHOLOGY

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Fit morbus hereditarius et transit a patre ad filium
Paracelsus, 1529.

In relation to clinical psychology and its proximate
interests, psychopathology, juvenile delinquency and
even pedagogics (Heilpädagogik), the problem of con-
genital syphilis looms up with increasing importance as
the results of recent studies are examined and older
data pondered anew; for we find not only that syphilis
in the ascendants may account for nervous and mental
abnormalities covering the entire gamut of neuropsy-
chic disturbance—from mere nervousness to complete
idiocy—but also that timely and active treatment may
improve or even cure such neuropathy or mental
enfeeblement, or arrest the progress of deterioration.
The subject, besides, is rather singular insasmuch as
the diagnosis is not easily inferred, the facts being
purposely or not purposely withheld, or come upon
by means of barest clews. With a little more knowl-
edge of the effects of inherited lues, many a nervous
symptom, neurosis and aberrant characteristic enig-
matic before will have been explained.

Acquired syphilis in childhood is not common (occa-
sionally contracted from a wetnurse), nor does its
recognition usually present any difficulties. With con-
genital or hereditary lues, however, lies the rub. In
the majority of cases the anamnesis leaves us in the

* Lecture in the course on clinical psychology at the College of
Physicians and Surgeons, Columbia University.
1. Oppenheim, H.: Lehrbuch, Ed. 6, ii, 1281. Ziehen also states
that he saw the intelligence defect of a 13-year old feeble-minded
girl entirely clear up on energetic antiluetic treatment. See also L.
Findlay, The Use of Neosalvarsan in Mental Deficiency, Glasgow Med.
dark, the mother having no knowledge of an infection or denying it or, again, remembering absolutely no occurrence of "secondary" luetic manifestations in the child.

To ferret out the facts one must question both parents, not only as to their knowledge of primary infection, but also as to occurring secondary symptoms, in which case one may sometimes elicit enough for a tentative diagnosis. Not infrequently, however, secondaries may be so mild as not to be noticed by the individual (Nonne\(^2\)). A superficial (that is, rapidly carried out) examination of the parents may show some sign or stigma (of lues, or degeneracy) which may aid in our summing up, or even some stigma of congenital lues; that is, the mother of the child in question may herself have been congenitally luetic and have passed the infection to her offspring—congenital syphilis in the third generation. And if she shows no stigmata (she will in all probability have no knowledge of her condition), the problem of detection becomes even more difficult. But, one will ask, did not the child itself show signs of secondaries at birth or in very early infancy? These may also have been so mild as to be overlooked—or possibly there were none (the still undecided problem of "late congenital lues").

In approaching our subject, let us first examine the factor of heredity itself in this connection: How does syphilis in one or both parents\(^3\) affect their offspring?

The organism of the disease, *Treponema pallidum*, may be transmitted to the embryo, which is then also syphilitic—the organism circulating in its blood. The morbid products or toxins of this organism have an inhibiting potency, and the evolution of the embryo or fetus, if the germ plasma itself continues to unfold, may come to various incomplete issues, that is, (a) it may cease developing and be extruded (abortion); or (b) the fruit grows, but is

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3. There is still an unsettled controversy as to whether there can be a paternal hereditary syphilis, that is, whether the spermatozoon can pass the syphilitic organism to the embryo, the mother remaining free (from disease, or getting it from the paternally infected embryo) (or from disease, or getting it from the paternally infected embryo) (or from disease, or getting it from the paternally infected embryo) (or from disease, or getting it from the paternally infected embryo). From disease, or getting it from the paternally infected embryo); or whether the husband must first infect the wife, who then infects the child through the placenta, passes it on to the embryo. The latter is probably the case, and the former is more widely believed than the latter. For our own problem this is of little moment. See Deutsche medizinische Wochenschrift, 1914, Nos. 15, 24 and 25.

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born before the normal expiration of intra-uterine life (premature birth), in which case it is immature, frail, below weight, possibly marasmic—and hence more vulnerable to disease. This premature child may die directly after birth, or soon thereafter; (c) the fruit may develop for a time, or even to term, and be born dead (stillbirth). Hence the frequent history from luetic mothers as to sterility, abortions, stillbirths, premature children, children dying soon after birth; and hence the importance of inquiring if this was the case, in taking an anamnesis. On the other hand, such a mother may bear several children without having had abortions, etc., who remain alive, and with but few or mild symptoms. In a case noted by Milian, such a patient had six living children, though she was luetic.

A study of Nonne's material,\(^4\) for instance, showed the following: Ninety syphilitic families were examined; eight remained sterile. In the remaining 82 families, 350 pregnancies occurred. Ninety-one, or 26 per cent, ended in abortions, 10, or 2.9 per cent., in stillbirths, 66, or 18.8 per cent., died small, 183, or 52.3 per cent., remained alive. Of the latter, only 119 were examined. Thirty-six cases were found normal, 83 pathologic. Expressed in other words, 47.7 per cent. of the fruits of the syphilitic parents died or were extruded before term. Only 52.3 per cent. remained alive, and one could reckon that of these, 35.8 per cent. were pathologic. If but one parent was diseased, there was a child mortality of only 37 per cent.; if both were diseased or gave a positive Wassermann reaction, between 47 and 53 per cent.

In eighteen families, A. Fournier counted 161 pregnancies of which 137, or 85 per cent., were stillbirths. J. N. Hyde\(^5\) found that 116 out of 121 syphilitic infants died in the first year, etc. Hochsinger himself speaks of 67 families in which there were 266 pregnancies, of which only 142 came to term, but 76 infants died within the first days; 124 were premature and dead. Lesser\(^6\) examined 89 children (of 35 families), of whom 32, or 33 per cent., were well, 57, or 66 per cent., were syphilitic. Of these, 29 had signs of congenital lues. (Findlay and Robertson, *Glasgow Med. J.*, vi, December, 1914, p. 413, found in ninety-seven syphilitic families, 19 per cent. of twenty-seven pregnancies ended in abortions or stillbirths; but in nineteen non-syphilitic families, 19 per cent. of 123 pregnancies also ended in a similar fashion.)

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Again, (d) the child may be born at term and show signs of syphilis and nevertheless live on. These signs may be few and not very distinct, may be transient, or may flourish. They are the so-called "secondaries." Or (e) there are no signs whatsoever at birth, in spite of obtaining syphilis, the "secondaries" breaking out a few weeks later. Or (f) there is nothing present for weeks, months or years, the disease being latent, and becoming manifest only after from eight to twenty-eight years in the form of "tertiary" lues (so-called "syphilis hereditaria tarda," or late hereditary syphilis, or terciarisme d'emblée).

That tertiary symptoms occur similar to such in the adult without the earlier appearance of "secondaries" still remains a much mooted question. So recently Finkelstein again challenged the theory and said he believed that the "secondaries" in such a case either had occurred in utero, or appeared after birth but were of so slight a nature as to be overlooked (a few spots on the soles and nothing more, or a slight induration of the soles, or a fleeting nature, or a light anemic yellow discoloration of the skin, or near examination a very slightly enlarged spleen, or an enlarged and sensitive cubital gland. These may even first be seen in the fifth or sixth month, etc.) This has no very great importance for us, however, who are to remember that whether there is a real syphilis hereditaria tarda or whether this is only an "overlooked" syphilis (syphilis occulta), the abnormality of the patient before us may be due to lues even if there be no history of "secondaries."

B. There is a second way in which syphilis of the parents affects their offspring. Such parents are themselves constitutionally affected by the disease, and hence the germ cells uniting to form the embryo may also, one or both, be affected, depleted or inactivated, just how cannot be said; possibly it is a molecular disturbance, possibly chemical through the poisonous metabolism of the treponeme itself; possibly it is but a condition of very low vitality, a marasmoid state.

Howsoever this may be, the fact remains that this embryo, without containing the infective organism itself, enters from the start on a darkly checkered career whose fate may be almost as sinister as that of the really infected embryo. Here the development may also be inhibited or retarded; or the fetus may not develop to term; or be born prematurely, or with deformities, dystrophies, aplasias, etc.; or the child at term is marasmic, puny, weak and miserable. For us it is especially to be borne in mind that in the general invalidation of such an individual, the nervous system bears its share, at times the very brunt of the defect. Besides, its lack of resistance makes it prone to further injury. This, then, is the effect of germ damage, not germ infection. Such children will give no Wassermann reaction. They are not syphilitic but neuropathically or psychopathically constituted or marred. No doubt both facts A and B may at times be combined. Strictly speaking one might call the cases of germ damage as the really hereditary, and of germ infection as the congenital. In the former, nothing can be accomplished by specific treatment. What a problem this for the sociologist and the eugenist enthusiast!

But a third possibility must be borne in mind, namely, C, that such parents may have an entirely healthy child and one that remains so. Such parents may have several children, some of whom are affected, some remaining normal. A normal child may follow a diseased one, and such may again be followed by one with syphilis. No rule whatever is observed here.

Not irrelevant in this connection are some recent observations by Nonne and his assistants (see articles of Hauptmann and Raven). It would appear (though

7. Oppenheim (Lehrbuch, Ed. 6) states that such may break out even in the third or fourth decennium. Fournier gives the possible limits as from 3 to 28, but 12 as the maximum. Rale found the first symptoms to appear between 17 and 18. The greater number of cases seem to occur between the twelfth and fourteenth years.
9. See Case 1 at end.
it has not yet been definitely established), that the disease condition in the children surviving in syphilitic families is the less severe the more intense the disease was in the parents, as if the virulence of the disease becomes more "attenuated" in the offspring, the more severe or outspread was its passage through the parent. This study leads one also to believe that in families in which one parent remained free (chiefly the wife in Nonne's series), the greatest percentage of affected children occurred, in which case the prognosis was better for the offspring, should it survive, if both, rather than one parent, were affected. This, too, however, Raven points out must still be substantiated in the material of others, as it is not in accord with former findings. More conclusive is the observation that the augury is much graver for the offspring when not the father but the mother is diseased. Fifty-two syphilitic men occasioned 154 pregnancies, of which only one-half matured or remained alive. In twenty cases of maternal syphilis there were seventy-four pregnancies, of which only a fifth came to term and remained alive. A. Fournier also found that in purely maternal syphilis, 84 per cent. of the offspring were affected; in paternal syphilis, 37 per cent.

We keep in mind, then, the possibility of children who are actually syphilitic, such who were germinally affected but not infected, and who now present abnormalities, that is, psychopathic divergencies (but no syphilis), and finally such as may be entirely normal, or again only apparently normal, the abnormality to break out later. These facts necessitate our remembering, in taking the anamnesis of any child, that we must inquire carefully about the condition of other children in the family, for another child may give the clue of a syphilis that otherwise would never be dreamed of.

For instance, a little patient is brought because of headache and fatigue; the mother knows nothing of syphilis—does not know what it is. The child shows no signs of any kind. Syphilis would scarcely occur to one's mind. A brother has epilepsy and enuresis, and is slightly debile. Even that scarcely points to a specific disease, though it brings up the possibility. But a third child has an interstitial keratitis. That makes one take a Wassermann reaction.

Let us now see what the condition may be both in the patient and in the parents of the patient, and in what way the interrogation and examination of the latter may put us on the scent of lues in the child, or help in clearing up the doubt when we suspect it but find no signs: We may get a history, in the parents, of a primary infection. But the father may deny it and the mother in a large percentage of cases is entirely ignorant of it. One or other may give a history of "secondaries," or of "tertiary" symptoms, or evidence signs of tertiary syphilis, or of tabes or paresis. And finally, one may happen on signs of congenital syphilis in the parents—the patient having a condition of congenital syphilis in the third generation. Wherever, then there is a positive history, or luetic signs are present in the parents, we must reckon with a possibility (even a rather strong possibility if the patient is one of the first three children) of congenital lues in the child, in which case, signs or no signs, etc., a Wassermann test of the child is imperative. Nonne even remarks that not only on the children of pacitics, should one make a Wassermann reaction, but on the children of the children of the same. Of course we shall make a Wassermann reaction on the mother always, even when the mother's history and examination are negative, but a symptom or sign in the child suggests lues; and sometimes in coming out positive it will lead us to a definite diagnosis. But just here we are more often thwarted by the curious fact that the mothers of luetic children may give a negative Wassermann reaction. How explain it? I have already referred to this. It has been thought that the mother in carrying a syphilitic fruit may attain an

15. Cassel reported that of 41 mothers of congenitally luetic infants, 34 denied any knowledge of infection. Yet 19 of these, or 56 per cent., gave a positive Wassermann reaction. Lesser found the same in 19 out of 27 cases, Boas in 13 out of 33, and Knöpfelmach in 19 per cent. of his cases. In Cassel's cases 7 showed no signs whatsoever; 11 had a negative Wassermann reaction, 3 showed symptoms, 6 had neither signs nor Wassermann reaction. Again Riethe, testing the wetnurses at the Dresfeter Stäublingheim, found this 10 per cent. gave a plus Wassermann reaction, though they had shown neither signs nor symptoms of the disease. These facts incidentally show that there is no absolute way of telling that a wetnurse is non-syphilitic, though this is probable if besides the absence of signs and symptoms, both mother and children of mother show a negative Wassermann reaction.


18. According to R. Miller (Note 50) only about 15 to 20 per cent. of untreated mothers who bore luetic children some considerable time ago give a Wassermann reaction.
immunity without contracting the disease (Colles' mothers), such a mother being then syphils-immune but not syphilitic. Lessera however, asserted that when such mothers were tested-directly after the birth of their syphilitic child, nearly 100 per cent. of them gave a positive Wassermann reaction. He therefore believes that when a negative reaction occurs in such mothers there has been a spontaneous cure, but that such mother has been syphilitic. A negative Wassermann reaction in the mother, then, does not negate the possibility of syphilis in the parents having caused the obtaining abnormalities in the child before birth.

Now what may be the various conditions obtaining in the children (or grandchildren) of syphilitic parents? I shall note only what is of importance to us in clinical psychology and as neuropsychopathologists. The severe cortical involvements we need scarcely dwell on, for it is well known and taken for granted that idiocy and imbecility are, in good proportion, due to congenital lues, and a careful clinician will in all such cases make a Wassermann test and look over the individual for stigmata, history or no history. Of far greater importance, and offering decidedly more danger and possibility of oversight, are the milder manifestations; and so we had better start from the least enwighted end of the scale.

We have seen that an entirely healthy child may be born to leucitic parents; and again a child may be born who is only apparently healthy, but who has a latent syphilis which may become active later (and which child might give a positive Wassermann reaction, if tested). Such cases are not exactly rare in the literature, and would more often be noted if constantly thought of and properly diagnosed. Suddenly out of a clear sky, symptoms may appear; or the child advances normally up to its tenth or eleventh or twelfth year, and then without apparent or ascertainable cause ceases in its mental development or begins to deteriorate. The first sign may indeed be mere mental defatigation (as in a case of Nome's), which then soon shifts into the sorrowful state of early paresis. Or, again, epileptic seizures may first occur and initiate the mental change and final cessation; or suddenly an amaurosis may be contracted.

Whereas the various visceral bone and skin manifestations are common in the infancy and early periods, the nervous system, when affected, shows severe changes usually about puberty. But even before this time there may be enough evidence if the possibility of such is remembered. So Hochsinger pointed out that, in his very extensive material, such children showed an "irritable weakness" or excitability, and especially a fatigability, of the general nervous system, and nothing more. This, in fact, was his most frequent observation. But there are still other manifestations to bear in mind. Such a child may be just "headachy," particularly at night, or it may have a tendency to migraine, chronic vomiting, dizzy spells or epilepsy. A choreic condition may exist; that is, a persistent chorea may be leucitic and not rheumatic.

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20. In a similar way dementia praecox may first evidence itself. In this connection it may be interesting to point out that dementia praecox has been thought of as standing in some direct or indirect relationship to congenital syphilis. See, for instance, Meggendorfer: "Über Syphilis in der Ascendens von Dementia Praecox-Kranken, Serodruck aus Deutsch. Ztschr. f. Nervenheilk., 1914, II.


22. In one of my cases, however, the headaches occurred especially and regularly in the mornings on getting up.

23. See Cases 1 and 2 at the end. Milian reported a case (Society Report, Deutsch. med. Wochenschr., 1914) in which a child had chorea. The mother and father gave a negative Wassermann reaction. Later the chorea was cured, but the child remained decidedly clumsy. Both in the child and in the mother the Wassermann reaction later became positive. This child showed the following abnormalities: a somewhat protruding or vaulted forehead, hair growing into the forehead, moderate stump nose and slit-eyes, iris of two colors, linear transverse nail leukoza, irregular position of teeth, abnormalities of outer ear, etc. (These abnormalities are stigmata, partly indicative of lues and partly of degeneracy.) Elsewhere (Soc. med. f. h., Nov. 29, 1914) Milian stated that congenital lues is found in a very large proportion of chorea cases, and that chorea thought or not a manifestation of hereditary syphilis. In this paper was thoroughly investigated a case of congenital syphilis in which chorea runs over three or four months (the usual type seen in association with or after rheumatism), and when we get no history of previous primary syphilis, the case may be kept in mind. A cardiac murmur will not aid us in the diagnosis, for in the experience of the authors, both congenital and acquired, it is one of the most frequent factors in causing disease (see Warthin, Am. Jour. Med. Sc., May, 1914; also Findlay and Robertson, Congenital Syphilis, Glasgow Med. Jour., xxxiv, No. 6); hence in both rheumatic and
A neurasthenia,^{24} hysteria or hysteroneurasthenic condition may be the clinical expression of a congenital syphilis. It is interesting to remember that Freud^{25} pointed out that in more than half of the severe hysterias and obsessions, etc., treated by him, the father of the patient was syphilitic. These patients showed no bodily symptoms of lues, and their psychopathic constitution was looked on by him as a "leptic inheritance."

In some cases the child, mentally normal or even unusually advanced, may show a mental unrest, lability of mood, and especially, as Nonne^{26} has pointed out, disturbance of sleep (see Cases 4 and 5). Nonne, like Hochsinger calls attention to this nervous depletion or "below-parness" of children with syphilitic ascendants; or again, a physical and mental adynamia, or at times a lack of joy and interest in things for one so young.^{21}

A teacher may report of such a child that it has difficulty in attending and learning or that it is easily exhausted on slight brain effort. These children are mentally normal. There are others, however, who show slight enfeeblement (the "debile" of the German and French classification) or backwardness or deeper grades of defectivity running into imbecility. Speech defects of various kinds may occur, and especially often, centrally caused impairment of hearing.

The mental deterioration may be of quite a different character, involving the ethical or moral sense. Though nurtured in a good family and in refined surround-

ings, such an individual may gradually become ungov-
ernable, coarse and even shameless, have attacks of rage, do violent things, lie and steal, and even stoop to prostitution. (Nonne^{28} notes two such cases in girls of good family). These changes come on mostly after the second dentition, and are often associated with mental deficiency. In fact, Hochsinger believes that when such character anomalies develop in early childhood and in connection with lessered intelligence, it as a rule "hangs together" with paternal syphilis. Thus many a juvenile malefactor has to thank his miserable state to the unstable nervous system bequeathed by the syphilis of an ancestor.

The importance of these facts in connection with clinical psychology, of psychopathology and even pedagogies, and especially in relation to juvenile delinquency, is evident, and yet how little do we read of it in our text-books!

Following our careful questioning of the parents as to primary infection, the occurrence of abortions, stillbirths, children dying early, etc., of "secondaries" on themselves (polyglandular swelling, rash, headaches, loss of hair, ulcerated throat, etc.), and finally of "tertiary" manifestations (paralyses, etc.), then as to "secondaries" in the child (the patient) itself, at or soon after birth (snuffles, nasal discharge, indurations, excoriations, eruptions,^{26} scaling skin, pelligus, especially near palmar and plantar regions, "sore finger nails," bone, joint or visceral disease, "eye trouble," and possibly great restlessness, crying,^{20} sleeplessness, a miserable or cachetic state generally, and lastly, convulsions^{31}), we examine our patient for signs of congenital or hereditary lues.

These signs, or "stigmata," as they are called, are sometimes, not always, quite evident, especially in the second period of childhood (later in life some of

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26. When this is noticed chiefly at the time the child is being banded or moved, it is most probably due to bone involvement (esophagitis). A case brought to the clinic had been diagnosed as "neuritis!" Two succeeding children of this family had the same manifestation as their initial syphilitic symptom, in spite of the fact that the mother had been treated (very probably, though, not thoroughly).
27. When a hydrocephalus obtains, these symptoms may be due to it. In a series of such cases studied by Hochsinger, chronic vomiting was also noted. The convulsions may, however, be due to gummatus processes, or meningitis, etc., or it may be a "genuine" epilepsy due to the syphilis but to the defective brain.
them may fade out or disappear). They are the saddle or stump nose, Hutchinson’s teeth (namely, the upper, permanent, inner incisors, notched crescentically on the biting edge), opacities on the cornea (marks of earlier interstitial keratitis[32]) and rhagades, or radial thickenings and scars, peribuccial or especially at the corners of the mouth or junction of the nose and lip (also at times on the fingers near the roots of the nails, and perianal). These rhagades are the most important signs, and Hochsinger speaks of them as unquestionably pathognomonic. The Hutchinson teeth may at times be due to other serious disturbances of nutrition through acute or chronic disease before the advent of the second dentition. When proper treatment is pursued early in childhood, they rarely are found. They are unusual enough as it is, not by any means seen as often as the text-books make it appear. Far more often one finds the two-notched (or three-ridged) type of tooth, there being several of such, or all being of this form. The teeth are undersized and often irregularly “planted.” Difficulty in hearing may also be mentioned here (due to neuritic disease of the acoustic nerve or labyrinth). It is usually double-sided and severe, associated with vertigo and subjective noises. The three conditions of notched teeth, corneal opacity and defective hearing have been described as frequently associated (so-called Hutchinson’s trias).[33]

Heubner[34] pointed out that one may at times also find, added to this trias, a double-seated hydrops, or stiffening of the knee joint. It may occur with little if any pain and cause no increase of temperature; more rarely it may be found with pain, etc., and be somewhat similar to tuberculosis.

Another important set of signs are general undersize (microsomia) including a sexual underdevelopment or delayed puberty (sometimes, in fact, a state of general infantilism[35]), “saber-legs” (tibias with the convex bend, anteriorly), and especially an abnormality of the scapula, termed “scaphoid scapula” (described by Graves of St. Louis, some years ago). Nonne found this the most frequent sign in his series of cases (thirty-one times in fifty-eight children). To this “bone-group” one may add the skull, which may at times give its quota of corroboration. Here one notes a bulging of the frontal eminences or a general frontal enlargement (so-called “Olympic forehead”). The frequent coexistence of rickets, however, should be kept in mind, and rickety “bosses” not mistaken for luetic changes. One must also recollect the possibility of hydrocephalus. Sometimes found, one of the effects of lues.

Thus far enumerated, the signs when present are easily seen and determined without specialized investigation. Nasal and ophthalmoscopic examination, however, may give valuable, if not (as in the latter case) convincing data. The nose may show signs of gummatous destruction of bones and cartilages (hence the “saddle nose”) or septum perforation. Or the soft palate may be ulcerated or even perforated. It has also been pointed out (a fact which, however, helps us nowise in diagnosis), that luetic children frequently have adenoid growths in the nasopharynx, and because of this, enlarged cervical and submaxillary glands. Parenthetically it may here be mentioned, however, that cubital glands, when present on both sides, are mostly syphilitic and of considerable diagnostic value. They may be found at any age up to puberty. In infancy, Götzky[36] considers them almost pathognomonic of syphilis, and in older children decidedly suspicious. At times it may be the only clinical sign of latent lues. Unilateral cubital swelling is mostly not syphilitic. Normally these glands are not felt.

The ophthalmoscope may show pigment deposits in the choroid, rather conclusive evidence of a former syphilitic choroiditis or chorioretinitis, or changes in the retina itself. The iris may show abnormalities, and synechiae may be present. Even an optic neuritis

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32. Lesser (footnote 6) stated that a parenchymatous keratitis may be the earliest, even the only sign, and may in fact first appear in the tenth year (usually, though, between the eighth and tenth). Lademann saw it appear even much later, and states that even at the age of 20 an individual may develop a keratitis on a congenitally luetic basis, never having had any symptoms before.

33. Is this trias really frequent? I have not seen it often, nor have others whom I have questioned.


35. Peritz: Nervenkrankeiten des Kindesalters, p. 466. Peritz shows several good pictures.


37. Cases have been described in which the luetic abnormalities in a child were so mild and atypical, with no patent physiologic signs, that no diagnosis was made until by chance the ophthalmoscope showed this choroidal pigmentation.
may be found. Nor must one forget the possibility of an Argyll Robertson pupil. The vestiges of an old keratitis or one still active may be seen, and this also is of great diagnostic importance.

Before terminating this chapter of signs and stigmata, let me refer to the enlarged spleen, often associated with enlarged liver, frequently found, and finally to the possibility of infantile cerebral paralysis luetic in origin.

The last-named fact is to my mind an important one and ought to be thought of. It is certainly neglected in our textbooks. Fournier found lues in the heredity of nearly all these cases; so did Althaus (cited by Nonne). Of late Bing and Peritz likewise put great stress on syphilis as the possible etiologic factor. Oppenheim also states that hereditary syphilis appears not infrequently to be the cause of this condition. Other writers, however, have not thought that it plays much if any part. But let us consider the following: There are ever so many difficult and protracted, even instrumental labors, and yet in comparison but few cases of infantile cerebral paralysis. Here we must think of some predisposing factor. Again Littel and others noted that ever so many of these cases occurred in premature infants. We can understand how a trauma through pressure or pulling can cause meningeal (venous) hemorrhage, but how is such hemorrhage explained by the easy delivery of a premature infant? (Beside, enough cases occur in which no satisfactory cause whatsoever can be given.) Ziehen mentions the fact that small bleedings may occur, and evidently do, without giving any symptoms, and may occur at times even in normal births. He cites Weyhe as having found such bleedings 122 times in 959 necropsies on infants. In twenty-three of these, lues was present. In the best chapter on the subject that I know of, Heubner analyzes this matter, and also concludes that Littel's etiology is acceptable only in a minimum number of cases and only as a helping or accessory factor. He states that the necropsy studies showed more and more that the brain aplasia when found was not due to meningeal hemorrhage but to simple inhibition of development, and he holds with Ganghofner that the condition is a sign of a certain degenerative condition of the mother. Very interesting also are the findings of Ranke, who noted that the brains of newly born luetic infants gave evidence of considerable structural changes, especially localized about the blood vessels, and which lead to meningo-encephalitic processes. In these foci he found the treponeme. His most important statement, however, is that in these brains, bleeding occurred very easily. If one bears these facts in mind, and also that prematurity, so frequently given as the etiologic moment, is also one of the most frequent accidents in syphilis, and that likewise an endarteritis obtenes often in young syphilitic infants, and may evidence itself as an acute cerebral children's paralysis the luetic etiology (hemorrhage due to luetic arteries, or a meningo-encephalitis or mere aplasia due to inhibition of development) of at least a considerable percentage of these cases must appear to us obvious and acceptable.

Not to be confused with luetic stigmata are the stigmata of degeneracy, which are frequently found in congenitally luetic individuals, but indicate hereditary degeneration, or at least are signs pointing to hereditary degeneration or germ damage, but not to syphilis. Where such germ plasm has been vitiated, the individual resulting from it may show the invalidation of the stuff he is made of in both mental and physical abnormalities: these abnormalities are the tags of his inherited taint or defectivity—hence stigmata degenerations. There are both psychic and somatic stigmata; they may be found in psychotic individuals, idiots, imbeciles, psychopathic constitutions, etc. They have nothing specifically to do with syphilis (and must not be mixed in with luetic stigmata as some writers do) though syphilis may be one of the causes of inherited degeneracy.

Much has been argued about the diagnostic value of these somatic stigmata. Their presence is no index of the amount of inherited psychopathy, for but few may be present in an individual showing severe psychic degeneracy, while at times, on the other hand, one or more may be encountered in the normal individual. On the whole, however, one may see in them with Ziehen, when several are present, decided diagnostic intimation of inherited defect.

38. Oppenheim (Lehrbuch, Ed. 6, Part 2, p. 1281) states that an ophtalmoplegia interna or even just an Argyll Robertson pupil may be the only sign of disease of the nervous system in hereditary lues.
42. Heubner: Lehrbuch, ii, 145; et seq.
44. Heubner: Lehrbuch, i, 653.
45. Findlay and Robertson (Glasgow Med. Jour., bxiiii, No. 6, p. 408) state that, as evidenced with the Wassermann reaction about 45 per cent. of cases of spastic diplegia seem to be due to congenital syphilis.
These stigmata are, for instance, abnormalities of the skull, of teeth, gums and palate, of the external ear, of the hands (webbed fingers or polydactyly, etc.), asymmetries, abnormal hair growth (as across the nose bridge, irregularly into the forehead, especially a double hair spiral), abnormal pigmentation of the iris, abnormalities of the genitals, etc. Possibly left-handedness may be added here, also an abnormally small head, etc.; sometimes even a single stigma may be most meaningful, as the double hair spiral.

Finally a word as to the Wassermann reaction in these cases. No very great amount of work has been done along this line of study, yet enough to give us some valuable information. As to the Wassermann reaction itself, it should be remembered that this is characteristic but not in the strictest sense specific of syphilis. It is occasionally also found in malaria and certain stages of scarlet fever and often in leprosy. Nonne believes that rarely and exceptionally it is found in multiple sclerosis. A negative Wassermann reaction means nothing, just as a positive Wassermann reaction does not mean syphilitic disease, for it may occur in a person who once had lues but is now free of subjective or objective signs of disease, that is, is well. A positive Wassermann reaction means that an individual once was luetic, or that treponemes are still present.

Raven found in 119 children examined of syphilitic parents, 36 to be entirely well, 83 pathologic. Of these 83, only 27 gave a plus Wassermann reaction. Fourteen had it alone, without signs or symptoms, 13 had a plus Wassermann reaction, plus stigmata (of lues or degeneracy).

R. Müller found that late congenitally luetic children all gave a plus Wassermann reaction, if clinical signs were present.

53. Veeder and Jeans also report a case (p. 291) which shows that a negative reaction cannot be regarded as showing that the case is cured or quiescent.
attention in this article and to point out the importance of looking for lues. Veeder and Jeans, for instance, tested three cases with indefinite and obscure pains and found a positive Wassermann reaction. The Roentgen rays have at times shown up luetic bone changes where none were surmised (Dingelmann and Schmetz, also Götzky). The testing of brothers or sisters of children who, for instance, had a keratitis, showed a plus Wassermann reaction when there were absolutely no signs or symptoms. Careful, in fact the most careful history taking is necessary, with all our alertness and sharpness set into activity; and with the aid of the Wassermann (and possibly luetin) test we shall discover a very large number of congenitally luetic whom we never suspected before.

REPORT OF CASES

CASE 1.—M. X., girl, aged 9, at present a generally high-strung alert, sensitive, somewhat restless child, is timid, but has no complaints. Birth was difficult, very slightly before normal end of term; she was cyanotic, and had to be resuscitated, but showed no abnormal signs or symptoms, though weighing six pounds. During the first year of her life she showed no abnormalities, save that she sighed constantly. At the age of 6 she had her first manifestation of chorea, which began with her attending school. This persisted three years (up to the time of coming under my treatment), at times very mild and at others extremely severe. It would be brought out even on a little excitement. During this entire period she would complain of occasional dizziness, "as if her head were going round." Four years ago she began to have difficulty in hearing, which was at first quite extreme, but gradually grew better, and is now normal. For a year she has had frontal headaches, especially on getting up in the morning. Physical examination shows no stigmata, save a mild scaphoid scapula. In the recumbent position a soft systolic murmur is heard at the apex. Mental examination is normal. When the patient was seen a year ago the Wassermann reaction was positive (at present negative). The mother of the child has had tertiary manifestations for five years. The Wassermann reaction was at first positive but now is negative. The father died of nephritis (apparently syphilitic). The child's chorea disappeared very soon on antiluetic treatment. Several physicians must have seen this child (who was always with her mother) while the

mother was being treated for syphilis, yet not one thought of diagnosing syphilis in her, or giving her antiluetic treatment; nor did the treatment she had received for her chorea seem to help her in the least.

CASE 2.—The father of the child was infected three years before marriage; he was treated for neurasthenic headache and cerebral fatigue. He married after several antiluetic "cures." The wife aborted three times, then remained sterile for four years, and finally bore the present patient, who is now 7 years of age; he developed physically and mentally normal, save that learning was very laborious for him, as he could only with difficulty fix his attention. The child was very active, and easily frightened; was a restless sleeper and ate poorly. At the age of 6 he acquired chorea, which would not improve under arsenic, but did rapidly under the administration of potassium iodid, as did also his remaining symptoms. 19

CASE 3.—The father of the patient at the age of 20 was luetically infected. He received several inunction treatments. He died soon after 40 of paresis. The mother, apparently, as is alleged, is entirely well. After one abortion she had four children, of which the first died soon after birth. The other three children (two sons and one daughter) developed physically entirely normally. The eldest of these, however (son) remained behind mentally, and finally had to be given over to an institute for feeble-minded children. The second child (daughter) became chlorotic at puberty. She suffers now from insomnia, nervous tachycardia, and extreme fatigue on bodily and mental exertion. The youngest child (son), who is mentally rather exceptionally developed, suffered since his seventeenth year from habitual headaches, migraine attacks and insomnia. Now, in his twenty-first year, as he was preparing for examination to the bar, he came down with an outspoken neurasthenia. From the report of the family physician, it is learned that this boy, when he was 1 year old, had a periosteal disease of the left tibia, which cleared up on antiluetic treatment. 20

CASE 4.—The mother was strong and healthy. The father was infected eight years previously and was now showing the first signs of tabes. The child, boy, aged 3 years, was mentally alert and physically normal. He was unusually irritable and unsteady. He slept very restlessly, falling asleep only long after midnight—and then awakening again to remain awake for hours. Occasionally also he walked in his sleep. Hydrotherapy, sedatives, change of climate, etc., did not help, but potassium iodid began to improve the con-

\[ \text{S. Cannata (Pediatría, xxii, No. 7) states that the luetin reaction is specific for syphilis. The test is not always positive in congenital lues, but nevertheless gives better results than the Wassermann reaction.} \]

\[ \text{55. Nonne's Case 388, Syphilis und Nervensystem, Berlin, 1909.} \]

\[ \text{56. Case of Binswanger's (see his Neurasthenie, Jens, 1896, pp. 55-56).} \]
dition, which has steadily (after three years) become about normal. 51

Case 5.—Boy, aged 4 years, was brought to Nonne because he slept restlessly at night and had attacks of fear. The child showed nothing on examination, no signs of rickets, degeneration or lues. History of the father showed syphilis in the latter, ten years previously. Sedatives and tonics gave no relief. Under potassium iodid treatment the immediate improvement was striking.52

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