

XLIII.

THE STATIC LABYRINTH IN SYPHILIS.*

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Syphilis, excepting tuberculosis, is perhaps the most prevalent disease with which we as physicians have to deal. Its frequency is startling, its manifestations are legion, and no organ or tissue, from intrauterine life to the grave, is free from the liability of its attack. The discovery and demonstration of the spirocheta pallida, and the Wassermann reaction in the blood and the cerebrospinal fluid, have conclusively proven the syphilitic nature of many diseases which we have heretofore considered and studied as clinical entities; therefore, though we may retain certain names for the sake of classification, we must accept the fact, as one author has stated it, "that these diseases are syphilitis, and always syphilitic."

We know that syphilis of the cerebrospinal system may simulate any disease of the brain and spinal cord substance, their vascular supply and their coverings, and it is only by the examination of the blood or the spinal fluid that we can definitely determine whether or not a central nervous system lesion is syphilitic in origin. A vast amount of laboratory work along this line has shown that brain syphilis is by no means always a late manifestation of the infection, and thereby it has become possible to diagnose lues of the central nervous system long before the appearance of the classic objective signs that we are used to consider as pathognomonic, such as the Argyll-Robertson pupils, the diminished as abolished tendon reflexes, areas of increased sensibility, etc.†

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†Wile and Stokes: Involvement of Nervous System During the Primary Stage of Syphilis. *Journal A. M. A.*, March 20, 1915, p. 979. Six cases of early syphilis with involvement of nervous system in four.

Collins: Brain Syphilis. *Journal A. M. A.*, July 10, 1915, p. 139 (Vol. LXV, No. 2).

Such signs in fact belong to a later stage of the infection, and indicate that an irreparable destruction of the parenchymatous cells has already taken place; whereas in the early stage of an invasion, it is vascular structures and the meninges which are involved, and though it does not necessarily follow that a basilar meningitis of syphilitic etiology must lead to an involvement of the brain or cord substance, there is always this possibility, which undoubtedly may be the better averted the sooner appropriate treatment is instituted.

The subject of brain syphilis is of prime importance to the otologist, for the reason that the preponderance of evidence in many investigations goes to show that the eighth nerve is more often affected in all stages of syphilis than any other of the cranial nerves; that the lesion is probably a true neuritis of the eighth, its nuclei, its trunk, or its peripheral distributions; and that its involvement is indicative of the luetic invasion of the central nervous system.

For many years we have recognized that certain forms of deafness were due to hereditary syphilis. The first contribution to the subject was probably published by Feichtman¹ in 1857, though the classic paper of Hutchison and Jackson² may be considered the pioneer work, especially as these authors correctly defined the nerve deafness which is characteristic.*

It is not only in hereditary syphilis, however, that the auditory nervous mechanism may become involved. Habermann³ in 1896 reported sixty-six cases of syphilis of the internal ear, of which thirty-four were attacked during the secondaries. Mayer⁴ in 1911 reported sixty-five cases of syphilitic internal ear disease, thirty within the first year of the infection, thirteen within from three to ten weeks following the primary sore. Politzer⁵ reported one case occurring seven days after the chancre. All these cases, and many more by various authors, were reported before the advent of salvarsan, to the use of which so many cases of deafness has been accredited. It is far from the purpose of this paper to reopen that controversy, for it is now pretty well understood that insufficient

*C. Ernest West has given a complete historical review of the subject in his chapter "Aural Syphilis" in the monumental "System of Syphilis" edited by D'Arcy Powers and J. K. Murphy.

treatment with the arsenical preparation may do harm, although the extent of this has undoubtedly been exaggerated. In 1915 W. W. Willcut⁶ presented, before the American Medical Association, a report of two hundred and ninety-three syphilitics examined by him in Urbantschitch's Clinic. None had had salvarsan. In fifty-six cases of a month's duration or less he found fifty-two showing the characteristic deafness of syphilis. All had negative Wassermann reactions, but the diagnosis was certified by the demonstration of the spirocheta pallida in the primary sore. G. P. Wintermute⁷ reported three cases of acute syphilitic acoustic neuritis before the same session of the American Medical Association (San Francisco, 1915), and gave complete and valuable data concerning the symptoms, course and treatment of these conditions. The cases studied, both by Willcut and Wintermute, are most conclusive, and the bibliographies furnished by these authors are very complete. Evidence is surely not lacking that the eighth nerve is often affected in primary syphilis, nor is proof wanting that it is also often involved in the late syphilides, and that aural symptoms may be one of the first signs in tabes dorsalis and general paresis. Ellis and Swift⁸ found deafness of the nerve type in forty per cent of a series of tabetic cases. Shaller⁹ found the internal ear involved in sixteen cases out of thirty-five patients with early tabes. Deafness is now mentioned by all neurologists as one of the symptoms often found in the preataxic stage of tabes dorsalis.

Naturally, there have been many different views respecting the portion of the ear and of the nervous apparatus of the ear that is affected in syphilis. Sexton¹⁰ attempted to show that the deafness in syphilis was due to lesions in the middle ear, but this contention was completely refuted by St. John Roosa.¹¹ Moos¹² in 1887 found changes in the vestibule, in the organ of Corti, and in the ampullæ of the semicircular canals, but a normal nerve trunk, in a patient with secondary syphilis who had died soon after ear symptoms had arisen; and thus the trend of opinion from then on was to consider the ear lesions of the secondaries as intralabyrinthine.

Deafness occurring in tertiary syphilis was studied by Rosenstein,¹³ who found lesions in the nerve stem, in the nuclei, in the roots, and in one case in the roots alone (Ellis

and Swift⁸). Mannasse¹⁴ has given a most complete picture of the morbid anatomy found in a patient with tertiary syphilis who had been deaf for one year before death. He found changes in the nervous structures of the cochlea, of the semicircular canals, and also in the nerve trunk. In tabes with deafness Habermann¹⁵ found gray atrophy of both auditory nerves, and atrophy of the nerve endings in the cochlea and in the semicircular canals. The nuclei were intact, the lateral and median acoustic roots much atrophied. Bruehl¹⁶ found degeneration in the nuclear region of the eighth nerve. Mayer¹⁷ studied five cases of general paresis with deafness, and found atrophic changes in all the nervous elements of the ear from the organ of Corti to the medulla. Rosenstein¹⁸ believes that deafness in inherited lues is due, in many instances, to a basal meningitis, though other observers place the lesion within the labyrinth. In 1912, Knick and Zaloziecki¹⁸ examined the spinal fluid in nine cases of early syphilis with deafness, and found a high cell count in all nine and a positive spinal fluid Wassermann in eight. They claimed, therefore, that the deafness in early syphilis is due to a syphilitic meningitis which involves the eighth nerve.

Ellis and Swift⁸ corroborated this work in seven cases of their own, and concluded "that all lesions of the eighth nerve are possible manifestations of a disastrous form of a general infection—syphilis of the central nervous system." Willcut⁸ believes that the involvement of the cranial nerves in early syphilis is due to a toxic irritation of the nerve sheath and the peripheral end organs. He mentions the eighth, the first and the seventh nerves, and says: "The acoustic, for some unexplainable reason, appears to be the most sensitive of all the cranial nerves." Willcut and many authors seem to overlook the anatomic peculiarity of the eighth nerve being devoid of neurilemma; it is, therefore, all the more liable, on account of its position, to become inflamed during the course of a basal meningitis or from toxic substances circulating in the cerebrospinal fluid. It is hardly possible to doubt the observations of any of the authors quoted, consequently, though we must accept the conclusions that the lesion is to be found in the nerve tissue, we must also accept the fact that the eighth nerve may be attacked centrally, axially or peripherally;

therefore, the careful clinical study of this particular class of patients by the newer and more accurate methods of otologic examination should put us well on the way to that precision in diagnosis which Robert Bing says is wanting—namely, the differentiation between lesions of nerve or its supranuclear tracts and lesions of the percipient structures in the internal ear. It is possible to separately examine both the cochlea branch and the vestibular branch of the eighth nerve, and in the latter tests the phenomenal work of Bárány has been so amplified by the methods and teachings of Randall, Jones, Fisher, Brumm, and others of the “Philadelphia School,” that localization of eighth nerve lesions has certainly passed beyond the stage of hypothesis.

It is generally taught that if the cochlea division of the eighth nerve is found to be normal, the so-called static labyrinth and the vestibular nerve will also be normal. In syphilis, however, it is possible for the vestibular branch to become involved at some point along its course, while the cochlea branch may remain normal. That the reverse may be true is admitted by all. In a large percentage of syphilitic cases both divisions of the eighth nerve will be affected and, therefore, both the auditory and the static labyrinth should be tested. It should be remembered that patients with the acoustic neuritis of syphilis may hear the voice surprisingly well and may not complain of deafness until questioned; the tuning forks, therefore, offer the best means of correct diagnosis in these cases.

The typical hearing defect is a shortening of the duration of perception by bone conduction, out of all proportion to the shortening of the duration of perception for the same fork by air conduction; the retention of good hearing for the low forks, with a loss of perception, or a reduction in duration of perception, for sounds of high pitch. All tuning fork tests should be made under the correct law of decrement of the vibrations (Dundas Grant¹⁹); therefore, we should attempt to determine the shortening in the duration of perception as compared to the normal, and not simply the duration of perception itself.

The Schwabach test is best made with the opposite ear excluded by a “noise apparatus” regulated to that intensity

which is just sufficient to prevent contraauidition. If the Rinné is made by separate tests of bone and air conduction, it is not infrequent to find that bone conduction is actually reduced out of all proportion to the reduction of perception by air conduction, as physically impossible as this may seem, and such a reaction is indicative of an acoustic neuritis rather than of an intralabyrinthine lesion. I have found the C⁵ (4096 d. v. s.) fork of more use in routine work than the Galton whistle, for the reason that the intensity is so great with the latter instrument that partial defects in high register are difficult to determine.

No matter with what care we may attempt to determine reductions in audition, we must always depend upon purely subjective methods. The tests of the vestibular apparatus are to a large degree objective, and in normal individuals the reactions are surprisingly constant, when the tests are made with precision and with due regard to details. In the rotary tests the after-nystagmus should last for twenty-six seconds, the patient having been turned ten times in twenty seconds. The vertigo should last under the same conditions for twenty-four seconds. Nystagmus should be induced in forty seconds by douching the ear with water at 68° F. These are the normal responses found by I. H. Jones, who has further simplified the interpretation of the reactions by giving us the very easy rule to remember, that the nystagmus, the falling and the past-pointing, are all in the direction of the endolymph flow, with the vertigo in the opposite direction. Dr. Jones and his associates further teach that each semicircular canal has a separate nystagmic and vertiginous tract. They have been able to locate approximately, and to postulate, the situation of these pathways in the brain stem, the cerebellum and the cerebrum; hence; their methods of localization in the eighth nerve lesions, and in intracranial conditions involving the eighth nerve, are the most accurate that we have.

In the last eighteen months I have had the opportunity of making an ear examination of a creditable number of patients with syphilis and syphilitic central nervous system lesions. A number of cases have been examined in the neurologic clinic of the Mercy Hospital, and I am greatly indebted to the neurologist-in-charge, Dr. A. C. Gillis, for his cooperation. Many

of these patients did not complain of ear symptoms, though it was not difficult to demonstrate an associated aural involvement. A number presented predominant internal ear disturbances, and in a few instances the examination of the ear led to the diagnosis of serious diseases of the central nervous system. Though the auditory function was disturbed in most of the cases, the tests of the static apparatus, made by rotation and cold douching, were of far greater value in absolutely confirming the presence of a lesion in the nervous structures of the ear than were the tests of audition.

Generally speaking, the abnormal response of the static labyrinth in syphilis may indicate a lesion of the vestibular nerve at any point from its nuclei to its peripheral distributions. If we may have "islands of deafness," we likewise may have islands of static inhibition; if these defects are situated neuraxially, the reactions from the vestibular tests may simulate those seen in brain stem, cerebellar or cerebellopontine tumors. As a rule, however, the syphilitic responses indicate involvement of the eighth nerve trunk or its peripheral distributions, and the neuraxial symptoms are likely to be so atypical that they indicate lesion of such widely separated areas that simultaneous involvement of these tracts by a new growth would be impossible, whereas a degeneration of these different pathways is not at all improbable. The most characteristic reaction of syphilitic internal ear disease is a lowering and confusion of all the responses; this may vary from the totally dead labyrinth, giving no responses, to the cases showing all the normal reactions reduced in degree. For example, the nystagmus will last but ten to fifteen seconds or less, and will show fine oscillations in which the slow and quick components cannot be easily differentiated. Vertigo is absent, or it lasts but a few seconds. Falling is not definite. The patient, if tested quickly after rotation, will past-point with one arm and not with the other, or will only past-point for a few inches, or will past-point incorrectly. The responses may be intensified by increasing the stimulation; thus, nystagmus may be absent after ten turns in twenty seconds, to become evident for some seconds after ten turns in ten seconds. Douching with water at 68° F. will bring on the same reduced or confusing reactions after a much longer period than the normal forty seconds, or it may require

much colder water to bring out any response. Furthermore, it is apparently possible in these cases for one semicircular canal of the same ear to be more affected than the others; hence, we may get all normal responses from rotation with the head in the upright position (horizontal canals), and no reactions with the head forward (vertical canals), or vice versa. Such a lesion would have to exist in the crista ampullaris of one canal only, or as a partial degeneration in the nerve trunk itself, before the nystagmic and vertiginous tracts separate.

Increased irritability of the static labyrinth, evidenced by prolonged nystagmus, with marked vertigo and nausea, is a sign which may not be overlooked. Crossed past-pointing, right arm to the left, and left arm to the right, after turning to the right, and vice versa, has been noted, as has complete reversal of the past-pointing, both arms to the left, after turning to the right, and both arms to right after left turning. Variations from time to time in all the responses must be expected, and may be of value in following the progress of retrogression of the syphilitic process. The reactions, in most instances, point to the ear which is the more involved, and as a rule both are implicated.

I have been able to study twenty-eight patients in whom the diagnosis of syphilis has been positively established, and though the number is far too small to be of statistic value, the clinical data obtained has indicated the general signs just enumerated. Certain groupings of these cases, and individual histories, are not without interest. The histories are arranged in two series: first, those patients in whom ear disturbances were the complaints for which the patient sought relief; second, those patients with central nervous system diseases upon whom a routine ear examination was made, irrespective of aural symptoms. For the sake of brevity, a synopsis of the salient features are given, and details are reported in full only when they have seemed essential.

There are twelve patients in the first group, who came complaining of deafness, tinnitus and vertigo, the frequency of the complaints being in the order named. Deafness and tinnitus were usually both present, but vertigo was not voluntarily mentioned, though histories of slight attacks were elicited in

five cases. A shortening of ten seconds or more in the duration of perception by bone conduction for the C² (512 d. v. s.) tuning fork was the basis for further examination of the internal ear, and in every patient under discussion, both the cochlea and the vestibular functions were found to be impaired. Five of these patients were the victims of hereditary syphilis, four of whom had interstitial keratitis. These five cases show practically all the variations of syphilitic eighth nerve involvement.

Case 1.—Marie P., colored, aged nine years. Admitted December 6, 1916. Hereditary syphilis. Interstitial keratitis in active stage. Positive blood Wassermann. Sudden and practically complete nerve deafness of both ears, of three weeks' duration. Both static labyrinths without response in any particular or from any test. No improvement under vigorous mercurial treatment. When the patient was first seen there was a slight amount of hearing present; this has now been destroyed. The child enjoys being literally whirled in the chair, in any position, and can immediately stand erect without a tremor.

Case 2.—Sydney D., white, aged seventeen years. Hereditary syphilis. Incomplete nerve deafness of three years' duration. Positive blood Wassermann. Static labyrinths markedly affected on both sides, though the right ear is the deafer of the two.

Right turning (head upright): Nystagmus for ten seconds. Vertigo for twenty-five seconds. Past-pointing negative in both arms.

Left turning (head upright): Nystagmus negative. Vertigo for ten seconds. Past-pointing of right arm negative. Left arm to right a few inches.

Both ears negative to cold douching in all positions.

Case 3.—Edith L., white, aged sixteen years. Hereditary syphilis. Incomplete nerve deafness of the luetic type, more marked in right ear. Static labyrinths involved. Blood Wassermann positive.

Right turning (head upright): Nystagmus for twenty-five seconds. Vertigo present, but patient complained more of nausea. Past-pointed with right arm correctly to right. Left arm negative.

Left turning (head upright): Nystagmus for fifteen seconds. Vertigo slight. Both arms failed to past-point.

With the cold caloric, nystagmus could be induced from both the horizontal and the vertical canals of both ears. Nausea more marked than vertigo. Past-pointing all negative, except that left ear stimulation of both the horizontal and the vertical canals caused the left arm to past-point correctly.

Case 4.—Mary B., white, aged seventeen years. Admitted July 11, 1916, complaining of deafness of eighteen months' duration, more marked in right ear. Hearing tests showed incomplete nerve deafness of luetic type.

Right turning (head upright): Nystagmus for eight seconds. Vertigo negative. Past-pointing with right and left arm, three inches to right.

Left turning (head upright): Nystagmus for three seconds. Vertigo negative. Past-pointing with right arm negative; with left arm, three inches to right.

The girl has a brother totally deaf from measles (?). Her teeth are Hutchison in type. Her blood Wassermann was positive.

Case 5.—John M., white, aged ten years. Interstitial keratitis in subacute stage. Deafness complained of. Impossible to make satisfactory hearing tests. Right turning gave nystagmus for fifteen seconds. Left turning did not arouse nystagmus. Blood Wassermann positive. Although the tests are incomplete in this case, there is sufficient evidence to show that the cochlea and the static functions are both disturbed.

In cases 2, 3, 4 and 5, the hearing is not markedly impaired. All these people can hear the conversational voice without much difficulty. The vestibular tests have been of far more value in correctly determining the nervous character of the aural disturbances than have been the tests of the audition.

The other seven cases in this group were patients with acquired lues, from whom it was impossible to obtain a history of the primary lesion, except in two instances. The value of the ear tests in this particular may be demonstrated by the citation of one case.

Case 6.—Katherine H., white, aged seventeen years. Admitted August 1, 1916, complaining of deafness of the right ear, which had started July 6th. The hearing tests were very un-

certain, but the duration of perception by bone conduction for the C² fork seemed shortened about thirteen seconds in the right ear, and ten seconds in the left.

Right turning (head upright): Nystagmus for twenty-two seconds. Vertigo very slight. Right arm past-pointed to left. Left arm did not past-point.

Left turning (head upright): Nystagmus for seven seconds. Vertigo negative. Right arm did not past-point. Left arm past-pointed to right.

The caloric tests and turning with the head in various positions gave confusing, conflicting and abnormal responses. No history could be obtained from the patient. Her blood Wassermann, however, was positive; and her hearing rapidly improved under antisyphilitic treatment.

The next two cases are of interest from the valuable indication given by the ear examination of serious central nervous system involvement.

Case 7.—Deafness. Optic atrophy. Argyl-Robertson pupils. Negative blood Wassermann. Positive spinal fluid Wassermann. Tabes dorsalis.

Louis S., white, aged forty-four years. Admitted June 12, 1916, complaining of deafness, more marked in the left ear. The duration of perception by bone conduction for the C² fork was minus twenty-five seconds; duration of perception by air conduction, minus fifteen seconds. The duration of perception by air conduction for the C fork was minus ten seconds; for the C⁵ fork, minus five seconds. The right ear showed a slight reduction in the bone conduction. The patient said that his eyes were also bad, and ophthalmoscopic examination disclosed a beginning primary optic atrophy, more marked in the left eye. Vision right eye, 18/20; left eye, 18/49. Pupils unequal, irregular and sluggish to light. Patellar reflexes absent. The man admitted a primary lesion occurring twenty-five years ago.

Right turning (head upright): Nystagmus for twenty-five seconds. Vertigo and nausea. Right arm did not past-point. Left arm past-pointed to right.

Left turning (head upright): Nystagmus for twenty-five seconds. Vertigo and nausea. Right arm did not past-point. Left arm past-pointed to right.

The diagnosis of tabes was simple enough in this case, and, of course, was not dependent upon the ear examination. After intravenous and intraspinal treatment with neosalvarsan, the eye condition improved slightly, and the responses of the static labyrinth became perfectly normal. Unfortunately, we were only able to follow this case for six months when he disappeared from the clinic, and so far we have not been able to locate him.

Case 8.—Nerve deafness. Marked increase in the duration of the after-nystagmus with nausea and vomiting. Negative blood Wassermann. Strongly positive spinal fluid Wassermann. Tabes dorsalis.

Ella R., colored, aged thirty-eight years. Admitted July, 1916, complaining of deafness of the right ear of four years' duration, of the left ear of a few months' duration. The bone conduction of the right ear for the C² fork was negative; the air conduction, minus twenty-five seconds. The C fork was shortened twenty seconds in duration of perception, the C⁵ fork could not be heard. The left ear showed bone conduction, C² fork, minus twenty seconds; air conduction for the same fork, minus thirteen seconds. The C fork was but slightly shortened, the C⁵ fork was normally heard.

Right turning (head upright): Nystagmus for fifty seconds. Vertigo, nausea and vomiting. Past-pointing normally to right with both arms.

Left turning (head upright): Nystagmus for forty-five seconds. Vertigo, nausea and vomiting. Past-pointing normally

The cold caloric brought on the same severe reactions from both ears in all positions of the head. The blood Wassermann was negative.

The woman was put on mercury for a week, and at the end of that time the Wassermann was again negative. She drifted away from the clinic to return in January, 1917, complaining that her hearing was worse, and that she had headache and "stomach pain." Her pupils were now small and sluggish to light, while before they had been normal, and she was strongly urged to have a spinal puncture, but refused. On May 10, 1917, she consented to the examination, and the spinal fluid examination was as follows: Cell count, thirty-one per cubic

centimeter. Wassermann strongly positive. Ross Jones and Pandy, positive. Diagnosis, tabes dorsalis.

The four remaining cases in this group are not without interest, but they shall have to be dismissed with mention only. All had an acoustic neuritis of syphilitic origin, with involvement of the static functions.

From the work done at the Mercy Hospital I have selected sixteen cases for study, which may be grouped under the heads of the neurologic diagnosis of cerebral syphilis, tabes dorsalis, and general paresis.

Seven of these patients may be classified as cases of early brain lesions, six of whom came to the hospital with the complaint of severe attacks of vertigo, and the examination of the ear showed the static labyrinth to be definitely involved in five out of six. The patient who did not complain of vertigo had had an involvement of the ninth, tenth and twelfth cranial nerves. His static labyrinths were apparently normal; the tests, however, were not as complete as I should like them to have been, and he did show a definite "island of tone deafness" for the C³, C⁴ and the C⁵ forks, although he heard the upper range of the Galton whistle.

One patient was seen the day following a severe attack of vertigo. Certain details of her history are of interest.

Case 9.—Annie M., colored, aged twenty-nine years. Admitted March 1, 1917, complaining of severe headache and vertigo. The night before, she had fallen and had to be carried to bed. She gave a definite history of lues, the primary lesion having occurred in 1910. Examination showed a positive Romberg. Tenderness on palpation over skull. Neurologic diagnosis, syphilitic meningitis. Her ear drums were normal; her hearing tests showed a reduction in the perception by bone conduction of twenty seconds in both ears. All other tuning fork tests apparently normal.

Right turning (head upright): Nystagmus for twenty-three seconds. Vertigo for fifteen seconds. Past-pointing negative with both arms.

Left turning (head upright): Nystagmus for twenty-one seconds. Vertigo uncertain. Past-pointing negative with both arms.

Two days later, right turning gave nystagmus for twenty-

one seconds; left turning, nystagmus for twelve seconds. One week later (March 28, 1917), with the patient much improved generally, right turning, nystagmus twenty-two seconds; left turning, nystagmus, fifteen seconds. The patient felt so much better that she did not return to the clinic and could not be located. It would seem, from this case, that the vestibular tests offer a most accurate means of following a process of this kind.

I can only touch upon the findings in the other patients of this group, as anything less than complete histories would be of little value. The presence of a positive Romberg in these patients is suggestive of the aural nature of this sign.

Case 10.—Wm. G. M., white, aged twenty-five years. Admitted July 27, 1916, complaining of severe vertigo of three weeks' duration. Romberg positive. Neurologic symptoms pointing to a cerebellar tumor. Spontaneous vertical nystagmus on looking up. Hearing normal. Vestibular tests indicative of a neuraxial lesion, but exceedingly variable and changing from day to day. The patient denied lues, and his blood was negative to the Wassermann test on two occasions. The spinal fluid showed cell count, forty-two; Wassermann inconclusive; globulin test positive. On August 30, 1916, the patient became suddenly and completely deaf in the right ear. He died September, 1916, and the postmortem examination showed an old syphilitic meningitis with a degenerated gumma in the middle lobe of the cerebellum, extending to both lateral lobes, particularly of the right side, and involving the region of Deiters' nucleus.

Case 11.—Chas. L., white, aged forty-seven years. Admitted August 10, 1916, complaining of sudden and complete deafness of both ears and of severe vertigo of ten days' duration. Romberg positive. Neurologic symptoms indicative of a lesion in the region of the corpora quadrigemina. Spontaneous nystagmus to right. Vestibular tests indicative of a neuraxial lesion, though the responses were uncertain and not of great localizing value. Blood and spinal fluid showed strongly marked positive Wassermann. The man left the hospital, but died in a few weeks. No postmortem obtained.

Case 12.—M. M., white, aged thirty-five years. Admitted July 6, 1916, complaining of vertigo and deafness. Typical acoustic neuritic defect in hearing. Nystagmus endured for

forty-five and thirty-five seconds with the head upright, and vertigo and past-pointing were normal. With the head forward, right turning elicited neither nystagmus, vertigo nor past-pointing. On left turning the reactions came, though normally. Positive blood Wassermann.

Case 13.—Louis M., white, aged forty-two years. Admitted November 10, 1916, complaining of vertigo, deafness and tinnitus. Romberg positive. Vestibular tests showed reduced nystagmus from both ears, and no vertigo or past-pointing. Hearing defect of luetic type. Primary lesion in 1913. Positive Wassermann.

Seven cases of *tabes dorsalis* in various stages have been studied. In four of these both the cochlea and the vestibular branches of the eighth nerve were involved. The cochlea branch alone in one, the vestibular branch alone in two. These patients were not markedly deaf; indeed, a history of hardness of hearing could only be obtained in three of the cases. Four of these patients had primary optic atrophy. Generally speaking, the aural defects were not of wide extent, but they were none the less striking.

Case 14.—Ethel W., colored, aged thirty-five years, with *tabes* and optic atrophy, showed normal nystagmus in both directions, but on right turning the right arm past-pointed correctly to the right, while the left arm past-pointed to the left. On left turning she past-pointed with the right arm to the right, and with the left arm to the left. This past-pointing error was present in all positions of the head.

Case 15.—Harry T., white, aged thirty-six years, with *tabes* and optic atrophy, showed nystagmus lasting for twenty seconds in both directions, but on right turning he past-pointed with the right arm to the right, and with the left arm to the left. On left turning the past-pointing of both arms was correctly to the left. The error of the left arm on right turning was present in all positions of the head.

Case 16.—Columbus A., colored, aged fifty years, has been under observation by Dr. Gillis for several years. He presents a cerebellar type of *tabes dorsalis* with a positive Romberg. His hearing is normal. He has a spontaneous nystagmus, vertical in type, on looking up, and he shows but one abnormal vestibular response—viz., a failure in past-pointing corresponding to a brain stem lesion.

One case of general paresis showed complete reversal of the past-pointing.

Case 17.—Minnie S., white, aged thirty-eight years. Neurologic diagnosis: General paresis. Positive Wassermann in spinal fluid. Hearing normal. Romberg suggestive.

Right turning (head upright): Nystagmus very fine in type, apparently lasting for twenty seconds. Both right and left arms past-point to the left.

Left turning (head upright): Nystagmus the same as in right turning. Both arms past-point to the right.

The error in past-pointing is present in all positions of the head.

Finally, I must beg indulgence for a work of such magnitude from which, as yet, I feel incapable of drawing any but the most general conclusions. Syphilis, as a cause of internal ear diseases, is a factor which may not be considered lightly, or as a rarity; and as the static mechanism, as well as the auditory apparatus is often involved, we as otologists have, in the vestibular tests, a means by which an early perversion of the eighth nerve function may be detected. There probably are no pathognomonic vestibular responses in syphilis, but the very confusing and conflicting reactions are in themselves indicative of multiple lesions, a characteristic of any syphilitic invasion. If syphilitic neuritis of the eighth nerve always points to an involvement of the central nervous system, and in one sense this must be true, then, in all justice to the patients, they should command the combined knowledge of the neurologist, the syphilographer, and the otologist, for Ellis and Swift have shown that the prognosis need not be so grave if treatment is instituted in time.

BIBLIOGRAPHY.

1. Feichtman: *Zelt. f. Naturh. v. Hellk. in Ungain, Oldenberg*, 1857.
2. Hutchinson and Jackson: *Med. Times*, 1861, 1.
3. Habermann: *Die luetischen Erkrankungen des Gehororgans*, Jena, 1896.
4. Mayer: *Wien. med. Wchnshr.*, 1911.
5. Politzer: *Lehrbuch der Ohrenheilkunde*, Stuttgart, 1908.
6. Willcut: *Disturbances of the Acoustic Nerve in the Early Stages of Syphilis*. American Med. Assoc. Transactions, Section on Otol., Laryn. and Rhin., 1915.
7. Wintermute: *Auditory Neuritis*, *ibid.*

8. Ellis and Swift: Involvement of the Eighth Nerve in Syphilis of the Central Nervous System. Jour. Amer. Med. Assoc., May 1, 1915, Vol. 64, No. 18, p. 1471.
9. Shaller: Early Diagnosis of Tabes Dorsalis. Amer. Med. Assoc. Transactions, Section on Neurology, 1916.
10. Sexton: American Journal of Medical Sciences, 1879.
11. Roosa, St. John: Archives of Otolology, 1879.
12. Moos: Arch. f. Path., Anat. u. Physiolog., Virchow, 1877.
13. Rosenstein: Arch. f. Ohrenh., 1905, Vol. 65, 193.
14. Manasse: Archives of Otolology, 1903.
15. Habermann: Arch. f. Ohrenh., Vol. 33.
16. Bruehl: Deutsch. Otol. Gesethch., June, 1907.
17. Mayer: Arch. f. Ohrenheil., Vol. 72, 1.
18. Knick and Zaloziecki: Berl. klin. Wchnschr., 1912, Vol. 49, 639.
19. Grant, Dundas: Transactions of the Ninth International Otological Congress, Boston, 1912.