[*Note*: *Due to the age and condition of the original paper, some of the text of this paper was either illegible or outright missing. In these cases, we have noted the missing sections. We present the paper in any case as most of the paper is present.*]

Deutsche Zeitschrift für Nervenheilkunde, 74:251-259, Leipzig, 1922.

Etiology of Multiple Sclerosis

Max Gerson, Bielefeld

The etiology of multiple sclerosis is still unclear. In the last few years a large amount of research material has been published: pathological-anatomical investigations by Siemerling, Raeke, Westphal, Merzbacher, Spielmeyer and others, experimental trials by Simons, Kuhn, Steiner, Hauptmann, Marinesco, Rothfeld, Freund, Hornowski and Kalberlah and clinical summaries and discussions on this issue by Friedrich Schulze, von Strümpell and others, repetition of which is superfluous here. The decision is mainly influenced by pathological-anatomical findings, leaning towards an infectious disease, even if doubts by Strümpell remain regarding contraction, the absence of infection and the fact that internal organs are unaffected. A series of serious investigators could not confirm the findings on spirochetes, whereas some only found vague indications. In theory, Nonne drew attention to some of the inconsistencies arguing against hypothetic spirochetes. In addition, a classical picture of multiple sclerosis can occur due to syphilis. Casirer succeeded in proving anatomically real multiple sclerosis with syphilis present. It remains doubtful, whether the numerous clinical images of multiple sclerosis are due to only one uniform etiological moment.

This uncertainty causes me to communicate several clinical observations made in 11 cases of multiple sclerosis.

The symptoms here are shortened, indicating only the most striking pathological changes. The case histories only mention the course of the illness.

Listed below is the onset the illness of two cases

Case 1: W. St. 17 years old. Has been suffering from headaches and increasing weakness in arms and legs for six months. Rapidly fatigued.

In the beginning stages, but significant temporal pallor of the left eye (optician Dr. Kühn). Conjunctiva reflexes absent on both sides, crossed double images, internal insufficiency. Minor ataxia is present in the right eye. Both upper abdominal reflexes weak, the others are missing. Right knee-jerk increased significantly, right Oppenheim positive. Trace protein in the urine. No findings in the sediment.

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1) Diseases of the spinal cord and peripheral nerves 1920. S. 33.
2) Also refer to paper by I. Schuster, "Ztschr. F.d. ges. Neurol u. Psych. Ed.65

Case 2: I. L., 20 years old. Has been receiving treatment at the Municipal Hospital in Bielefeld for some time for a stomach ulcer. Showing temporal pallor on both sides and nystagmus. Abdominal reflexes missing completely on the right-, only weak on the left hand side.

Five moderately severe cases

Case 3: Miss E. T., 28 years old. Has suffered from weakness in the left leg, later on the right leg after allegedly contracting influenza.

Abdominal reflexes are missing completely on both sides. Patellar clonus on the left-hand side, right-hand side patellar reflex increased. Foot clonus on both sides, no Babinski reflex present. Spasms and ataxia are present in both legs, mild degree of spastic-paretic walk. Feeling in the right big toe joint when positioning foot is not present. Mild intension tremor present on the left-hand side. Wa. R. in the blood.

Case 4: Miss L. B., 35 years old, 1). Experienced increasing weakness in the left leg 3 years ago. For the last 3 months also in the right leg. Also suffering from general physical tiredness and mental fatigue at work – teacher.

Mild nystagumus, no abdominal reflexes present on the left-hand side. Patellar reflex increased on both sides. Hamstring reflex increased on the right-hand side, foot clonus on the left-hand side. Obvious Babinski is present on both sides. Force of foot and toe flexors reduced on the left-hand side. Upon standing clawing of the toes on the left foot. When walking left leg slightly limping. Mild spasms are present in both legs, obvious ataxia on the left-hand side.

Case 5: I. L., 33 years old. Suffered from impaired vision 5 years ago, which improved. Suffered headaches, dizziness, weakness in the left arm 3 years ago, improving later. Has been suffering from weakness all over the body, dizziness and difficulty walking for approximately 1 year.

Temporal pallor and nystagmus are present. Speech is slurred. Abdominal reflexes are missing completely on both sides. Patellar reflex increased on both sides, foot clonus on the left foot, left hamstring reflex increased, Babinski on the left-hand side. Obvious signs of Romberg: uncertain, spastic-paretic walk. Wa. R. was found in the blood and liquor. Liquor shows minor increase in protein, few lymphocytes.

Case 6: A. B., 44 years old. Fell ill in the field with back pain in 1917. Walking became very difficult and only possible using a walking stick. Walking has improved since 1920, but becomes rapidly fatigued. Has difficulty speaking from time to time. Sad, upset and without energy most of the time.

1) It is worth noting that the younger sister is suffering from an even further advanced form of multiple sclerosis.

Nystagmus is present when looking to the right. Speech somewhat slurred. Upper abdominal reflex on the left-hand side is weak, middle and lower abdominal reflexes are missing. The left-hand cremasteric reflex is weak, the right-hand one can hardly be triggered. Both patellar and hamstring reflexes significantly increased, no clonus, obvious Babinski on both sides. Spasms and ataxia are present in both legs. Sensation to touch and sting reduced throughout most of the right leg. Periosteum and tendon reflexes are increased in both arms, obvious intention tremor. Wa. R. in the blood as stated. Puls accelerated.

Case 7: C. B., 38 years old. Suffered from rheumatoid arthritis at 18 years of age. Suffered from pain and weakness in the legs in the field in 1916, which improved after a short time. In 1917 experienced difficulties walking and speaking and tremor of the arms.

Nystagmus is present when looking to the right. The upper right abdominal reflex is normal, the upper left reflex is weak. All other reflexes are very weak. Cremasteric reflexes are very weak on both sides. Patellar reflexes increased on both sides. Hamstring reflexes normal on both sides. The plantar reflex is very weak on the right-hand side, Babinski present on the left-hand side. Strong ataxia in both legs, walk is spastic-paretic, with a limp in the right leg. Periosteum and tendon reflexes increased in both arms, obvious ataxia. Sensation to touch and sting reduced throughout both legs. Euphoria. Wa.R. in the blood.

Three severe cases

Case 8: F.M., 38 years old. In 1917 suffered from a weakness in the left hand and slight speech disorder. Soon after, a tendency to unmotivated laughing or crying. At the end of 1918 weakness and stiffness in both legs – with the left leg being more affected. Progressive deterioration with spasmodic forced laughter, euphoria and, from time to time, double vision. For the last quarter of a year, the temperature was between 37.3 and 37.6 every afternoon.

Temporal pallor on both sides and nystagmus when looking to the right. Tongue deviates to the left. Slowed speech, chanted. The left arm is severely spastic, mild atrophy of the interossei, increased periosteum and tendon reflexes on both sides, with the left-hand side being more affected. Passive movement in the left leg is very difficult. Abdominal reflexes on both sides are missing. Patellar reflexes are normal on both sides. Hamstring reflex on the right-hand side is weak, increased on the left side, sometime foot clonus. Babinski is present on both sides. Active movement in the left leg is hardly possible, somewhat better in the right leg. Walk is very spastic-paretic, cannot walk alone unaided anymore. Suffering from ataxia in the arms and legs. Feeling of positioning is missing in the left toe joints and the end joints of the left finger. Wa.R. in the blood and liquor. Liquor shows a slight increase in protein and lymphocytes.
Case 9: W. O., 37 years old. Suffered from fever and a cold in 1914, which was followed by a feeling of weakness in the arms and legs. The arms show an improvement whereas the legs are getting progressively worse. Experiencing severe deterioration since suffering from influenza in 1918. Walking has been

very difficult since then. Abdominal reflexes are missing on the left-hand side; only the right-hand side reflex maintained and very weak. Cremasteric reflexes are missing. Patellar reflexes are weak on both sides. Foot clonus is present on both feet. Plantar reflex is normal on the left-hand side, Babinski on the right-hand side. On both legs a spastic muscular system is present, severe ataxia. Feeling of positioning is missing on all toe joints on both feet. Periosteal and tendon reflexes increased in both arms, severe intension tremor and ataxia. Chanting type speech, nystagmus when looking to the left. Wa. R. in the blood.

Case 10: A. B., 43 years old. Suffering headaches and visual disturbances 6 years ago. Later on, increasing weakness and unsteadiness in both legs.

Very extensive temporal pallor on both sides. Abdominal reflexes are missing completely on both sides. Patellar reflexes are normal on both sides. Foot clonus and Babinski are present on both sides. No feeling of positioning in all toe joints on the right-hand side, very reduced on the left-hand side. Suffering from severe spasms and ataxia in both legs. Extremely severe spasmodic walk. Periosteum and tendon reflexes increased in both arms, minor ataxia. Speech is slurred and has a tendency to laugh. Wa. R. in the blood and liquor. No protein and cell proliferation.

Case 11: F. E., 42 years old (clinically not quite sure, therefore presented more extensively). Suffered from pain in both shoulders and the right arm after a feverish cold in the field in 1915. Suffered from a feverish illness for a few days in the middle of 1916, which was followed by unsteadiness in the legs. As all skin and tendon reflexes were missing, tabes came to mind and the man was released from hospital. Early stage of papilledema in January 1920; corneal reflexes weakened on both sides, conjunctival reflex is missing on the left-hand side. Visual field for white and red near normal, for green concentric, somewhat concentrated (Dr. Kühn).

All skin and tendon reflexes are working, positive Romberg. Walk is unsteady. Feeling for touch, less for sting, decreased in both hands and forearms as well as decreased feeling of positioning in the finger and toe joints. No increase of the minor papilledema in the following time period and since the examination in July 1921 has been observed. In addition to the more prominent locations, already atrophies with very minor vascular involvement (as described by Marburg as characteristic for multiple sclerosis[1)]). Visual field normal for white, concentrically concentrated at 10^0 for green, internal insufficiency, crossed double images. All three abdominal reflexes on the right and left-hand side are present, cremasteric reflexes are normal, patellar reflex on the left-hand side somewhat weakened, increased on the right. Both hamstring reflexes are weak, Oppenheim on the right. Minor ataxia present in both arms and legs, no spasms. Romberg is present. Walk is unsteady. The pelvis is poorly fixated on the left. Feeling of touch and sting decreased on the left side of the face and the distal parts of the arms and legs. Feeling of positioning decreased in the right knee joint and all finger and toe joints. Speech is normal. However, he says that swallowing and speaking are difficult from time to time, which has happened twice per 14 days, making his teeth very sensitive and chewing impossible.

Here an improvement in the reflexes has been observed already using the pseudo-tabetic form of areflexia, some became spastic. (S. Oppenheim, textbook 1913, page 442) Wa.R. in the blood and liquor negative; no protein and cell proliferation.

[1]) German Journal of Neurology, edition 68, page 38.

Amongst the six operated cases were:

Case 1 and case 2[1]) hypertrophic nasal and throat mucous with increased secretion, hyperemias, venous congestion in the arches. Fissures with fibrinous coating. The tonsils were hypertrophic, fissured, with liquid-purulent detritus in the lacunae. – During extra-capsular curettage, many smaller and larger purulent foci were removed.

Case 3: Nose and throat mucous membranes partly hypertrophic, partly atrophic. In the left tonsil, in a perputial sac, which is slightly sticky externally, numerous caseous fetid grafts were found as well as in a pocket just above the plica transversa. Upper right showed individual grafts as well as in a further pocket of a transverse septum. Extra-capsular curettage produced substantial purgation of putrid content.

Case 4: Nose and throat mucous membranes mainly atrophic. Both tonsils are small, atrophic, with increased consistency. Detritus is present predominately on the left-hand side, to a lesser degree on the right-hand side. Grafts and fibrinous exudation in some obliterated tonsil crypts. Increased and thickened connective tissue septa between the lymphatic tisue present. During the extra-capsular tonsillectomy, both were found to be heavily permeated with pus and intra-tonsillar abscesses, partly with concrement formation.

Case 8: Atrophy of the lower, hypertrophy of the upper concha, developing into atrophic rhinitis: venous hyperaemia of the arcus and the velum. Caseous detritus is present in the upper pole in both tonsils. Tonsils moderately indurated, palatal arch thickened. Dilated lacunae openings. During tonsillectomy an extra-capsular abcess the size of a thimble was found on the right as well as a small intra-tonsillar abcess on the left-hand side together with numerous other sites of inflammation.

Case 9: Atrophic rhinitis, mucosa of the pharynx generally hyperaemic; severe hyperaemia of the mucous membranes in the palatal arches and the velum. Tonsils on both sides are fairly coarse and increased consistence. Severe Detritus is present on the left-hand side, a little less on the right-hand side.

[TEXT MISSING]

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1) The findings were identified by the consultants Drs. Beier, Grave- and Itzerot.

HIGIER

[TEXT MISSING]

… of the onset of speech, chewing and food digestion, respiration and phonation with oppression and respiratory crisis, with profuse salivation and rigid facial expression with intact mobility and muscular trophicity. At one point a dysarthritic boy stammered for days. A usually healthy elderly person suffered typical myasthenia of the bulbar muscles, leading to their exhaustion by the evening with mastication suspended after 6 – 8 movements. This could only be continued after several seconds of rest. However, gradual variations in muscle weakness and electric, myasthenic reactions were missing.

Apart from paresis of the depressor muscles, difficulty in moving the tongue and the velum, I repeatedly noticed trismus – never atrophy or fibrillar convulsions. The coexistence of amine and hyper-amine, muscle rigidity and the compulsion to laugh out was not a rare occurrence. A great rarity compared with that, however, is the preserved innervation of the oral muscles with isolated losses of mimic, phonation, articulation, mastication and deglutition. A characteristic feature was the difference between the willful and the automatic-reflexive motion of the bulbar muscles: as Wexberg already pointed out it was better preserved in the oral and pharyngeal muscles. I did not observe the spastic, explosive laughter of the pseudo sclerotics, but a rigid, cumbersome smile.

Severe polymorphs of the eyeballs were observed. Paresis, inertia and unequal pupils were proven to be present in the majority of cases with noticeable, severe restitution. Diplopia, convergence paralysis, nystagmus and ptosis belonged to the more volatile symptoms. With regards to diplopia the paretic muscle could not always be found. In some young cases of palsy random changes between myotic pupilloplegia with prompt reaction to light was observed.

From time to time I observed a typhoid-associated weakness of rolling the eyeballs up and down. Light inertia was often associated with inertial convergence. The accommodation palsy usually did not show signs of mydriasis. Rapid changes of eye disorders, often within the course of one day, changes of pupilloplegia with reduced reaction to light (Westphal) and unstable ani …

[----------TEXT MISSING------]

In order to guarantee an unaffected observation, both incipient cases remain untreated.

As well as various disorders affecting internal organs, Päβler [1]) also mentioned general disorders of the nervous system, in particular disorders of the peripheral and central nervous system, were caused by abscesses of the tonsils. I would briefly like to mention that I, myself, saw healing in some recurrent cases of neuralgia and gradual multiple neuritis once a tonsillectomy had been performed.

In our cases atrophic rhinitis, as well as the tonsils, have to be taken into consideration when looking for a gateway for the illness.

I found seven different views on the origin of atrophic rhinitis in laryngological literature by in particular Rundström[2]) and Wright[3]). However, it is always mentioned that none is satisfactory.

Operative findings suggest that there are primary purulent foci on the tonsils causing inflammatory changes in the surrounding sensory nerves. As in case 1 and 2 this initially leads to hypertrophy, mucosa with hyper-secretion, eventually leading to atrophy with a loss of secretion, finally leading to the growth of altered bacterial flora[4)] and maybe atrophy of the bone. This would be an analogy of the vaso-trophic condition of the skin following an illness of the nerves in the area.

According to experiments by Henke[5]), the bacteria, which penetrated the lymphatic vessels in the nasopharynx, then arrive at the tonsils where they are washed out by a continuously flowing lymph stream. This means of protection of the affected tonsils (also called muddy filters) will fail. If the body is weakened by an earlier injury, an infectious disease, an accident or post-partum, it may succumb to the penetrating pathogens. This leaves the body open to disorders of the kidneys, infections of the peripheral nerves, and more rarely to perivascular and vascular infections of the central nervous system. This may well be within the range of biological possibilities. In his works Friedrich Schulze has repeatedly pointed out the existence of particular pathogenic germs, which can remain in the central nervous system or anywhere in the body, occasionally spreading further. According to my findings one could assume the affected tonsils and nasopharyngeal mucous membranes to be such an area. Von Strümpele's objection that an endemic or even epidemic occurrence or transmission in siblings or spouses has not been observed would then be void. In his work on multiple sclerosis and brain tumors[3]) Marburg says that in case number 3 it was preceded by severe nasal suppuration. In most cases prior nose and throat infections were negated. One of my cases (case no 8) complained about a throat infection from a long time ago. Nobody else knew anything about it.

Therefore we have to assume these infections develop slowly and gradually.

On several occasions I observed minor ailments of other organs, 3 x low protein excretions, 2 x toxic heart and vascular disorders and on one occasion, a constantly recurring bowl disease – a clinical sign for a general infection.

A clinical diagnosis on whether abscesses in the tonsils are present was positive in the majority of cases, especially with aid of the suction method. In some cases experienced specialist physicians continuously maintained that the tonsils are healthy or atrophic, lying somewhat low, but are free – or that they are of normal size, not fused and just a little thick and shiny on the surface. Especially in these cases curettage of the inner and outer capsules found plenty of smaller abscesses, 2x retro-tonsillar abscesses with foul-smelling pus of the size of one

thimble, 1 to 2 teaspoon full. (Case 8 and two cases of multiple neuritis). In his works[1)] the laryngologist, Rudolf Steiner, points out that in more than half of all cases peritonsillar abscesses were found.

It can hardly be assumed that all these tonsillar and nasopharyngeal mucosa changes should be secondary findings as such abscesses have already been proven to be the source of chronic disease in other organs. Additionally, multiple sclerosis, as well as nephritis in the tonsils, neuritis or polyarthritis, reacts with a renewed, but quickly passing worsening of the symptoms locally, which lead to peculiar reactions of the central nervous system. Multiple sclerosis is certainly showing an increased sensitivity to all infections, but not to this degree and with the characteristics demonstrated after the operation in this case. In some cases other epicenters in the body's lymphatic system could be considered to be the origin of multiple sclerosis.

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[1]) German Mag. for diseases of the nervous system, ed. 38 & 65 page 8

[2]) Neurolog. Centralbl. 1918, no 12

[3]) German Mag. for diseases of the nervous system, ed. 68, page 33

21. Mr Walter Börnstein, Frankfurt am Main

On the seat of the cortical gustation center[2])

Despite the works of Magendie, Flourens, Ferrier and others, it has not been possible to determine the seat of the cortical gustation center. Even the latest extensive paper by von Henschen in 1918 leaves the question open. The common assumption of the gustation and olfaction centers being one or very close together is, due to anatomic and physiological reasons, improbable. It seems more probable for the gustation center to have its seat in the operculum as said by Bechterew. During a number of specific examinations (4 cases were particularly characteristic) a gustational disorder was found in lesion in the crossed half of the ??? (text illegible) in proximity of the operculum.

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[1]) Monatsschr. f. Ohrenheilkunde (Monthly Otology Magazin, Laryng. U. Rhin. 1918, pg. 337

[2]) Not presented due to time contraints