The Journal of the American Medical Association

Published under the Auspices of the Board of Trustees.

VOL. XXXVIII.

CHICAGO, ILLINOIS, JANUARY 4, 1902.

No. 1.

Original Articles.

THE SPECIFIC AND NON-SPECIFIC LESIONS OF THE BRAIN RESULTING FROM SYPHILIS AND THEIR INFLUENCE UPON DIAGNOSIS, PROGNOSIS AND TREATMENT.

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That syphilis is due to a specific poison that is capable of causing the development of luetic lesions in non-infected persons is an indisputed fact. Whether this micro-organism belongs to the vegetable kingdom and may be classed among the bacteria, or whether it may be found in the lower forms of the animal kingdom and may be classed with the plasmodia, or whether it may occupy a place between the bacteria and plasmodia must be determined by the scientific investigator. We are as ignorant of the true nature of the virus as was Fernelius, when nearly 400 years ago he formulated the seven laws of syphilis. Syphilis of the brain can not be acquired except the virus enter the blood or other juices of the body through a denuded mucous or cutaneous surface. From the external manifestations of syphilis that we are able to observe, we are justified in reasoning concerning the internal manifestations of the disease, the early developments of which we can not see. While the virus of syphilis reaches the different portions of the body through the medium of the blood and the other fluids of the body, yet it attacks the most vulnerable solid tissues with which it comes in contact. This is shown by the development of a chancre at the point at which it finds entrance into the body. The most vulnerable tissues of the body seem to be the skin, the exposed mucous surfaces, especially of the mouth and throat, the iris and the membranes and blood vessels of the central nervous system, those of the brain standing first. It is a well-known clinical fact that when the skin, mouth, nose and iris escape, or to express it differently, in the absence of the secondaries, the brain and probably other viscera are much more likely to be affected. This would indicate that the virus expends its force on the tissues which it first attacks, or probably the true condition might be better expressed by saying that when the virus of syphilis once invades a tissue that that tissue then loses much of its resisting power to the poison and is most likely to be the seat of subsequent manifestations of specific lesions.

Pathologic Conditions of the Intracranial Struc-

tures in Syphilis of the Brain.—The lesions of the brain that may directly or indirectly result from syphilis may be divided into three classes: 1, the specific or luetic lesions, those that are caused by the poison of syphilis and by nothing else; 2, those lesions of the brain that may have a specific or non-specific origin, and 3, the non-specific lesions of the brain that occur as indirect results of the syphilitic virus.

It is of the utmost practical importance when possible to distinguish between the specific and non-specific lesions affecting the intracranial structures. Unless such a differential diagnosis is made we are at a loss as to treatment and totally at sea in regard to prognosis. Only the specific lesions, those that occur as direct results of the poison of syphilis, are especially remediable to antisyphilitic treatment; while the numerous injuries caused by the presence of specific lesions or the degenerations resulting from toxemic or other blood states which may possibly result from syphilis, are in no way specific in character, and yield no more readily to antisyphilitic treatment than do lesions resulting from causes wholly unassociated with syphilis.

GUMMA.—The typical specific lesions of the brain is the gumma. The first thing observed, especially in the meninges, is a small spot of inflammation. This soon leads to a knot of granulation tissue, which has been variously described as gray, grayish-red, semi-translucent or gelatinous. Early the new-formed tissue is extremely cellular and later becomes fibro-cellular. This mass of ill-formed granulation tissue, while it may continue to increase in size, soon begins to undergo caseation and fibroid degeneration in one or many spots. Evidence of congestion and inflammation mark the first stage and hyperplasia with degeneration, the second. The formation of the gumma is the direct result of inflammation, which undoubtedly has been lighted up by the poison of syphilis. It will be seen that it differs from the ordinary products of inflammation in being less highly organized and in having a greater tendency to degeneration. When the inflammation set up by the virus of syphilis is acute and violent, the inflammatory products at first vary but little from those of an ordinary or non-specific inflammation. It takes time for the gumma to assume its characteristic form. One of the greatest characteristics of specific intracranial inflammation, especially of the meninges, is its tendency to hyperplasia. The gumma varies in size from a line in diameter to several inches. It may be yellowish, reddish or of a grayish-white appearance, yet it usually has, when of recent formation, the feel and consistence of glue. No matter how large the gumma may become, nor how long it may have existed, it never becomes highly organized.

The degeneration may take place in one or two ways; either by caseation and fibroid degeneration, or by fibroid degeneration alone. Formation of masses of cicatricial tissues occurs as the result of fibroid degeneration. Gummatus deposits may be single, but they are frequently multiple. The brain substance adjacent to the gumma rarely ever remains intact, as the inflammation spreads to this. Gummata are most commonly found at the base of the brain, especially early, and the region of the interpeduncular space is a favorite site for the deposition of the products of specific inflammation. The sixth and branches of the third cranial nerve are often affected in the early stage. Gummata are often found on the cortex of the brain, invading the sulci, especially of the so-called motor region. They occasionally are found in the pons, rarely in the substance of the cerebrum and least frequently in the substance of the cerebellum. Gummatus inflammation of the membranes, both of the pia and dura is frequent, in fact, this is the most common form of intracranial specific inflammation. In inflammation of the membranes the tendency to hyperplasia is very great and the membranes, especially the pia, become so thick in the neighborhood of the optic chiasma as to present the appearance of a tumor. Inflammation of the dura is usually diffuse and the tissue form undergoes the fibroid change. It is thought to be most common in inherited syphilis. In the pia the inflammation is more commonly local, and the inflammatory products may undergo caseous, or fibroid change.¹ Finally, the walls of the arteries are frequently the seat of inflammation and of gummatus deposits. The sheaths of the cranial nerves undoubtedly may become specifically inflamed, and the nerves themselves damaged, either by extension of the inflammation, by pressure from gummata, or by constriction caused by the cicatricial tissue. That the nerve fibers and the brain substance have a greater immunity from the poison of syphilis primarily than do the meninges, the nerve sheaths and the blood vessel walls seems to be almost, if not, universally conceded, but that the brain and nerve substances are never the primary seat of specific inflammation, is doubtful, to say the least.

Some of the Specific Lesions and the Non-specific Ones that Result from Syphilis.—We must remember that while a specific poison is the cause of gummata, these by their presence, lessening intracranial space, by their irritating effects, encroaching upon and injuring adjacent structures, produce intracranial lesions that are in no way specific in character. Before considering the walls of the blood vessels, as affected by syphilis, let us try to study some of the effects of specific inflammation of the meninges. In an attempt at a differential diagnosis between the direct and indirect effects of syphilis, we must keep widely separated the idea of specificity and non-specificity.

I can not reiterate too often that this distinction is of the greatest practical importance in the prognosis and treatment of every case of intracranial syphilis. For the one class of lesions early in the disease, we have approximately specific medicaments; for the other there is no known medication of especial value. Recent gummata at the base of the brain exert pressure on the solid structures and interfere with their functions, the symptoms of the disturbed function depending upon the size of the deposits and their situation. The cranial nerves are compressed and irritated, and the membranes

are inflamed. If appropriate treatment is instituted in the early stage, before caseous and fibroid degeneration takes place, and before injuries to the structures compressed become well advanced, the whole morbid process may be removed and a cure, so far as the local brain trouble is concerned, is complete. But, unfortunately, vigorous treatment is not always instituted early; it does not take long for compression of nervous tissue to impair permanently, although it may not destroy, the function. Caseous and fibroid degeneration sets in early. Although the gummatus substance may be completely absorbed, the fibroid and caseous degeneration leads to the formation of cicatrices and bands of contracting cicatricial tissue. These are non-specific lesions, are not specially influenced by specific medications, and remain to compress and throttle the cranial nerves and keep up irritation to a greater or less extent in the membranes and in the brain substance. If, perchance, they remain latent, they mark the situation for subsequent specific inflammation on the application of any of the various exciting causes. It is to these non-specific lesions at the base of the brain, which have resulted from specific inflammation, that many cases of impairment of vision, blindness, ocular paralysis, and chronic basilar headaches are due. These, of course, fail to respond to antisypilitic agents. Many cases of optic nerve atrophy and of permanent paralysis of one or more ocular muscles may be thus explained, both in children and adults; for, it must be remembered that gummata occur as the result of hereditary syphilis as well as from that which has been acquired.

Gummata on the convex surface of the brain are most likely to occur in and around the motor convolutions. The effects, both from the specific and non-specific lesions, on these structures, the cortex and membranes, are the same as when the inflammation takes place at the base, but the symptoms are different. The same remarks apply to the effects of syphilitic growths within the brain substance. It may be added that intracerebral syphilitic growths are rare and probably arise from some portion of the pia. The characteristic form of specific intracranial inflammation is syphilitic meningitis. It is doubtful whether this form of meningitis, in the true sense of the word, is ever acute. In other words, an apparently acute attack of syphilitic meningitis is secondary to a pre-existing local and chronic specific inflammation of the membranes. If this is true, and I have seen no reason to doubt it, a syphilitic meningitis is always subacute or chronic; although acute outbreaks are not uncommon. Great thickening of the cerebral membranes sometimes takes place from specific inflammation. These thickened areas or patches in the membranes denote chronicity, and in consequence, while they are due to syphilitic inflammation, they are degenerative in character, composed of bands of firm fibrous tissue, the results of caseous and fibroid degeneration, and are not purely syphilitic in character. At the base of the brain these bands of fibrous degeneration are most commonly found in the neighborhood of the optic chiasma and in the interpeduncular space. Syphilitic meningitis, involving the convexity of the brain, is not infrequent, especially affecting the pia over the anterior and parietal lobes in the vicinity of the longitudinal fissure. According to my experience, when the dura is inflamed in this region the pia rarely altogether escapes; especially have I observed this when the cranial bones of the vault have been inflamed. The pia,

1. Gowers: *Littsomian Lectures on Syphilis and the Nervous System*, 1892.

on the other hand, may be inflamed and the dura remain free. In syphilitic inflammation of the pia the cortex is involved in the inflammatory and degenerative processes. We have a meningo-encephalitis which is partly due to meningitis and partly to degeneration of the arteries of the cortex. Here, again, while the specific inflammation has been mild and the specific exudate comparatively slight, the non-specific lesions are extensive, very destructive, will not respond to antisiphilic medication, and hence are permanent in their effects.

There is one other change in the membranes to which I wish to direct attention. This is chronic thickening of the dura in the posterior cerebral fossa. It is commonly found in childhood as the result of inherited syphilis. I have only seen one case of this affection. It occurred in a child about 3 years of age, the offspring of infected parents. It had been sick for several weeks before it was admitted into the Arapahoe County Hospital. The head was extremely retracted; the body became greatly emaciated; but the patient lived, after it came under my care, one year. At the autopsy the dura of the posterior fossa, especially that portion surrounding the pons and medulla, was thickened, and the seat of old fibrous deposits. Some of these cases are said to have recovered. It is just possible that some cases of reported recovery from tubercular meningitis may have been cases of syphilitic meningitis.

Blood Vessels in Syphilis of the Brain.—We now pass on to syphilitic inflammation and degeneration of the blood vessels of the brain. Syphilitic endarteritis and peri-arteritis have been observed and minutely described by nearly every writer on syphilis of the brain, but so little has been said about syphilis of the veins that I shall quote from a case cited by Mickle.²

CASE 1.—Female, aged 45, died in an epileptiform attack due to hemorrhage at the brain base, from rupture of a syphilitic vein. There were clear signs of early general paralysis, and tabetiform ones due to diffuse embryonic meningo-myelitis. Syphilitic lesions affected the large vessels of the brain and cord. A diffuse vascular lesion affected the cerebral gray cortex, and in a less degree the subjacent white substance.

The large arteries of the brain present slight patches of ordinary secondary obliterative endarteritis; but the chief lesion is an infiltration of the embryonic cells in the adventitia, forming an intense peri-arteritis, diffuse, but in places nodular—the first stage of a gummatous process. In the veins the muscular connective tunic is lesed; in them the infiltration, although usually diffuse, takes here and there a nodular character, even more clearly, and so as to form miliary gummata when “caseification” sets in. The venous lesion exceeds the arterial at the brain base, where it has led to hemorrhage, and at the surface of the cord, and is largely independent of the meningeal and arterial lesion. The endovena does not show any proliferation; when it is affected it is so through invasion by the gummatous process. Hence, the ulterior tendency of the luetic arterial lesion is to narrowing, obliteration, and their results; in the veins, to hemorrhage.

Personally, I have not met with a case of venous hemorrhage due to syphilitic degeneration of the vessel.

ARTERIES: Gowers, in speaking of arterial degeneration, states that “except in the arteries of the brain and in the aorta, syphilitic disease is scarcely known.”³ In this he is mistaken, as syphilitic degeneration of the spinal arteries is not uncommon.

The walls of the medium, and sometimes of the larger, arteries of the brain are affected by a syphilitic inflammation and gummatous deposit, involving especially the

inner coats—the intima and endothelial lining—of the vessels. This process is known as endarteritis, and on account of the tendency to obliterate the caliber of the affected vessel is sometimes spoken of as arteritis obliterans. In some cases, before the caliber of the vessel is closed, a small branch given off by the vessel is blocked and the blood supply to a very small area of the brain may be cut off. If a small artery, especially of the cortex, is thus closed, the collateral circulation may be sufficient to prevent necrotic softening, but it may not be sufficient to maintain the normal condition of the part, and this may explain the spots of induration found in the cortex of the brain, especially in syphilitic children. When the caliber of a medium or large vessel is occluded, necrotic softening in the area of distribution of the affected artery must take place, as the collateral circulation of the brain is so meagre that Nature is unable to provide against such violence. It is important to remember that the final closure of the vessel is due not to gummatous deposits, but to a blood clot formed at the narrowed portion of the vessel. Thus, while the greater portion of the vessel's caliber has been occluded by a specific lesion, the closure is finally completed by non-specific material that is not specially influenced by anti-syphilitic treatment. We have, then, in all cases of syphilitic occlusion of a vessel two non-specific lesions; one, the blood clot that completes the occlusion of the vessel, and the other the necrotic softening in the area of the brain to which the occluded vessel is distributed. These facts should be borne in mind in the prognosis and treatment of all cases of syphilitic thrombosis.

Gowers states: “A second effect is the production of aneurysm of the larger arteries—those that are the special seats of the disease.”³ The explanation given for the formation of aneurysm is inflammation and degeneration of the vessel wall by which extensible fibroid tissue is substituted for the elastic elements.

Mills, in his volume on “Disease of the Brain and the Cranial Nerves,” says peri-arteritis attacks the smaller arteries and may give rise to miliary aneurysm and hemorrhage. In a case recently under my observation peri-arteritis involving the nutrient vessels of the pons resulted in local softening and death. Peri-arteritis of the nutrient vessels of the brain is not rare, but hemorrhage into the brain substance from specific inflammation of the vessel is infrequent.

I shall not tarry to discuss cases of supposed specific inflammation of the substance of the brain. Charcot and Gombault reported a case of chronic disseminated inflammation, with foci in the cerebrum, pons and optic nerves, and the tissue in many of the foci had undergone caseation. Barlow has observed foci of sclerosis in the cortex of the brain, without arterial degeneration in syphilitic children.

Gowers observes: “The spinal nerves seem to escape the influence of syphilis, and the cranial nerves suffer chiefly at the base of the brain, where several are involved in a focus of inflammation. To this some of the nerves to the eyeball seem to offer an exception; they may suffer alone. The facial nerve presents a remarkable freedom. The nerves that arise from the side of the medulla are often involved, especially the hypoglossal and spinal accessory causing palsy of the tongue, palate and vocal cord on one side. These palsies are seldom due to any other cause, so that their occurrence should suggest syphilis at once to your mind.”⁴

2. W. Julius Mickle: On Syphilis of the Nervous System, Brain, 1895, part I, pp. 115, 116.

3. Gowers: Syphilis and the Nervous System, p. 1.

4. Gowers: Littsomian Lectures, Syphilis and the Nervous System, 1892, p. 43.

Many of the nerve degenerations, both nuclear and system, are caused by the poison of syphilis. Some of these are evidently due to a primary degeneration of the nerve elements, and others may result from inflammation of the interstitial tissue. Whatever may be the method by which the degeneration is brought about when it has once occurred, antisypnilitic treatment can do no good.

It should be borne in mind that multiple lesions in syphilis of the brain are the rule. It is not uncommon to find most of the larger arteries at the base of the brain affected with gummatous deposits. In cases of syphilitic tumors of the brain it is the exception to find the blood vessels healthy. On the other hand, the walls of the arteries are the seat of numerous gummatous deposits, while the other structures of the brain are free from the appearance of syphilitic disease. We may conclude from this that the blood vessel walls are the most vulnerable intracranial tissue to the virus of syphilis.

TREATMENT OF NEURASTHENIA.*

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I suppose we all recognize the fact that there never can be any ready-made, hand-me-down treatment for neurasthenia. When we consider neurasthenia from an etiological standpoint, and then attempt from this consideration to evolve a rational treatment that will not only cure the patient but at the same time make him or her comfortable enough to live until the cure is effected, we certainly have ample food to satisfy our appetite for conundrums.

It is all very well to say that the principal causes of neurasthenia are toxin, malnutrition, fatigue, or emotions, and that the treatment depends upon the cause. This of course is so, but it is just as true that we may have a patient that is long on causes and has a neurasthenia that is caused by all of these conditions combined, and this is not all, as the nervous system governs and controls all the organs of the body, and as these organs take advantage of the weakness and irritability of their masters to act in all sorts of irregular and uncomfortable ways, it is no easy task to tell whether their action depends upon the neurasthenia, or the neurasthenia depends upon them. When we consider this jumbled up mess of cause and effect and effect and cause, it is not surprising that we sit down in despair and wonder what to do first. I find it best to first carefully examine the patient, being sure to ascertain the condition of each and every organ as well as possible; also inquire into the habits of the patient. This examination may or may not give us much information, but if it does not, it gives the patient confidence in us, or at least helps to do so, and this I consider a very important part of the treatment.

CONFIDENCE OF THE PHYSICIAN.

All of us, of any considerable experience, have treated many cases of neurasthenia successfully and probably as many more without success, and as we carefully review our work we are unable to see why one patient should have recovered, and another failed to do so. It is often humiliating to see a patient whom we have failed to benefit, gradually and continuously improve, under possibly the same treatment, in the hands of another physician. We may break the force of the fall by de-

claring that there was nothing the matter, or they would have improved just the same in our hands. I do not believe that such is the case. The different results are due simply to the fact that we did not have the confidence of the patient, and our successor did. I have become so certain that confidence in my ability to do the very best that can be done for them is such a necessary factor in the bringing about of a cure in these cases, that I rarely take charge of a case of neurasthenia where there seems to be a lack of faith in me; it is true that it sometimes takes more tact and management than I possess, to successfully pass them over, and I am forced to treat them, but I do so with my mind fully prepared for their departure.

Do not think that I believe the only thing necessary to cure these people is faith; for, such is not the case; but a neurasthenic nervous system is lame, and in such a condition that it must have someone as a nervous crutch to help it along from time to time, until once more strong enough to go alone, and as a rule the attending physician must be the support. This is plainly demonstrated by the fact that, after seeing the physician and having a chance to relate their various bad feelings, and being reassured that these are nothing to be alarmed at, etc., for considerable time the patient gets along nicely. But after so long a time we have to go over the same ground again; they require the same reassuring and once more are strengthened and feel better for a longer or shorter period varying according to the condition of their nervous system or the external conditions brought to bear upon it. These things often seem childish to us, who are strong and well, but they are important to the patient, and it is for this reason that our visits and consultations with these people should not be hurried nor too far apart.

DIET.

Proper feeding is an important, and often neglected part of the treatment. I have seen delicate women who were what we might call natural neurasthenics—that unfortunate class who are launched into life with a nervous system so frail that it can scarcely supply sufficient nerve force to meet the ordinary demands of living—I have seen these delicate women systematically starved for years, while they underwent all kinds of scientific treatment: drugs galore, electricity, massage, and even starvation by rule at well-known sanitariums. After having endured these things, without benefit, while in the hands of men of reputation and skill, they naturally wandered outside of the regular routine and took in the novelties, as osteopathy, etc. After all this I have seen them improve as if by magic when put on a liberal diet of common-sense food, accompanied by such medication as assists nature in digesting and assimilating the food taken. It is not feeding these people properly to place them on a diet composed of these high-priced concentrated foods, that one teaspoonful equals in nourishing power the flesh of two oxen and ten sacks of flour; their stomachs will not stand it, more padding is required. Neither is chopped feed, no matter how well roasted, nor how well and attractively it may be packed, nor how skilfully advertised, nor how highly endorsed by medical authority. We are too apt to think that, because the patient complains of unpleasant and disagreeable sensations in the stomach after having taken food, that the stomach is being worked beyond its capacity, and that the amount of food must be diminished. It has often been a surprise to me to see how one of these starved and pampered

* Read at the Fifty-second Annual Meeting of the American Medical Association, in the Section on Nervous and Mental Diseases, and approved for publication by the Executive Committee: Drs. Frederick Peterson, Hugh T. Patrick and H. A. Tomlinson.