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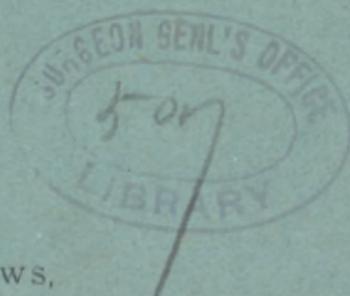
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*THE PATHOLOGY OF LOCOMOTOR ATAXY.*¹

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LOCOMOTOR ataxy has found its place in all of our modern text-books on nervous diseases amongst the lesions of the spinal cord. As our knowledge of the pathologic changes in tabes became more exact we found that the spinal cord was not by any means the only part of the nervous system affected. Sclerotic lesions of the posterior spinal roots of the peripheral, spinal, and cerebral nerves, occasionally, also, alterations in the nuclei of cerebral nerves, were discovered. The substance of the brain, it would appear from these reports, is not, as a rule, diseased.

An attempt to explain the genesis of the symptoms in almost any case of locomotor ataxy will soon convince us that comparatively few of them can be attributed to the spinal cord. We know that such disturbances of the functions of the bladder, rectum, and genital organs, as are commonly found in tabes, can be caused by spinal lesions; they do, however, also occur in diseases of cerebral origin.

¹ Read before the Chicago Medical Society, February 5, 1894.



Westphal's symptom—the disappearance of the knee-jerk—seems to be undoubtedly a spinal one. The discoverer of this most important sign was fortunate enough to be able to localize its origin very exactly in the spinal cord, his researches proving that degeneration of a region called by him the “Wurzeleintrittszone,” by French authors “bandelettes externes,” was associated with loss of the knee-jerk.

As to the localization of the most important symptom of this disease, the one from which it was named, we know nothing of a positive nature whatsoever. Attempts have been made to explain it by assuming it to be due to sensory disturbances in the joints. All of them have, in my opinion, been decided failures. If there were a connection between these two symptoms, then every case with marked ataxy ought also to present marked sensory disturbances, and any case with slight sensory disturbances only slight ataxy. This is not the case. I have myself seen a case in Heidelberg with very marked incoördination of the movements of the lower extremities. The man has repeatedly been subjected to a very exact examination within the last few years, which has never revealed any sensory disturbances which could have caused the ataxy. Other cases are on record presenting complete anesthesia and not a trace of incoördination.

Jendrassik has called attention to the fact that, leaving locomotor ataxy out of consideration, there is no case on record which proves that ataxy can be caused by a spinal lesion. Some few observations, it is true, are quoted as proofs of this statement,

but in some of them the brain was never examined ; in others, when this was done, cerebral changes were found as well as spinal lesions.

Never, to my knowledge, has anybody succeeded in producing incoördination of movement by experimentally injuring the spinal cord. There are quite a number of cases on record in which degeneration of peripheral nerves has been found in ataxic subjects. Here, too, it seems that the brain was never examined.

Pitres has reported a case of pseudo-tabes with ataxy, in which spinal cord, spinal roots, and spinal nerves were found to be intact. Does not this observation clearly demonstrate that we must search for the lesion higher up in the nervous system, in the medulla oblongata, pons, or brain.

Glötz's researches have induced him to assume the existence of a center for coördination above the spinal cord. He describes the alterations of motility in his dogs after removal of portions of the brain by comparing the movements to the "Hahn-entritt," the step of a cock, a name which is frequently used in Germany in designating the walk of the ataxic. Wernicke and Kahler have seen cerebral lesions followed by ataxic movements exactly like those in tabes, and within the last year or so a similar association was described in a tumor of the frontal lobe—if I am not mistaken, by Bruns. I have seen two cases of this nature, one some years ago, without, however, being able to give the details to-day. A second was kindly referred to me for examination by my friend, Dr. Carl Beck, only a short time ago. The patient, J. B., is a waiter, fifty years old.

His parents are alive, his mother having had a stroke of apoplexy when seventy years old; the father is said to be healthy. B. has partaken of stimulants somewhat freely. In 1870 or 1871 he had a chancre, followed by exanthemata. He is married, but has no children. He has been ill for the last two years. At first the toes of the right foot became cold and numb. He was then delirious for about two weeks, and it is impossible to ascertain whether or not he had delirium tremens. At the end of this time he attempted to get up at night, but fell, as both of his legs were paralyzed. The arms were not weakened; his sphincters acted perfectly well; he was not unconscious; there was no anesthesia. After five or six days he was so far improved that he could get around with the assistance of two canes. The left limb soon regained its normal power; the right one, however, caused him a good deal of inconvenience, as he had intense pain in it whenever he tried to stand or walk, but it too did not remain paretic, nor did it become rigid. Seven or eight months after this there was a syphilitic ulcer on the right shoulder. Pain began to make its appearance in the right arm and the right upper portion of the trunk, being intensified by every movement. Then the speech became impaired; there were paresthesiæ in the right arm; and movements became clumsy. In the right side of the head he always has a painful sensation of heat. One and a half years ago he had double vision for about four months, which disappeared spontaneously. He never had headaches, and did not vomit. The memory is not impaired,

nor have the functions of the higher senses suffered.

The right pupil was larger than the left, and the reaction to light was lessened on both sides. Marked tremor of the tongue and facial muscles existed. The speech is thick. There is paresis of the right arm, but no atrophy. The deep reflexes are less marked on the right than on the left side. Pronounced ataxy of the right arm exists. Sensibility is normal. In the legs there was also ataxy on the right side, and the knee-jerk was lessened on both sides. When the patient closes his eyes and places his feet close together, he sways slightly. The abdominal reflexes are absent. The most scrutinizing examination reveals but one additional symptom, but this one alone enables us to reach a diagnosis. There is a choked disc on the left side. Hence this is a case of tumor of the brain, with marked ataxy of the right extremities.

One more point deserves to be mentioned in this connection. Although few, there are some cases of locomotor ataxy in which the onset of the incoordination is quite acute. I have been fortunate enough to see two cases of this kind. It will hardly do to attribute a symptom that appears quite suddenly to the chronic, very slowly progressing lesions, such as we find in the spinal cord. Of late, attempts have been made to improve the condition of the ataxic by systematically practising movements, and they have been very successful. Can we learn with our spinal cord? Can practice decrease the number of degenerated fibers in the posterior columns?

A part of the sensory disturbances in locomotor

ataxy may readily be explained by lesions of the posterior columns; others, however, it appears to me, are due, to a change not in the conducting but in the perceiving organs. In this connection I would mention the symptoms of allocheiria, polyesthesia, and after-pain, all of which are to me intelligible only if I assume that the function of apperception and not that of mere conduction is impaired.

The symptoms just enumerated have very probably their origin in some part of the nervous system other than the spinal cord. There is another series, no less numerous than this, which is surely due to extra-spinal lesions. Amongst these, ocular disturbances are both the most important and the most frequent, very often preceding by years every other sign of illness. I refer to the atrophy of the optic nerve, to ophthalmoplegia interna and externa. Eulenburg has compiled statistics on the frequency of these symptoms, comprising sixty-four cases of locomotor ataxy. In these he found strabismus divergens 19 times, 4 times associated with paralytic ptosis; strabismus convergens, 6 times; mydriasis, 6 times; myosis, 28 times; amblyopia, 31 times.

Topinard has examined 102 cases of tabes and found amblyopia in fifty-one, or exactly 50 per cent. These statistics ought to be considered sufficient proof of the fact that the ocular disturbances are more than chance complications, that they are very important and constant symptoms, as constant almost as any found in tabes. I have mentioned the impaired reaction and increased or decreased size

of the pupils amongst the extra-spinal, or rather, cerebral symptoms, as I am convinced that this is the correct localization for them. Although we know exactly where to search for the so-called cilio-spinal center in the spinal cord, nobody has, to my knowledge, ever found any lesion of this portion in a case of locomotor ataxy. Bramwell is certainly fully justified in attributing the loss of pupillary reaction to light and the decrease in size of the pupil to cerebral changes.

There are quite a number of other symptoms of clearly cerebral origin not infrequently found in ataxy, such as atrophy of cerebral, auditory, olfactory, and other nerves; paralysis of accommodation, hemiatrophy of the tongue, paralysis of the sensory or motor portions of the fifth nerve, lightning-pains in the head, attacks of vertigo, transient apoplectiform seizures, transient hemiplegia, convulsions in cases which never develop general paralysis (Kahler), various forms of insanity, besides the exceedingly frequent combination with paralytic dementia, drowsiness, headache, hyperemia cerebri, and increased irritability. Steinthal has noticed, and Erb confirms his observations, that patients suffering from locomotor ataxy are very frequently remarkably gay and satisfied with their fate. Athetosis has been observed by Cruveilhier, Hammond, Leyden, Laquer, and others.

If cerebral changes are as common in locomotor ataxy as the frequency of cerebral symptoms would seem to make probable, why are they so rarely described? Whoever has attempted to find microscopic lesions in a brain without knowing just where to look

for them will not hesitate to answer this question. It is an undertaking that requires more patience and more time than most observers wish to give to it. The difficult and tedious task has, however, been undertaken in a few cases within the last few years. And the results are just what we must expect if we consider the symptoms. In every case examined, diffuse changes in the cerebrum have been discovered. Jendrassik was the first to undertake these researches. He examined the brain of two individuals who had suffered from locomotor ataxy, but had not presented any symptoms of general paralysis, and in both cases he found cerebral lesions. Somewhat later I had occasion to control his results in another case in which the inspection at the time of the post-mortem examination did not indicate that the brain was diseased. Unfortunately this case was not a pure one, as the microscope revealed the existence of diffuse syphilitic lesions in the membranes of the brain and spinal cord. This was the first time that meningitis syphilitica had been found associated with the typical tabetic degeneration in the posterior columns. Since then a number of similar cases have been published. Kraus has recently communicated the result of his most diligent and exact researches on the pathology of locomotor ataxy. Unfortunately, like most of his predecessors, he, too, has not taken the trouble to make a microscopic examination of the brain; but even so, his work is of the greatest importance to us. He has examined the brain macroscopically thirteen times and has found changes in the brain-substance proper, the cerebral nerves, or the mem-

brane with the naked eye, thirteen times. The lesions discovered were :

	TIMES.
Atrophy of the optic nerve	2
Atrophy of the olfactory nerve	1
Atrophy of the brain	4
(Perhaps also present in a fifth case.)	
Pachymeningitis	4
Leptomeningitis	6
Edema of the brain	4
Hydrocephalus internus	2
Multiple encephalomalacia	1

A good deal of work still remains to be done, but this much I think can be said to-day : That the brain is diseased in the majority, probably in all cases, of locomotor ataxy ; that quite a number of the most constant symptoms of this disease are very probably, and some of them surely, not of spinal, but of cerebral origin ; and that locomotor ataxy is certainly not merely a spinal disease, but one rather of the entire central and peripheral nervous system.

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