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EYE SYMPTOMS IN TABES DORSALIS.*

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Mr. President and Gentlemen,—Knickerbocker's "History of New York" begins with an analysis of the various theories on the subject of the creation of the world. The author justifies the relevancy of the subject-matter on the ground that if the world had never been created there were grave reasons for supposing that New York city would never have had an existence. If with the title "Eye Symptoms in Tabes" I commence with a description of the nervous system, you may suppose that I am about to use a similar argument in justification, but it is not so.

The right understanding of the ocular troubles of tabes is so dependent upon a clear conception of the pathological process taking place in the peripheral and central nervous systems that I plead for no indulgence for spending a few minutes in running over the anatomy of a portion of the spinal column and its relation with the nuclei of the cerebral nerves.

This is all the more important, inasmuch as recent researches (pre-eminently those of Dr. Mott) have brought about a considerable modification in the views held as to the pathology of tabes. Charcot pointed out as a remarkable anomaly that, whereas the spinal disease was a centrifugal degeneration, the affection of the optic nerve was a centripetal one. It would appear that there is no such anomaly; the spinal disease itself is centripetal.

The modern method of describing the nervous system is, as you are aware, by means of systems of neurons. The nerve-cell is the neuron body, its axis cylinder process the axon or neuraxon. This axon may terminate in free arborization, or become continuous with a medullated nerve fibre. Certain other protoplasmic extensions of the cell terminate in finely-branched, plume-like twigs; these are dendrons. The neurons of the cerebrum and the cerebellum are spoken of as upper neurons, those of the spinal cord as lower neurons. Tabes dorsalis is a "primary, progressive degeneration of the first afferent (sensory) projection systems of neurons, by which peripheral sensations are cut off from the various parts of the central nervous system, the commonest and most obvious

* A paper read before the Windsor and District Medical Society.

anatomical change being degeneration of the posterior spinal roots and the posterior columns of the spinal cord."* If this is so, we have to inquire how it is that we get cerebral nerves affected.

The cells which degenerate in this disease are those of the ganglia of the posterior roots of the spinal nerves. These cells have T or Y-shaped processes, the lower limbs of which descend the nerve to terminate in sensory end-organs, while the upper limbs enter the column of Burdach, then pass into the column of Goll, and ascend the cord to terminate in the bulb in the region of the gracile nucleus. It is reasonable to assume that the part furthest from the trophic centre shows the effects of diminution of vitality the soonest; hence the beginnings of the degeneration take place in the terminal twigs. Suppose, for example, the ends of the nerve in the muscle spindles, which are considered to be the muscle sense end-organs, begin to lose vitality, a small stimulus is insufficient to start an impulse upwards to the brain; hence delicate movements begin to be difficult or impossible—we have commencing ataxia. The sclerosis of the cord, which has been thought hitherto to be the primary lesion, is now looked upon as a secondary process due to the hyperplasia of the neuroglia elements, most probably the result of the irritation of the products of degeneration.

Suppose, now, that the degenerative process commences in the upper limb of the axon: an irritation is set up in the region of the nucleus gracilis of the medulla, and, through what channels we do not know, this irritation finally affects especially the nuclei of the second, third, fourth, and sixth nerves, though other cerebral nerves are sometimes affected. One of the cases to which I shall refer presently shows an affection of the first. For the last two years he has been unable to taste (*i.e.*, smell) his food properly. This is rare. In all Duchenne's cases he only had one in which the olfactory nerve was affected. The eighth, ninth, and twelfth are more often included in the degenerative process.

I will consider the various eye symptoms as far as possible in the order of their advent. Perhaps neuralgic pains in the eyes—at the back of the eyes—are amongst the earliest symptoms of which patients complain, in the same way that the lightning pains are frequently the precursors for years of ataxia. But in general, I think the contracted and immobile pupil may be considered the first symptom that we can seize hold of, so to speak, and say: "Here is a basis of suspicion that we have to do with a case of tabes."

What is taking place when the pupil contracts normally under

* Allbutt's "System of Medicine," vol. vii.

the influence of light? An impulse is sent along certain fibres of the optic nerve, and this is reflected down certain fibres of the third nerve, which terminate in the short ciliary nerves, and the sphincter muscle of the iris is put into a state of contraction. Dilatation is accomplished by the relaxation of this reflex action when the source of the stimulus, light, is withdrawn.

The action of the third may, however, be inhibited by stimulating the sympathetic in the neck. By *cutting* the sympathetic, a certain amount of contraction results, showing there is a tonic balance ordinarily in action. Now, what is the condition in the contracted immobile pupil? Some stimulus (equivalent to the stimulus of light) is causing the third nerve to put the sphincter iridis into a state of tonic contraction. Remember the extreme delicacy of the light reflex. May we not suppose that commencing degeneration of the termination of the upper limbs of the spinal ganglion neuraxons is setting up through some intermediate fibres a slight irritation in the region of the middle cells of the nucleus of the third, starting them working in the only way they can work, namely, to contract the sphincter iridis? This irritation is going on in the dark, so that when the light is removed there is no dilatation of the pupil as in the normal eye. Later on in the disease we no longer get irritation here, but atrophy. Consequently there is paralysis of the sphincter iridis—mydriasis, not myosis. An interference with the sympathetic, associated with anæsthesia of the light-reflex fibres of the optic nerve, probably plays some part in the phenomenon, but this would not explain the pinhole pupils, though it might those cases of moderate contraction, with immobility, which are very common. No doubt the process is often complex, but even an incomplete conception of a morbid process may be of use in helping us to form a mental image of what is taking place. The books have much to say about the loss of the light reflex, but I wish to lay stress upon the loss of the darkness dilatation. I have not seen it brought out, but a fixed contracted pupil carries with it a very important subjective symptom, namely, an increased difficulty in seeing in a bad light. Every photographer knows what an enormous diminution in the brightness of the image on the focussing-screen the introduction of a stop produces. So in tabes, the small pupil shuts off a large amount of the light falling into the eye, and sometimes the deficient vision at night-time sends the patient for advice. Take this case: J. B., forty-five, a coachman, applied at the Royal Westminster Ophthalmic Hospital in September, 1898, complaining of failing sight at night. His vision was $\frac{2}{3}$ in each eye. Mr. Work Dodd, who

was the surgeon in charge, noticed his pupils, tested his knee-jerks, and found they were absent. It transpired he had had some weakness and fugitive pains, but, with these exceptions, the difficulty in driving at night-time was his only subjective symptom.

I have notes of a case of a country clergyman who came to London about his sight, his trouble being that when he came out of church after the evening service he could not see sufficiently well to reach his home without assistance. It was found that he was suffering from tabes. Of course, *ataxic* patients have increased difficulty in walking in the dark, because their deficient muscle-sense is not helped out by the added information accorded by the eye. But even in their case *part* of the difficulty is due to the defective illumination of the retina.

To test for myself the disability under which a tabetic suffers, I upon one occasion instilled eserine into my right eye, and went for a country walk in the evening. The experiment was very instructive. I was very glad the "control" eye was there to help me. The difficulty is not so great in well-lighted streets. It was a *country* clergyman who felt the night-blindness.

There is a second result that must optically follow a contracted pupil, viz., improvement of vision in an ametropic eye. Objects are focussed now that could not be focussed previously. I am on the look-out, gentlemen, for the tabetic high myope who will volunteer the remark that he has noticed a decided improvement in his distant vision lately. I think I shall meet him.

The muscle of accommodation, the ciliary muscle, is supplied by fibres coming from the front portion of the nucleus of the third. I am not aware that interference with these fibres is often seen, but I think the following case is worthy of mention. A young woman of thirty or thereabouts applied some years ago at the Westminster Ophthalmic Hospital for failing sight for near objects. Six or seven years later she returned with well-marked tabetic symptoms. It may have been a coincidence—many are the causes for the loss of accommodation in a young woman—but it is not at all improbable that it was an early effect of the disease that preceded by some years the onset of more characteristic symptoms.

After the pupillary symptoms come, in chronological order, various oculo-motor paralyses, generally one-sided, and at this state often fugitive. The third and sixth nerves are affected with about equal frequency. A man will wake one morning to find himself with double vision, or with ptosis of one or both eyes, or with both diplopia and ptosis. On inquiry there is often the history of a specially hard day's work the day before, or perhaps a

little indiscretion in the use of stimulants. After, perhaps, three or four weeks, or as many months, the paralysis disappears, and may not recur for months or even years. A second attack may appear and disappear, though it generally lasts a longer time, and frequently leaves some paresis.

Recurrent fugitive ocular paralyses rouse most forcibly the suspicion of tabes, The fleeting character of these early symptoms is puzzling. There is evidently a trophic disturbance of the nuclear cells, causing a paresis in the muscles, but in some way or other the process stops, and the cells regain their vitality. Many of the other symptoms of tabes show recessions, but none exhibit such complete abatement as the oculo-motor paralyses, unless it be the lightning pains.

It may be that what is an apparent paralysis of one muscle is really an increased action, a tonic contraction, of the opposing muscle, due to an irritation of its nerve; but the fact that ptosis is seen so frequently negatives this assumption, as in this case there is no opposing muscle, unless we call the orbicularis palpebrarum, innervated by the seventh, the opponent.

I come now to the consideration of the optic nerve. No doubt it is often affected very early, but in the cases that I have seen there have generally been some fugitive or permanent paralyses first. A considerable number of cases of tabes show signs of optic atrophy sooner or later, and it often happens, when the disease is early characterized by this symptom, that ataxia is postponed. Probably many of the primary optic atrophies are really cases of tabes overlooked. The onset of the atrophy is gradual. One eye, as a rule, begins to fail sooner than the other. Occasionally portions only of the retina are affected, and we get scotomata, but, so far as I am aware, not central scotomata. Usually there is a gradual increasing anæsthesia of the whole retina, and, as at the best, the peripheral parts are not highly sensitive, the fields are early contracted, especially for colour.

I am making some investigations in the subject of colour fields, and have been led to devise a perimeter* in which coloured lights are used instead of coloured pigments, but my researches are too recent and incomplete to draw any definite conclusions as yet.

The ophthalmoscopic appearances of the fundus are those of white or gray atrophy. The normal optic disc is pale-rose-coloured from the presence within the nerve of the vasæ propriæ. In the sclerosis of tabes there is a loss of transparency of the nerve structure; these vessels are not seen, and light falling upon the

* Shown at the Ophthalmological Society, March, 1901.

disc undergoes a partial selective absorption, hence the frequency of the bluish tinge. The disc is bluish for the same reason that milk is bluish; the physiological cup deepens, and the lamina cribrosa becomes very distinct; the vision goes from bad to worse, and finally ends in total blindness.

Through the kindness of Dr. Savill, with whom I have the honour of working as clinical assistant, I have had the opportunity of carefully examining several cases of tabes, and have his permission to use the results of my examination and the notes made on their progress from time to time:

Case 1.—The case of C. F., a man of fifty, a boot-finisher, is so typical that I will go into particulars in some detail; further, it brings out a symptom that occasionally appears in the atrophic process, viz., colour vision, about which considerable interest gathers. In 1894 he was working one night, taking a bicycle to pieces. He remembers it greatly fatigued him; he was straining a good deal, and he felt quite ill afterwards and went to bed. The next morning he awoke with a squint, the right eye turning outwards. In seven weeks' time this passed away; he saw as clearly as ever. About this time he had "rheumatics" about the shoulders and in the arms. "He got the missis," as he remarked, "to rub liniment into the parts." He went on well until 1897, when he got very weak; then in April, 1898 (four years after the first attack), he was taken with a severe pain between the eyes, and his vision began to fail. About this time he had a severe chill while watching some sports upon a cold afternoon. The next morning he noticed he could not smell properly, and his food lost its taste. He soon afterwards began to notice a fog before his eyes when looking down the street; then he said it got "nigher," and the houses across the road were not as distinct as formerly; then he had difficulty in recognising passers-by, and soon he bumped against people in the street, and had to be led by the hand. He has had pain in the pit of the stomach, and has felt pain if, by accident, he receives a jerk when passing off a step or the like; he has had, and has still, numbness down the inside of the arms, and over the sides and front of the thorax. The right knee-jerk is increased, the left absent. He has no ataxia, but is a little unsteady when standing with heels together. He has atrophy of both discs, with very clear laminæ cribrosæ; the arteries and veins are both smaller than normal. Both pupils are fixed, the right widely dilated, the left moderately contracted. He managed to continue at his work until October, 1898, though he declares he felt rather than saw it. Since December, 1898, he has not been able to see clearly even a light, though he

knows roughly the direction of the window. His yellow spots are affected more than the periphery, so that when he turns to point at a light he loses it. He is aware when the lamps are lighted in his room in the evening. Since July, 1899, he has had coloured vision, and, with this remarkable characteristic, it alternates from purplish to yellow ochre on alternate days. This came on gradually, and at first he had the change of colour during the same day, but now he has his purple days and his yellow ochre days. He can tell you beforehand what colour his vision will be on any particular day, so regular is the change.

This phenomenon is, I think, unique; I can find no record of similar cases. The more intense the light, the lighter the shade of purple or yellow ochre. He can sometimes see a sort of pattern—"like a plaid," as he describes it—in a part of his coloured field, and this may be light blue or pink. The plaid or draught-board appearance is fixed, and is always to his right side. He says if a boy with a white collar passes him in the street, he sees the collar as "purplish" on his purple days. He only has coloured vision with his eyes open, and only when in the light.

Case 2.—F. J. H., thirty-four. A very remarkable case of coloured vision. He was shown by Mr. Work Dodd at the Ophthalmological Society two years ago. Whether with eyes open or shut, he has the sensation of a screen or veil of bright emerald-green before him, in the pattern or fenestrations of which appear spots of rose-pink. At first, in September, 1898, he saw the outside world behind the screen—objects moved behind it "like a pantomime transformation scene behind a dropped transparent curtain"; but now, poor fellow! there is no outside world in his vision. Like the previous case, he knows whether the room is light or not, but cannot locate a light exactly; also, like the previous case, he has to shade his eyes when coming suddenly into the light, as it makes them ache.

In 1891 he suffered from diplopia, unsteadiness of gait, and occasional stoppages in the water for a period of three or four months; but these got better, and remained so until 1895. He then noticed loss of sexual desire, which remained absent until 1898, when it slightly returned. In the autumn of 1897 he again had bladder troubles, which still persist. Towards the end of 1897 his vision began to fail. At first a stronger glass served him, but early in 1898 he had to discontinue his work.

Both knee-jerks are absent. He has Argyll-Robertson pupils, is unsteady when standing with his eyes shut, and is somewhat, though not markedly, ataxic. He complains that his fingers go numb, and that he has to rub them.

Since the above note was taken, he has developed a sort of alternation, as in Case 1. He notices that one day he sees an extraordinarily bright green, and the rose-tint is faint; the next the green is very dark, and little specks of yellow are seen in the pattern.

Case 3.—C. S., thirty-six, a musician. In November, 1894, he had an injury (not further described). In March, 1895, he hurt his spine, and began to lose his vision. The right eye was affected first, the half of the right of the field being involved; after this the left eye became affected in the right field, and *things appeared green.* Then he gradually got blind. In 1898 he had difficulty in walking; he kicked against things above the level of the ground; he had shooting pains, and a feeling round the "stomach" as if a cord were being pulled tight. He had numbness of the side of the face in 1895, but this disappeared, and gave place to slight hyperæsthesia in 1898. K.J. absent; A.-R. pupil; "fluttering feeling" in arms and legs and eyes. He died August, 1899, from a cerebral hæmorrhage.

Case 4.—A. L., forty-one, clerk. Admitted at the end of 1897. For two years he had been very "nervous," and so could not walk without difficulty. His trouble began in 1896 with numbness in the feet. April, 1897, had diplopia, which persisted until November, 1898. Had prickly sensations in the fingers of both hands for two or three months. In May, 1898, showed symptoms of Charcot's joints in the knuckles of left hand. (Dynamometer, right 60, left 45.) This symptom greatly improved, all pain ceasing in three months. In January, 1899, he had a sudden spasm in the throat in the middle of the night, causing difficulty in getting his breath. He is markedly ataxic, but seems decidedly better now than he was two years ago. He has now no longer diplopia, and his vision is unmpaired.

Case 5.—J. L., thirty-six, a musician. For five or six years painful cramps. January, 1898, diplopia and ptosis; left pupil larger than right; neither reacts to light; the uvula was drawn to the left when phonating; voice hoarse; K.J. absent; gait natural; urine sometimes slow in coming; fluids come through nose when drinking; palate and left vocal cord paralyzed; numbness in hands lately. His diplopia caused much inconvenience, so was given spectacles with an opaque disc on the left side. From time to time he has complained of dreadful pains in the legs, although there is no ataxia. Then these got well, and his chief trouble was his eyesight. He discontinued his attendance for seven months, but reappeared in January, 1900, in a parlous state. Palatal and

laryngeal paralysis, cannot swallow, has difficulty with breathing, and looks emaciated and extremely ill. He subsequently became too ill to come to the hospital, and I have been unable to get further particulars of his condition.

Case 6.—J. S., fifty-four, upholsterer. Failing sight for a year or two, much worse in last four months; has had shooting pains lately; right hand numb; right foot feels heavier than left; has had a weakness in the bladder; does not know when he is watering or not; during the last month occasionally passes water in sleep; can stand with eyes shut without reeling; gait somewhat uncertain; can distinguish what he stands on. K.J. absent; pupils unequal, A.-R.; optic discs atrophied and cupped.

Five months later sight getting worse every week.

Six weeks later committed suicide.

Case 7.—F. W. M., thirty-one, telegraphist. In 1894 had bladder trouble; went to St. Peter's Hospital, and was given a No. 9 bougie.

1895, bladder trouble again, difficulty in making water, often retention.

1896 (autumn), aching of right eye and dimness of vision.

January, 1897, K.J. absent, discs very pale, especially left; vision, $\frac{6}{12}$, $\frac{6}{36}$; occasional pains in both legs; sexual desire diminished.

April, 1897, left pupil larger than right, left disc paler than right; walking all right, slight difficulty in starting; analgesia over legs and back of shoulders.

December, 1897, marked optic atrophy both eyes; vision $\frac{2}{60}$ and $\frac{3}{60}$; analgesia of legs, not anæsthesia.

July, 1898, sight of left eye gone; not attended since.

Case 8.—J. E. N., thirty-three, insurance agent. *Four years ago* began to have sharp pains in the bend of the back, perhaps once in three or four months; the pain horrible, "like knife being stuck in and then turned round"; was treated for gall-stones.

Two and a quarter years ago got double vision, though there was no apparent squint; this lasted six or eight weeks.

May, 1899, got double vision again, and then, after a few days, left eye turned outwards. This disappeared in December.

March, 1900, he has medium-sized A.-R. pupils, no knee-jerks, no subjective symptoms; fundus oculi normal.

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