THE PHYSIOLOGY OF THE SYMPATHETIC IN RELATION TO THE EYE.*

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[Dr. de Schweinitz reviewed the entire literature pertaining to the subject, and from this review and the various opinions of the authors quoted thought the following conclusions might be drawn:]

1. Although lachrymal secretion may be caused by excitation of the sympathetic, and increased lachrymation by section of the cervical sympathetic or removal of the superior cervical ganglion, the sympathetic itself should not be considered the nerve of secretion for the lachrymal gland.

2. Dilatation of the pupil is probably caused by contraction of a set of radially arranged muscular or contractile fibers, the so-called dilatator pupillæ, which is supplied by the sympathetic, and by inhibition of the sphincter of the iris. The dilating impulse transmitted to the iris passes through the cervical sympathetic and in general terms along the mydriatic tract of the pupil, which proceeds from a center in the medulla as far as the second dorsal nerve, follows its communicating branch to the cervical sympathetic, and arrives at the internal carotid plexus, from which point it passes to the nasociliary branches of the nasal nerve, which as the long ciliary nerves supply the muscular tissue of the iris.

3. Although experimental and clinical evidence favors the presence of a center situated between the spinal cord and the exits of the sixth cervical and fourth dorsal nerves, to which Budge relegated the origin of the pupil-dilating fibers of the sympathetic, its existence has not been definitely proven.

4. Although the nature of the ciliary ganglion has not been positively determined in any one species of animal, and, although it differs greatly in different species, the weight of evidence is in favor of the ganglion belonging to the sympathetic system, at least in so far as man is concerned. The root fibers which belong to the oculomotor end in the ciliary ganglion, where a new neuron begins for the fibers which pass to the ciliary muscle and the sphincter of the pupil, i. e., the oculomotor does not act directly on the sphincter of the pupil, but only in association with the ciliary ganglion. There is a certain amount of evidence that this ganglion is related to the pupil movements in the form of a center, and it probably contains cells which are active in the sensibility of the cornea, but lesions of the ganglion itself, although they have been considered by Grosz to be the basal cause of true neuroparalytic keratitis, have not been proved to sustain this position by experiments, inasmuch as trophic changes have not been observed after extirpation of the ganglion. Removal of the ganglion has little or no influence on intraocular tension, and its excision is not a rational procedure for the relief of glaucoma.

5. There is no satisfying evidence that the sympathetic is related to the function of accommodation, and it has not been proved that the sympathetic has any power in causing negative accommodation, nor has it been demonstrated that alterations in refraction noted after stimulation of the sympathetic are due to actual change in the lens.

6. Electrical stimulation of the cervical sympathetic produces at first an increase and later a decrease of intraocular tension, the increase being probably due to an effect on the vessels of the eye. Slow-acting, mechanically produced irritation of the sympathetic causes a rise of tension, which, according to Lodato, is independent of dilatation or constriction of the blood vessels, and also independent of the state of the pupil. Section of the sympathetic, or extirpation of the sympathetic ganglion, is followed by a fall of intraocular tension, which probably depends on vascular and, perhaps, muscular changes. The lowering of tension is more decided after excision of the ganglion than after section of the sympathetic cords, but in either case the effect is a temporary one, and may not last more than a few days, and sometimes disappears within a few hours.

7. Electrical stimulation of the cervical sympathetic produces on the side stimulated a dilatation of the pupil as a result of contraction of the dilatator pupillæ, associated, perhaps, with an inhibition of the sphincter. At the same time there may occur on the opposite side a contraction of the pupil, which either depends on the consensual pupil reflex, or represents a reflex transmitted through the sympathetic fibers joining the cranial nerves in the region of the cavernous sinus.

8. Electrical stimulation of the cervical sympathetic causes retraction of the nictitating membrane and proptosis, owing to the action transmitted to the unstriped muscular fiber. In contrast to the general rule, irritation of the sympathetic in rabbits causes a retraction of the eyeball in the orbit, which has been attributed by Heese to a contraction of the orbital vessels and the anemia which this causes.

9. Electrical stimulation of the cervical sympathetic is followed by contraction of the blood vessels of the conjunctiva and of the iris, and perhaps by alteration in the caliber of the vessels of the retina, although observations on the last-named phenomenon have been extremely contradictory.

10. Stimulation of certain areas of the brain cortex causes dilatation of the pupil, associated, if the cervical sympathetics are intact, with all the symptoms of stimulation of the cervical sympathetic. Division of the sympathetic stops the other symptoms, but not the dilatation of the pupil, which is supposed to be due to inhibition of the tonic action of the third nerve (Parsons).

11. Sympathectomy or gangliectomy causes the following effects: Myosis, narrowing of the palpebral aperture, projection of the nictitating membrane, retraction of the globe of the eye, hyperemia of the vessels of the conjunctiva, increased lachrymal secretion, diminished intraocular tension, certain ophthalmoscopic and microscopic lesions in the eyeground, and possibly trophic disturbances.

12. The symptoms of sympathetic section or paralysis lessen after a time, myosis being the most permanent, lasting sometimes for years. The degree of permanence, however, of the paralytic phenomena varies much in different animals.

13. Myosis is greater after excision of the cervical sympathetic ganglion than after section of the sympathetic cord, because it is probable that a certain tone is exercised by the ganglion; that is, that it has a different, and, as it was, a stronger action on the eye than the nerve trunk itself (Levinsohn).

14. All the phenomena of paralysis of the sympathetic nerve, especially the contraction of the pupil, which follow extirpation of the superior cervical gang-

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lion gradually become less marked and may disappear or even give place to the opposite condition, especially if the animal is anesthetized or subjected to sensory or emotional stimuli. In other words, extirpation of the upper cervical ganglion causes the symptoms of sympathetic paralysis which may disappear and give place to the signs of sympathetic excitation. Such paradoxical pupillary dilatation may depend on degenerative processes in the post-cellular nerves of the ganglion (Langendorff).

15. The myotic pupil, which follows sympathectomy or gangliectomy, responds to light stimulus, is still further contracted by eserin, and may be dilated by atropin. It is uninfluenced by cocain, which, however, may exercise its influence in widening the contracted palpebral fissure.

16. A considerable excision of the sympathetic must be made in order to prevent a rapid regeneration.

17. Narrowing of the palpebral fissure, ptosis sympathica, and enophthalmos are probably due to relaxation of Müller's muscle, aided, perhaps, by atrophy of the orbital fat. It has not been proved, although it has been asserted, that there is an actual reduction in the size of the globe, that is, a true microphthalmos, under these circumstances.

18. Sympathectomy or gangliectomy may cause increased vascularization of the eyeground, perhaps hemorrhages in the ciliary body and ciliary processes, and alteration in the retinal ganglion cells.

19. Puncture of the restiform body produces just the opposite effects of destruction of the sympathetic (Dupuy).

20. Nicotin paralyzes the activity of ganglionic nerve cells in the sympathetic. Cocain dilates the pupil by stimulating the mydriatic nerve endings in the iris. Atropin dilates the pupil, partly by a paralytic action on the oculomotor endings of the sphincter, and, perhaps, by a stimulant action on the sympathetic nerve fibers, or more likely, by causing a general paralysis of the unstriped pupillary muscle. Instillations of adrenalin, ordinarily inactive in causing dilatation of the pupil, become exceedingly active when the sympathetic is cut or the ganglion removed, and cause under these circumstances marked dilatation of the pupil.

INFLUENCE OF RESECTION OF THE CERVICAL SYMPATHETIC

IN OPTIC-NERVE ATROPHY, HYDROPHTHALMOS AND EX-OPHTHALMIC GOITER.*

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I. SYMPATHETICECTOMY IN OPTIC-NERVE ATROPHY.

History.—Excision of the cervical portion of the sympathetic nerve for the relief of optic-nerve atrophy was proposed and executed by the writer in 1899. (The date of the first operation was June 24.)

Report of Cases.—A report of this case was included in a paper[1] which was read before the Ninth International Ophthalmologic Congress, in 1899. From that paper I now quote:

T. J., aged 46, an inmate of the St. Louis City Hospital, a laborer, was admitted on account of blindness. There was no history of syphilis, rheumatism nor any systemic disease. The patient was of limited mentality. No history of his family could be obtained. He claimed to have had good health all his life, with the exception of an attack of malarial fever several years ago. The patient has been a moderate drinker of alcoholic beverages. In appearance he was robust, and he complained only of loss of vision, which in the left eye had been failing for eleven months, in the right for seventeen weeks according to his statement. Until seventeen weeks before this time he could see enough with the right eye to get around. Since then vision had steadily declined until he had light perception only—and this apparent only when light was concentrated on the eye by the ophthalmoscopic mirror. Vision of left eye =0.

The pupils were widely dilated. The ophthalmoscope showed in the right eye a white disc, particularly on the temporal side; the arteries were slightly reduced in caliber, veins normal. There was shallow, atrophic cupping of the nerve head. The retina and choroid were normal, the vitreous and lens clear. The left eye showed a disc of a dead white color throughout the whole area, arteries very small, atrophic excavation pronounced, veins reduced in caliber and choroid normal. The macula was not visible in this eye, owing to the much reduced blood supply. The vitreous and lens were clear. Vision was as follows: R. E. = perception of concentrated light. L. E. =0.

Diagnosis.—R. E.= optic nerve atrophy. L. E. = complete atrophy of optic nerve and retina.

Treatment.—Resection of the right superior cervical ganglion of the sympathetic was done. The operation was followed by conjunctival congestion, lachrymation and contraction of the pupil, slight ptosis and hypotonia.

No appreciable change in the patient's vision followed, and ophthalmoscopic examination made two weeks after operation showed no change in the appearance of the fundus, except that a cilioretinal artery in the upper part of the disc had doubled in caliber.

I was led to make this experimental operation for several reasons: 1. The use of glonoin is often followed by an improvement in vision in cases of simple atrophy of the optic nerve. 2. Glonoin enlarges the retinal vessels, as has been proved by ophthalmoscopic examination. 3. There is no question that in glaucoma simplex—a disease in which there is an atrophy of the optic nerve—improvement in vision follows sympatheticectomy. 4. Excision of the cervical sympathetic is followed by an increase in the blood supply of the orbital contents.

My second case of sympatheticectomy for optic nerve atrophy occurred in the summer of 1900. It has not been reported, for the reason that an assistant lost the case history. The result, however, was all that could be expected.

The patient was an Irishman, a bartender by occupation. He was about 40 years of age. For several months the vision of both eyes had steadily declined. Several oculists had given him medical treatment for simple optic-nerve atrophy without result. The poorer eye had vision equal to the counting of fingers at two feet. I excised the superior cervical ganglion of the sympathetic nerve on the corresponding side. Vision steadily increased to 20/100. The patient was lost sight of, but one year later he sent word that his vision was excellent and that he was following his vocation.

I regret that this case can not be more fully described. My third case of atrophy treated by sympatheticectomy occurred in 1901.

J. H., aged 64 years, a native of New York City, for 18 years had been a sailor. For many years he was excessively addicted to the use of alcohol and tobacco. In 1888 he contracted syphilis, for which he was treated for two months. Vision in R. E. began to fail in 1898; in L. E. in 1900. He was admitted to the St. Louis City Hospital on Jan. 29, 1901. The hospital record shows that he was in good general health. He was given large doses of mercury and iodids.

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1. Ball: On Removal of the Cervical Sympathetic in Certain Ocular Diseases—Glaucoma and Optic Nerve Atrophy, Neuvième Congrès International d'Ophtalmologie d'Utrecht, 1899, p. 551.