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PATHOLOGY OF THE LAR-
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SES IN TABES DORSALIS.

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A CONTRIBUTION TO THE PATHOLOGY OF
THE LARYNGEAL AND OTHER CRISES
IN TABES DORSALIS.*

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FOR the opportunity of presenting this case of tabes, in which laryngeal crises, one of them proving fatal, are associated with a chronic neuritis of the roots of the vagus and accessory portion of the eleventh nerves on both sides, I am indebted to Dr. W. Gill Wylie, of New York, who gave me the autopsy material with the following clinical history:

"Mr. Van D—, a large, robust, healthy man, denying syphilis, had not been sick except with chills and fever, until four or five years previous to his death, when he gradually became uncertain in his gait, and began to lose the proper co-ordinative use of the muscles of the legs. Very gradually this condition grew worse, so that he could not stand upright very well and his arms became ataxic. Vision became impaired and he could see with only the lower half of the retina. He did not suffer pain. The bladder, bowels and digestion remained in good order until a short time before death. During the latter stages of the illness, the man had at times *attacks of choking*. He finally became insane, and *died in one of the choking attacks*. In this fatal attack there was apparently a paralysis of the laryngeal muscles."

Microscopical Examination.—The brain and first two segments of the spinal cord were removed at the autopsy. Sections of the cord show the characteristic lesions of tabes in the posterior columns. The sclerosis cannot be traced

* Read at the New York Neurological Society, November 5, 1889.



upward beyond the point where the nuclei of the posterior columns become well developed. The nuclei of spinal accessory and vagus, and the ascending roots of the vagus are normal. The ganglion cells and fine nerve fibres in the motor cortex are normal.

The root fascicles of the vagus and spinal accessory nerves on both sides were removed from the medulla and examined separately. Sections of these fascicles, (stained double with hæmatoxyton and acid fuchsine, and by Weigert's method) show the lesions of a chronic diffuse neuritis, —areas of neuroglial increase with the atrophy of the nerve fibres. There is an increase of nuclei in the sclerotic portions of the fascicles, and a few small round cells lie about the blood-vessels in one or two places in the damaged portions of the fascicle. (Some caution is required in deciding about degenerated areas in the vagus and spinal accessory nerve roots because both of these roots contain bundles of fine medullated fibres destined to pass to the sympathetic system; these fibres have a more abundant connective-tissue matrix than the surrounding fibres, and in oblique sections, stained by Weigert's method, bundles of these fibres look like sclerotic patches). The neuritis does not affect all of the fascicles uniformly, some are but very slightly involved by it, and the roots of the right side are much more extensively involved than those of the left side. The hypoglossal and trigeminal roots are normal except that in the latter a few small round cells accompany some of the coarser endoneural septa.

Laryngeal crises in tabes were first described by Fereol¹ in 1876, and Cherchvesky² in 1881 collected 18 cases and divided them, depending upon the duration of the attack, into mild cases, lasting about 90 seconds; medium cases, lasting from five to ten minutes; and severe cases, lasting several hours. The mechanism of these crises is not uniform in all cases or all stages of the attacks, they occur in two ways. An analysis of the published cases admits of their division into two forms: 1. A spasmodic form. 2. A paretic form.

In the first form, some of the attacks, probably as a rule

the earlier and less severe ones, are of a purely spasmodic character. The larynx is perfectly normal and the crises result from a contraction of the glottis abductors. Attacks of this kind, which may last for months or years are well illustrated by Landgraf's³ case, which was under careful observation several months, and the author using the laryngoscope, was assured of the reflex spasmodic nature of the crises. McKenzie³ and Cherschvesky² each give two cases of crises in which the larynx was normal.

The second form of the laryngeal crises includes the majority of the recorded cases. The crises in these cases are reported as due to a paresis or paralysis of the abductors. This is the so-called posterior paralysis of the Germans—*posticus-lähmung* Gerhardt. (Virchow's Archiv., xxiii., 1863). The writer thinks that in the great majority of these cases of posterior paralysis producing crises, are not due to the exclusive paralysis of the abductors, as is intimated by the term *posticus-lähmung* or posterior paralysis, but are due to a weakening or paresis of *both adductors and abductors*, and that in this paresis of both sets of glottis muscles the *action of the abductors* is impaired more than that of the adductors. How the function of the abductors becomes more impaired than that of their opponents in paresis of both sets of muscles, so that in cases of laryngeal crises, the abductors appear to be exclusively paralyzed, may be better understood by considering the experiments of Semon and Horsley, Hooper and Donaldson.

Semon and Horsley (Brit. Med. Jour., 1886, p. 405) have shown in a large number of experiments on dogs and monkeys (20 experiments stimulating the uncut nerve; 12 experiments stimulating the divided nerve), that electrical stimulation of the recurrent laryngeal nerve quite uniformly caused adduction of the glottis, except when the animals were deeply narcotized. The same result occurred even when the larynx was removed from the animal. These experiments show that although the *crico arytenoidei postici* are larger than any other of the intrinsic laryngeal muscles, yet under ordinary circumstances, they are not powerful enough to resist the combined forces of their opponents,

the adductors. When animals are deeply narcotized, stimulation of the recurrent produces the opposite effect—abduction of the vocal cords. (Hooper, *N. Y. Med. Jour.*, July 4, 1885; June 6, 1886, Semon and Horsley, *l. c.*); Donaldson, (*Amer. Jour. Med. Sci.*, July, 1886), operating on dogs, found that the abductors responded to a weaker current than the adductors; an initial weak current in the recurrent nerve becoming gradually stronger produced at first abduction, which gradually gave way to adduction of the cords.

A peculiar feature of the posterior crico arytenoidei, discovered by Semon and Horsley, is that these muscles lose their electrical excitability long before any of their antagonists, when the laryngeal muscles are individually stimulated. They become fatigued and die sooner than the other muscles. In other words, a greater degree of destructive metamorphosis is coincident with their activity. A still farther distinction between the abductors and the adductors is the morphological distinction pointed out by Simonowsky, (*Inter. Centralbl. f. Laryngol.*, Vol. ii., p. 346), and Gruetzner (*Breslauer Aertz. Ztschrift.*, 1883, No. 18), that the abductors belong to the red muscles of Ranvier; whereas the adductors, at least the thyreo-arytenoideus, belong to the so-called white muscles. (This histological distinction however is not definitely settled, for the researches of these authors were incomplete when published).

Semon, (*Berlin Klin. Wschrft.*, 1883, No. 46; *Archiv. of Laryngol.*, Vol. ii., p. 197), from a study of laryngeal paralyses of neural origin, made the statement that in cases of injury or organic disease of the centres or trunks of the motor laryngeal nerves, the abductor muscle or muscles succumb first. (Semon's statement is also supported by Rosenbach, *Breslauer Aertz. Ztschrft.*, 1880, Nos. 2, 3; *Berlin Klin. Wschrft.*, 1884, No. 17; *Virch. Arch.*, Bd. 99). The difficult thing to understand about Semon's statement is why the abductors should be thus selected when the same motor nerve and nucleus supplies both adductors and abductors. Why, for instance, should a chronic neuritis of the roots or trunk of the vagus containing the motor fibres

for all of the laryngeal muscles (except the crico-thyroids) cause an earlier or more extensive paralysis in the abductors than in any of the other muscles?

Unless the motor fibres of the abductors are relatively much more numerous than the adductor fibres—and this is not likely to be the case for the abductor group of muscles is more voluminous—there is no reason why a chronic neuritis of the vagus roots should not involve the adductor nerve fibres just as much as the abductor fibres. Probably the truth of the matter is, that with such a lesion, the abductors suffer no greater loss of power than the adductors, but as the direction of the fibres in the abductors—the posterior crico-arytænoidei—seems to put them at a greater mechanical disadvantage in moving the arytæmoid cartilages than the lateral muscles; a uniform diminution of absolute power in both the adductors and abductors, may produce a far greater impairment of the abductors than of the adductors. Experiments show that the equilibrium of the glottis inclines in favor of the adductors in the normal larynx (except in conditions like ether narcosis), and if the adductors are stronger in the normal larynx under ordinary circumstances, we should expect their strength still more prominently asserted in the disturbed equilibrium of the glottis, when both sets of muscles are paretic, for the subtraction of a given amount of absolute power from each set of muscles would diminish the action of the abductors in a greater ratio than the adductors, which are larger, stronger, and work at the glottis from a better mechanical standpoint.

Thus it may be that the predilection of the abductors to become paretic, as observed by Semon, is only apparent, and, as Gowers (*Dis. Nerv. Syst.*, 1888, p. 692) suggests, it would be better to speak of this condition as loss or impairment of abduction, rather than as paralysis of the abductors or posterior paralysis, in the sense that the abductors are exclusively paralyzed.

Believing that a good many of these so-called posterior paralytic forms—the second form of the crises—are caused by a chronic neuritis of the motor laryngeal fibres in the roots (or trunk) of the vagus, it seems that they occur in

this way. The neuritis causes a (nearly equal) reduction of absolute power in both the constrictors and dilators of the glottis, which renders their equilibrium so unstable that with the slightest reflex or direct motor impulses the adductors, working on a superior mechanical vantage ground, gain the ascendancy, and a crisis results. In other words, the exciting cause of many of the crises of this form, is a reflex or direct motor sent through the recurrent nerve to a paretic, unstable group of antagonistic muscles with the balance of power in favor of the adductors, operates like the stimuli of the recurrent nerve in the experiments of Semon and Horsley. This excites a spasm of the adductors which is intensified or prolonged and induced the more readily perhaps, from the impairment of the function of the abductors being greater than that of the adductors, although both sets of muscles are paretic to a nearly equal extent. When the adductors become fatigued by their tonus, or should a condition like ether narcosis occur from the attack, the crisis gradually ceases.

Such an explanation of this second form of the crises accounts for the sudden way in which they occur as attacks, and is supported by the fact that deadening the reflexes from the fauces pharynx and larynx by cocaine and bromides are of benefit in many cases.

Changes in the nerve roots and in the peripheral nerves are so frequently present in tabes, and central lesions in the medulla are so comparatively infrequent, that probably chronic neuritis of the roots—analogueous to the neuritis of the spinal nerve roots—or trunks of the laryngeal nerves is quite uniformly the lesion producing the laryngeal crises. The neuritis of the roots of the tenth and accessory portion of the eleventh nerves in this case explains very well how the laryngeal crises of both kinds may occur, if some points in the laryngeal innervation are referred to.

The superior laryngeal nerve is the sensory nerve of the larynx, and is composed as a rule entirely of fibres from the accessory portion of the spinal accessory. Vrolik (Kirke's Handbook of Physiology, 1889, p. 614), found in the chimpanzee that the internal division of the spinal accessory

passed directly to the larynx without fusing with the vagus. The supposed motor supply of the superior laryngeal nerve to the crico-arytænoidei postici is denied by Schech (Lit. of Biol., ix., p. 258). Stimulation of the central end of the superior laryngeal produces decided closure of the glottis (Krause, Virch. Arch., Bd. 98). The recurrent laryngeal nerve, supplying all of the muscles except the crico-thyroids, as a rule derives its motor fibres from the vagus; occasionally this nerve varies in its composition, and its motor fibres come partly from the vagus and partly from accessory vagus. The recurrent nerve also contains a few sensory fibres, the centripital path of which is not definitely known.

The statements of Bischoff, Longet, Bernard and Morganti (references given in Donaldson's paper), to the effect that the accessory portion of the spinal accessory is principally a motor nerve are quite untenable at present. If the superior laryngeal was chiefly motor, stimulation of its central end ought not to produce such a pronounced reflex closure of the glottis; this experiment indicates that the superior laryngeal is more a sensory than a motor nerve. A study of the central termination of the accessory portion of the spinal accessory seems to show that it is mainly a sensory nerve. Recurrent motor fibres like those of the vagus passing to the nucleus ambiguus may be seen in the adult medulla, passing in from the very uppermost roots of the accessory portion; but as the sections pass down to the middle and lower levels of the roots of the accessory portion, these recurrent fibres become fewer and fewer and disappear, so that it is doubtful if recurrent motor fibres like those of the vagus, exist in the middle and lower roots of the accessory portion. I think from this that all of the root strands of the accessory portion are sensory fibres from the larynx and pharynx, except the uppermost strands nearest the vagus, which contain motor fibres for the crico-thyroids and inferior constrictor, and a vicarious motor supply for the pharyngeal and recurrent branches of the vagus, which nerves seem to be subject to occasional variation in their composition, as they sometimes have a portion of their

usually almost exclusively vagal supply of motor filaments replaced by motor fibres from the accessory portion.⁴

A chronic neuritis of the roots (or trunk) of the accessory portion of the spinal accessory, irritating the laryngeal sensory fibres, would be responded to by motor impulses passing down the vagus which would cause, as in electric stimulation of the motor laryngeal fibres, adduction of the cords. This lesion would account for the first form of the crises. If this neuritis should be sufficiently destructive anæsthesia of the larynx would result. Krause and Fränkel each report a case of anæsthesia of the larynx in conjunction with tabetic laryngeal crises.

An early irritative stage of the chronic neuritis in the vagus roots (or trunk) might also cause crises of the first form, by stimulating the motor laryngeal fibres and producing direct spasms of the adductors. A later destructive of this neuritis might induce paresis or paralysis in both the abductors and adductors. But with this condition abductorial impairment is greater than adductorial, and this coupled with the experimental facts that the abductors are unable to resist the forces of their antagonists in the normal larynx and become fatigued sooner, suggests as an explanation of the second form of crisis, that at certain times with reflex or direct motor stimuli, (which might not be of sufficient intensity to provoke a spasm in the normal larynx), the adductors get the balance of power and their tonus may be enhanced by the greater degree of metabolism in the abductors. When both the vagus and accessory vagus are diseased together, the conditions are favorable for the production of violent crises of the second form.

Review of Some of the reported Cases of Laryngeal Crises in Tabes.—Krause⁵ collects eight cases which were examined laryngoscopically. The vocal cords were quite uniformly in the median or adduction position. Three of Cherchvesky's cases had paralysis of the left cord, and in one case there was paralysis of the abductors on both sides.

⁴ References to other less definitely settled points in laryngeal innervation are given by Lennox Browne, *The Throat and its Diseases*, 1887, p. 477.

Wegener's⁸ two cases had paralysis of the left abductors. Fränkel's⁹ case had paralysis of one cord and paresis of the other. Fournier (*Leçons sur le période prae ataxique de tabes d'origine syphilitique*, Paris, 1885), reports five cases of one-sided laryngeal paralysis in tabetics who had no crises. Kahler's⁹ case had paralysis of the left cord, and crico-arytænoideus posticus and dysphagia. Crises in this case could be elicited at times by causing the patient to swallow a glass of water.

Krause⁷ reports two cases, in one of which anæsthesia of the larynx was present, but there was good laryngeal reflex irritability so that every touch of the larynx with the sound elicited a crisis. In the second case laryngeal irritability was absent; the larynx could be sounded without awakening crises. In both cases crises could be produced by irritating the nasal fossæ. Oppenheim¹⁰ presented two cases having gastric crises. In one of the cases, the crises was of a purely spasmodic character for several years, but subsequently the crises assumed the features of the second form associated with loss or impairment of abduction. The voice became rough and the crico-arytænoidei postici and the thyro-arytænoideus became paretic. Pulse anomalies were also present in this case.

In the second case, the crises were very violent and one of them proved fatal. (Microscopical examination in this case showed neuritis of the vagus and accessory vagus roots) Besides the fatal crisis recorded in this paper, a third fatal crisis is instanced by Keller, (quoted by Cherchvesky). Krishaber¹⁰ had to do tracheotomy in an urgent case of tabetic laryngeal crises.

In a third case of Oppenheim's¹¹ there were spasmodic pharyngeal attacks followed by movements of swallowing. A fourth case, by the same writer, had paralysis of the right cord which remained immobile when the right recurrent nerve was stimulated electrically, while the left cord responded to stimulation of the left recurrent.

Landgraf's³ case of purely spasmodic crises of the first form is very interesting in connection with the fact, that two years afterward the patient developed paralysis of the

sterno-cleido-mastoid and trapezius on one side. (Reported by Martius, Berlin Klin. Wochenschrift, No. 8, 1887). This would indicate that the spasmodic reflex crises were caused by a neuritis of the accessory portion of the eleventh nerve, which subsequently extended to the spinal portion of the nerve. McBride's¹³ case of tabetic crises also had paresis of the sterno-cleido-mastoid and dysphagia, frequent heart action, and finally Cheyne-Stokes respiration, indicating neuritis of both the vagus and spinal portion of the spinal accessory.

In Weil's¹⁴ case, the crises, which as a rule do not appear until the tabetic symptoms are well established, were the initial symptoms of the disease, and enhances the suggestion of Buzzard and Semon, of testing the knee-jerk in all cases of laryngeal paralyses. Weil regarded the case as one of posterior paralysis. (Jeleneffy, Berlin Klin. Wochenschrift, 1888, p. 728, reviewing Weil's case, and finding that the crises were often originated by peripheral stimuli, questions whether there really was posterior paralysis). Weil instances Krishaber's,¹⁵ Lhoste's¹⁶ and Morgan's¹⁷ cases as of posterior paralysis similar to his own.

Some writers have reported cases of laryngeal cases due to ataxia of the laryngeal muscles. The consideration of ataxia of the glottis muscles is exceedingly complicated. A central lesion would be indicated in laryngeal ataxia rather than lesions in the roots or trunks of the laryngeal nerves, which are believed by the writer to be the cause of the crisis in the majority of the cases. If such cases of laryngeal ataxia-producing crises exist, it must be very difficult to distinguish them from the other two forms alluded to in this paper.

Summary of the Lesions in the Reported Cases.—Jean¹ found atrophy of the left vagus, accessory, and recurrent laryngeal nerves and left thyro-arytænoideus. Kahler⁹: thickening of the ependyma involving the right vagus nucleus, and principally the nucleus of the right accessory vagus nerve. (The paralysis in this case could be explained by the damage done to the recurrent or motor fibres, passing to the nucleus ambiguus, by the lesion in the sensory

vagus nucleus.) Demange¹⁹: lesions similar to those in Kahler's case. Landouzy and Dejerine: atrophy of the vagus roots and nucleus of the vagus accessory nerve. Oppenheim: neuritis of the vagus and accessory vagus roots and degeneration of the recurrent laryngeal nerve.

The *pharyngeal crises*—dysphagia, with or without spasmodic deglutitory movements, which occasionally are in company with the laryngeal crises—may be accounted for by a chronic neuritis of the roots (or trunks) of the ninth, tenth, or accessory portion of the eleventh nerves; but it is difficult to explain these crises in detail, owing to the complicated and apparently variable distribution of these nerves to the pharyngeal plexus. The simplest account of the pharyngeal plexus is given by Schwalbe ("Lehrbuch," 1878, p. 874). All three of the nerves of the eighth pair send both sensory and motor branches to the pharynx. The principal sensory conductor is the glosso-pharyngeal, which also sends motor fibres to the stylo-pharyngeus, and middle constrictor. The principal motor nerve is the vagus. The accessory fibres to the pharynx are sensory, except those to the inferior constrictor, and except when accessory fibres replace the motor vagus fibres in the pharyngeal branch of the vagus. A chronic neuritis in the roots or trunks of one or more of these nerves might produce reflex or direct pharyngeal movements, or a degree of paresis of some of the muscles, giving rise to dysphagia.

The *gastric crises* might occur in two ways: Reflexly—for instance, by a neuritis involving the sensory pharyngeal filaments; directly, by a neuritis of the upper three cervical nerve roots, or of the vagus accessory roots or of the trunk of the vagus, which is the path of the visceromotor fibres upon which depend peristaltic contraction of the thoracic œsophagus, stomach, and small intestines (Gaskell, "Jour. of Phys.," vol. vii.).

The *bronchial crises*—spasms of the larger bronchi, with attacks of rough coughing—might be caused by a neuritis of the vagus which carries bronchial sensory fibres, and the visceromotor fibres supplying the smooth muscles of the bronchi.

The *cardiac crises*—anginoid attacks, and the pulse anomalies occurring in tabes—might be caused by a vagus neuritis interfering with the cardio-inhibitory fibres. Cardiac disturbances might also occur from the cord lesion in the upper dorsal region, whence, according to Gaskell, the cardio-augmentor fibres issue in the anterior roots.

The reason of the other tabetic visceral symptoms may be made clearer by referring to the researches of Gaskell (*loc. cit.*) on the distribution and origin of the sympathetic fibres. The fibres of the sympathetic arise in the spinal cord and lower medulla, and issue thence, as finely medullated fibres in the anterior and posterior roots, in three great channels, viz.: The cranio-cervical outflow, in the vagus, in the spinal accessory passing to the vagus through the ganglion trunci, and in the upper three cervical nerve roots; the thoracic outflow, situated between and including the second dorsal and second lumbar nerve roots; the sacral outflow, from the second and third sacral nerves. The cranio-cervical outflow contains the cardio-inhibitory fibres and the visceromotor fibres for the stomach and circular fibres of the small intestine. The thoracic outflow passes upward to the cervical sympathetic trunk and ganglia, medially to the prævertebral ganglia (semilunar and mesenteric) and splanchnics, and downward to the hypogastric plexus. The thoracic outflow contains the visceromotor nerves for all parts of the body, glandular nerves, visceroinhibitory nerves, cardio-augmentor nerves, and visceromotor nerves for the circular fibres of the hind-gut, and some of the vaso-dilator fibres. The sacral outflow, composing the nervi-erigentes, consists of fibres passing directly to the hypogastric plexus, whence they pass upward to the inferior mesenteric ganglion and downward to the bladder, rectum, and generative organs. The sacral outflow contains vaso-dilator fibres for the penis (stimulation of the anterior second and third sacral roots produces erections in rabbits: Gaskell, "Jour. of Phys.," vol. viii.) and vaso-dilator fibres for the lower extremities. This outflow also contains the visceromotor fibres for the bladder, uterus, and longitudinal muscles of the rectum.

These three regions of the cord, whence issue the finely medullated fibres to the sympathetic system, correspond so closely with the situation of the cervical nucleus, the columns of Clark, and the sacral nucleus, that Gaskell thinks that the central origin of the sympathetic is partly in these nuclei. As the central tabetic lesions, or neuritis of the spinal nerve roots, may involve these portions of the cord, we may try to explain how some of the other visceral and trophic symptoms in tabes arise.

The tabetic lesion in the second and third sacral segments, interfering with the vaso-dilator fibres of the sexual organs, would account for some of the sexual disturbances and the *clitoris crises*. The paroxysms of rectal pain and tenesmus—*rectal crises*—if not caused by the lower dorsal portion of the cord lesion, involving the visceromotor nerves of the circular rectal muscles, may be caused by the sacral portion of the cord lesion interfering with the motor nerves of the longitudinal muscle layer of the rectum. The *vesical disturbances and crises, the urethral crises*, and the weakness of the rectal and vesical sphincters may be caused by the lesion in the second and third sacral segments involving the visceromotor fibres of these organs.

As the vaso-dilator and vaso-motor fibres to the lower extremities pass out of the second and third anterior sacral roots and lower portion of the dorsal region respectively, lesions in these portions of the cord may account for the trophic disturbances in the lower limbs, viz.: Brittleness of the bones, atrophy of their heads, etc., arthropathies of the knee-joint, changes in the tarsal bones and joints, flattening the foot (tabetic foot of Charcot and Féré), temporary œdema without renal lesions, local sweating, perforating ulcer of the foot, unprovoked ulceration of the toes, bad growth of the nails, etc.

Intestinal troubles are manifested by constipation, spasmodic attacks, and peculiar diarrhoeal attacks, which Pierret supposed to be of vaso-motor origin. If not due to a vagus neuritis, the intestinal symptoms may be caused by the lesion in the dorsal cord. The constipation may occur from a destruction of some of the visceromotor fibres to the gut.

The dorsal region of the cord probably contains the vasodilators of the guts.

The lesion producing the *nephritic crises* is difficult to locate. The path of the visceromotor fibres to the ureter is not known. According to Bradford ("Journal of Phys.," vol. ix., p. 358) the renal vaso-motor fibres issue from the cord in the ninth, tenth, and eleventh anterior dorsal roots. The lesion in this portion of the cord might interfere with the renal circulation.

LITERATURE OF CASES OF LARYNGEAL CRISES IN TABES.

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