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A Case of Subacute Unilateral Bulbar Palsy, with Autopsy.

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A CASE OF
SUBACUTE UNILATERAL BULBAR PALSY

*WITH AUTOPSY.**

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A BULBAR palsy of a bilateral type, taking either a subacute or a chronic course, is not a rarity. But when we come to examine the literature upon this subject, with reference to cases of a unilateral type, we find but few on record. Such have been reported by Pel (1), Ballet (2), Erb (3), Remak (4), Hirt (5), and a few others.

Ever since the year 1860, when Duchenne published his study on Progressive Glosso-labial-pharyngeal Palsy, more or less interest has been shown in regard to this subject. In 1870, Charcot and Leyden made known to us their views and established the fact that this disease was due to a chronic degeneration of the motor nuclei in the floor of the fourth ventricle. A few years later Joffroy (6) discussed another type of bulbar disease, quite distinct

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from the above, which he called pseudobulbar palsy. Very soon afterward Jolly (7) reported a most interesting case, with autopsy, of the pseudobulbar type, and now the literature abounds in the records of such cases. Among these are cases of Lepine (8), Eisenlohr (9), Oppenheim and Siemerling (10), and Ross (11).

I do not hesitate to add my case to the list of bulbar palsies, as the subject is still novel enough to warrant the description of every case in which a post-mortem examination helps to elucidate the clinical features of these palsies.

By the term progressive bulbar palsy we of course understand a paralysis of the lips, tongue, palate, and throat muscles, due to a degeneration of the nuclei within the medulla which give origin to the nerves which supply these parts.

The division of bulbar palsies into acute or apoplectiform (Leyden and Senator), subacute (Erb), and chronic (Erb, Duchenne, Wachsmuth) is the classification most generally followed now by our best authors, and I think it will answer for our unilateral cases as well.

In these unilateral cases the combination of symptoms varies: There may be present only a simple hemiatrophy and hemiparalysis of the tongue, with or without a hemiparalysis of the palate, pharynx, and throat muscles. It seems, however, that in all these cases the hemiatrophy and paralysis of the tongue are the most prominent symptoms, but those which give the patient the least annoyance. In former times this especially interested the anatomist, and as it seemed to occur frequently with tabes dorsalis, many authors, Ballet (12) among them, were of the opinion that it was extremely rare to find this condition without tabes. Charcot, Erb, and Strümpell soon corrected this erroneous idea, and proved by their enumeration and discussion of individual cases that this symptom could not be considered

a pathognomonic sign, but that it occurred often enough to necessitate, in such cases, an examination for other symptoms of tabes. Hirt states that most of the German authors up to 1885 considered a hemiatrophy of the tongue as a positive pathognomonic sign of tabes.

The case which I propose to discuss this evening is one which I presented before this society about nine months ago and it now claims our especial attention for the following reasons: 1. It is a case of subacute unilateral bulbar palsy verified by an autopsy. 2. Since so much ambiguity still exists with reference to the exact anatomical lesion which produces bulbar palsies, a statement of the facts in this case may throw a little more light upon this region of the brain in question. 3. The grouping together clinically of symptoms which result from an affection of these parts which naturally functionate together seems to me to be of great importance in arriving at a correct solution in regard to an anatomical diagnosis.

The patient, M. E., seventeen years old, was born of healthy parents. Found history negative. His mother is living and enjoying good health. His father died a short time ago with Bright's disease. The family history is negative with respect to any hereditary nervous disease. Patient has always been in good health up to two years ago, when he was taken down with a severe attack of perityphlitis, from which he recovered, however, after five weeks of illness. He had suffered from catarrhal pharyngitis more or less ever since childhood. Two years ago last spring the glands on both sides of his neck, in the region of the sterno-cleido-mastoid muscles, began to enlarge. In the summer of the following year (1892) an abscess formed in one of these glands on the right side, and had to be opened. After this the remaining glands began to create so much discomfort for the boy that after six weeks' endurance, on August 17, 1892, the glands on the right side, together with a large portion, as it seems, of the sterno-cleido-mastoid muscle, were ex-

cised at Mount Sinai Hospital. On September 1 of the same year a second operation was performed and the glands on the left side were removed. These glands were examined by Dr. L. Stieglitz and found to be of tubercular nature. The patient rapidly recovered, and nothing was noticed in the way of any disturbance of the parts which might have been involved in the operation. He was under treatment for his pharyngitis when, during the latter part of November, Dr. Friedenburg, who had taken charge of the case, discovered, on asking the patient to show him his tongue, that it deviated to the right side. Of this condition the patient was not at all aware until he was told of it. Shortly after this the patient experienced some trouble in swallowing. He soon became hoarse and coughed with difficulty. All these symptoms developed within ten days. It was in this condition that he was sent to Dr. Sachs's department at the New York Polyclinic. A thorough examination brought out the following points: 1. Very marked deviation of the tongue to the right side when protruded. 2. Distinct atrophy of the middle right half of the tongue; it appeared shriveled, and when taken between the fingers had a decided sponge-like feeling. 3. The electrical examination gave the following result: On direct excitation the faradaic response of the right half of the tongue was much weaker than the response of the left side. The contractions were not as prompt and sudden as in normal muscular tissue. On indirect examination a similar result was obtained, but the differences were not as marked. There was, furthermore, on direct examination, increased galvanic excitability of the right side, the contractures being of a slow and wave-like character. K. C. C. was greater than A. C. C. Taste and sensation were not at all affected.

When the mouth was held open and the patient breathed in a quiet manner, it was noticed that the rhaphe of the soft palate was pulled toward the left side, together with the elongated and slightly thickened uvula. The left arcade seemed to be smaller than the right, which hung down with the lower edge much farther than the left. The right arcade appeared broad and smooth. The left was drawn into longitudinal folds. As soon as he was asked to phonate "Ah" the paralysis became

more evident and the whole palate looked very much deformed. The left side was contracted still more and the right side remained perfectly motionless. Electrical examinations gave similar results to those obtained in the tongue. Sensation and taste were normal. There was great difficulty in deglutition. It was not with the same difficulty, however, as in cases in which both sides are involved, familiar to us from cases of chronic bulbar palsies. He would choke and cough, and after several attempts he would succeed in getting down his fluid or food. Solid food could be taken much more readily than liquid.

On looking at the posterior wall of the pharynx I noticed what I thought at first to be the formation of a growth, probably a tubercular mass, situated on the left side. In reality this proved to be the normal wall of the pharynx which was pushed further toward the front, while the right side seemed to be pulled back and further away from my view. The electrical examination here was also similar to that of the tongue. Sensation was normal. The patient's voice was hoarse and low-pitched; it was difficult for him to cough.

The boy was sent to Dr. Gruening for laryngoscopic examination. His report states that dysphonia is due to disease of the right recurrent laryngeal nerve, causing a complete unilateral palsy on the right side. The patient is poorly nourished and exhibits marked swellings on the neck from tubercular glands.

Looked at from the front, the neck on the left side appeared to be a little fuller and springing slightly forward. The right side was very much flattened. The position of the "larynx" and "hyoid bone" was somewhat altered. The great horn of the hyoid bone on the left side was very prominent and easy to locate. On the paralyzed side it was hard to find, and also appeared to be a little lower than on the sound side. The larynx was twisted a little obliquely and no longer parallel with the median line. As the boy swallows, his larynx is drawn or pushed upward toward the right side. Two other muscles on the right side of the neck seemed to have been involved in this case, the sterno-cleido-mastoid and the trapezius. The former can hardly

be excited either by the faradaic or galvanic currents, very few fibers responding. Either the greater part of the sterno-cleido-mastoid muscle had been removed during the operation for removal of the glands or the nerve supplying this muscle had been severely injured. Of the latter muscle only the upper portion was involved, and the faradaic and galvanic currents showed this very clearly, since the strongest currents produced only the slightest contractions. All the other cranial nerve functions were normal, and no other nervous disturbance in the body was anywhere to be found. Reflexes were everywhere normal, both superficial and deep.

The heart was normal, likewise the lungs. The pulse had ranged at different sittings between 90, 80, and 78 pulsations. Respiration 17, 19, and 18. Urine was normal. No change had taken place in these conditions up to the time when I presented him before this society, except that the reflex of the palate and pharynx had disappeared, together with a very much diminished faradaic contraction on the right half of the tongue.

It was thus in this condition that I presented him before this society, and two months following the boy died. A summary of the notes taken until the time of his death is as follows: Patient began to grow very much weaker, and suddenly on March 26, 1893, he had an attack of respiratory failure and a pulse which increased in rapidity to 140 beats to the minute. He recovered from this attack partly, and then continued in a condition of slight respiratory difficulty. Complained of hot and cold flashes constantly. His speech became worse, so that he could hardly speak above a whisper. There was excessive salivation and continual drooling from the right side of the mouth. On April 11th I found that he could hardly protrude his tongue beyond the edge of his teeth, but during all his sickness, and even now, his lips remained normal and could be brought into perfect action.

The palate and pharynx remained normal on the left side. April 20th he had another attack of respiratory failure and died in a very short space of time.

Such was the history of this case, beginning as a unilateral palsy of the tongue, soft palate, pharynx, and larynx on the

right side, the paralysis remaining stationary for a time, and then advancing into an incomplete bilateral palsy shortly before death.

The question which was of interest to us at the time when I presented him before this society was, Where could the lesion be situated which could produce such a palsy, and of what nature was it?

My aguments at that time were that the lesion might occupy one of three positions: First, a point just external to the exit of the pneumogastric and spinal accessory or vago accessorius and hypoglossal nerves from the skull—viz., just behind the angle of the jaw; second, on the surface of the medulla; third, it might be nuclear.

First, as regards the lesion behind the angle of the jaw. Here these nerves are situated very deep and in close proximity to two very important structures—the internal jugular vein and the internal carotid artery. I reasoned thus: A tumor, say an enlarged gland, pressing upon these nerves in this position may produce such a palsy as the above-mentioned. I spoke of this fact in connection with a similar report by Gowers (13) of a case of deep tumor of the neck situated in this region. In my case I hardly believed this to be the site of the disease for the following reasons: First, there was no arterial or venous disturbance of any kind, which one would naturally expect to find if the lesion were situated in close proximity to the internal jugular vein and internal carotid artery; second, the glosso-pharyngeal nerve, which lies in close contact with these nerves in this region, was in no wise involved in all its branches; third, that all the various parts which are supplied by the nerves in question, and which would be included if the lesion were peripheral, have not been affected; fourth, the electrical reactions are such as one does not usually find with a peripheral lesion.

A lesion on the surface of the medulla was more probable. In a similar case of unilateral palsy involving the tongue, soft palate, larynx, and also the sterno cleido-mastoid and trapezius muscles, reported by Mackenzie (15), he believed the lesion a nuclear one. But he concluded that because the sterno cleido-mastoid and trapezius muscles were involved, and the nerve branches supplying these muscles were close together on the surface, while the nuclei were some distance apart and could not be covered by a small lesion, the lesion was on the surface of the medulla.

Dr. B. Sachs (14) also mentions the fact that deposits either of a syphilitic or tubercular nature at the base of the skull may call forth palsies of the various cranial nerves, and in several of the cases cited I noticed that he records an involvement of the hypoglossal and vago-accessorius roots.

Now, as regards my own case, although we had a disturbance of the upper portion of the trapezius and of the sterno-cleido-mastoid muscles, which disturbance was accounted for in a satisfactory manner, I did not believe that the lesion in this case was confined to the surface of the medulla. For a lesion of such a nature as the one I supposed this one to be would have produced in this locality much more serious trouble than existed in this patient. There was no progression, nor were there any other symptoms present than those which I found at the first examination. Thus, from the general course of the disease, together with the symptoms present and cranial nerves involved, I excluded the surface of the medulla.

This brought us to the consideration of the only other possible localization of the lesion—viz., in the nuclei of the nerves which govern these palsied parts. These groups of cells form the conjoined nuclei of the spinal accessory, vagus, hypoglossal, and glosso-pharyngeal nerves,

and only a very limited localized lesion in this part of the medulla is sufficient to affect them. All the facts in the case lent their support to this view. The onset, which was subacute, the slow course of the palsy—viz., one nerve after the other becoming involved, and just those parts becoming palsied which usually functionate together—and then the slight electrical changes, together with the exclusion of the only two other possible places where the lesion might have occurred, all this pointed to the bulbar nuclei as the probable seat of disease.

As regards the nature of the lesion I reasoned thus: A boy, seventeen years old, healthy in every respect up to the time that he was taken sick, shows marked scrofulous disposition, and has a mass of tubercular glands on both sides of the neck. This aroused the suspicion that the lesion in the medulla might be tubercular, and a solitary tubercle in the region of the nuclei would explain all the symptoms. I thought that this would be the case; in this I was mistaken.

Autopsy.—This was made six hours after the death of the patient.

Roof of skull was normal. Dura was fairly rich in blood. Pia was smooth and delicate. No extravasations anywhere to be found.

Base of Brain.—Pia delicate and subpial spaces are all free from exudation.

Cranial nerves appeared normal.

Medulla.—To all outside appearances this was natural in form and not at all asymmetrical. It was neither too soft nor too hard. There was nothing unnatural in the fourth ventricle. Striæ acusticæ appeared all thoroughly developed. No hæmorrhages and no exudation. Cerebellum normal in appearance. Surface of brain: Cortex was a little pale. Hemispheres wree normal. I made a few cuts through the pons and medulla in the fresh state, but saw nothing in the way of a hyperæmia,

softening, or doubtful coloring. The gray substance was strongly in contrast with the white. Blood-vessels all appeared natural to the naked eye. I placed the complete brain in Müller's fluid and allowed it to harden for microscopic examination.

Microscopic Examination.—Transverse section through the speech center revealed nothing abnormal. Internal capsule likewise was found in normal condition.

The cruri cerebri were perfectly normal in appearance.

Third nerve nuclei also normal.

Sixth and seventh nerve nuclei normal.

The posterior longitudinal fasciculi are normal in appearance and well marked.

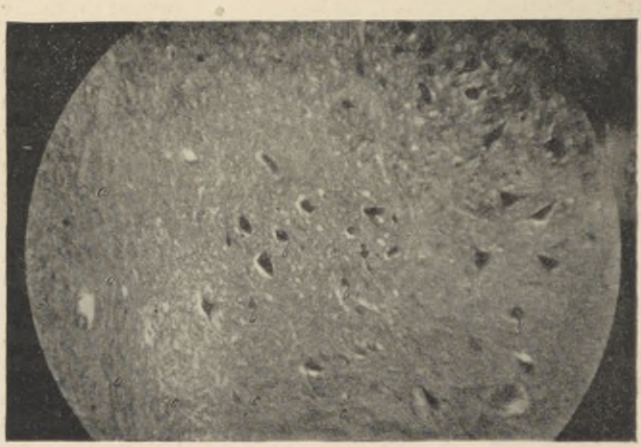

FIG. 1.—Hypoglossal nucleus, left side. Photomicrograph, from a section stained after Pal. *a*, median rhaphe; *b*, degenerated ganglion cells; *c*, lower limit of gray matter.

Transverse section through the pons showed nothing abnormal. The entire region, therefore, of the sixth, seventh, and eighth nerves I found perfectly natural in appearance. Ascending root of the fifth nerve clear and distinct.

As we approach the region of the ninth nerve, we find on the right side a very slight degeneration in the ganglion cells of

this particular nucleus. The column known best as the respiratory bundle appears almost completely degenerated. On the left side everything in this region appears perfectly normal with the exception of the slightest portion of the lower and

FIG. 2.—Hypoglossal nucleus, right side. From same section as Fig. 1. *a*, median rhaphe ; *b*, degenerated ganglion cells ; *c*, lower limit of gray matter.

outer part of the respiratory column, which appears to be affected in the degeneration. As we examine the sections from above downward, we observe a degeneration on the right side of the vagus, hypoglossal, and vago-accessorius nuclei, together with the completely diseased respiratory column. (See Fig. 4.) The vagus appears only very slightly affected, but the hypoglossal is diseased in a very marked degree. The ganglion cells are very much diminished in number, are abnormally rounded, and with but few cell processes left. They are in a very much atrophied state, shrunken and granular in appearance. Few that are left have any nucleolus present in their cell body. The ground substance does not seem to stain as readily on the right side as it does on the left, and appears to be less compact.

The nucleus ambiguus and Roller's hypoglossal nucleus appear natural. The degeneration extends on the right side down to the lowest level of the hypoglossal nucleus, and is most marked just at the point of exit of the vagus nerve. On the left side this nucleus is also not quite natural. The cells are diminished in number and slightly degenerated. (See Figs.

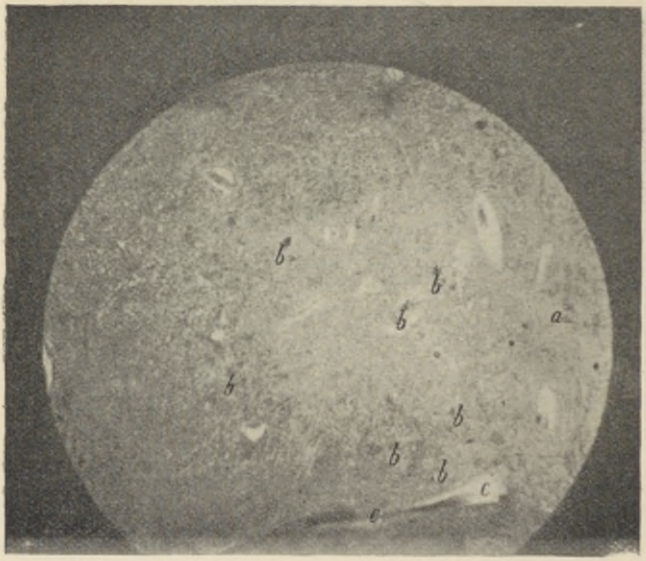

FIG. 3.—Same as Fig. 2, with larger field.

1 and 2.) Again, the respiratory bundle appears degenerated in its lower and outer parts throughout its entire course (see Fig. 5), except in the region of the ninth nerve, as above mentioned. The vagus nucleus on the left side does not stain very readily and appears slightly degenerated.

An examination of the intramedullary roots shows them to be decidedly less prominent and much thinner on the right side than on the left.

Thus, summing up all these facts of the microscopical examination, we find:

First. The motor cortex, internal capsule, crura cerebri, and pons of normal condition. The nucleus of the twelfth

FIG. 4. Photomicrograph of completely degenerated respiratory bundle, right side. *a*, margin of bundle.

nerve on the right side very much diseased, while on the left side only in a slight degree. The nuclei of the tenth, eleventh—viz., vago-accessorius—slightly affected; a little more on the right side than on the left. The nucleus of the ninth on the right side only very slightly affected. The respiratory bundle appears completely degenerated on the right side, while on the left, in the region of the hypoglossal nucleus, only its lower and outer portions are diseased. In the region of the ninth nerve a few fibers are affected in this lower and outer portion.

The intramedullary roots of the ninth, vagus, vago-accessorius, and hypoglossal nerves are less prominent on the right side than on the left. Otherwise everything appears to be perfectly natural up to the exit of the first cervical nerve in the spinal cord.

Let us now review the clinical symptoms in the light of the autopsy. Within a space of ten days this patient developed a complete unilateral palsy of the right side of his tongue, soft palate, pharynx, and right recurrent laryngeal nerve. There was no disturbance of his respiratory or cardiac organs, or any other conditions present which

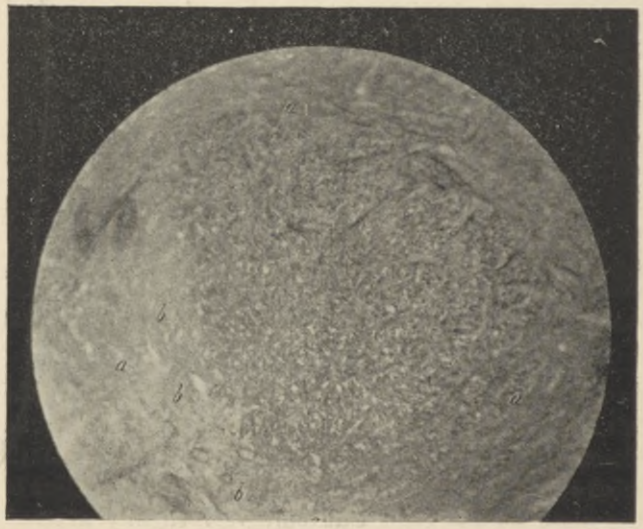

FIG. 5.—From same specimen as Fig. 3, left side. *a*, margin of bundle; *b*, degenerated lower and outer portion of column.

should have called our attention to the affection of any other cranial or spinal nerve, except that of the ninth, tenth, eleventh, and twelfth nerves.

We find, on microscopical examination, a marked degeneration of the nucelus of the hypoglossal nerve on the right side, together with slight degeneration in the adjacent nuclei, also a completely degenerated respiratory bundle. How is this to be explained?

It is unfortunate that authors still differ as to the interpretation to be given to these nuclei, more particularly those of the spinal accessory and glosso-pharyngeal.

Lockhart Clarke (16) remarks that the spinal accessory consists of two roots: a lower one, whose rootlets are collected into an external branch and supply the sterno-cleido-mastoid and trapezius muscles; an upper one, whose rootlets are collected into an internal branch, which joins the pneumogastric and is distributed to the larynx, pharynx, and palate. This has been proved beyond a doubt by the experiments of Reid (17), Bischoff (18), Bernhard and Beevor, and Horsley (19).

The lower roots arise with the spinal nerves from the anterior horns of the spinal cord, in the region of the cervical and upper brachial nerves. The upper roots have a double origin: first, from their own special nucleus, continuous behind the central canal with that of the pneumogastric, and the others from the proper nucleus of the hypoglossal nerve in front of the canal.

This close anatomical connection between vagus, spinal accessory, and hypoglossal nerves Clarke considers of the highest importance in regard to the light which it throws on the complex and associate movements concerned in deglutition, vocalization, and articulation. He suggests that these nuclei are the center for combined movements of these nerves.

It is just in this region that the lesion is localized in my case, with the additional involvement of the respiratory bundle, which does not appear to be mentioned in other

cases as a natural consequence of disease of the hypoglossal nucleus (Turner) (20).

As the autopsy proves that the hypoglossal, vagus, and the vago-accessorius nuclei, together with the respiratory column, were diseased, and if it is furthermore true that the respiratory column contains root fibers from the vagus, vago-accessorius, and glosso-pharyngeal nerves, then it is probable that some fibers of the glosso-pharyngeal nerve are connected with the hypoglossal nucleus.

The symptoms on the right side point to an involvement of the glosso-pharyngeal nerve—viz., the loss of reflex in the palate and pharynx and the involvement of the pharyngeal muscles. On the left side there was no involvement of the glosso-pharyngeal nerve, according to the symptoms present, and we found the greater part of the respiratory column intact, except its lower and outer portion, which can readily be accounted for by the beginning degeneration of the vagus and vago-accessorius nuclei.

According to the facts in this case, I offer the following explanation: The slight degeneration of the vagus and outer vago accessorius nuclei are not in comparison with that of the hypoglossal. Therefore, with really no involvement of the vagus at the commencement of the disease, as I have the right to suppose from the absence of symptoms characteristic of disease of this nerve, I consider the hypoglossal nucleus as the actual nucleus of origin for the supply of the tongue, palate, pharynx, and right recurrent laryngeal nerve. Also that the fibers of the glosso-pharyngeal nerve, which were diseased in this case at the beginning, must undoubtedly take their origin, together with the vago-accessorius, in this region, and then ascend in the respiratory column to the glosso-pharyngeal region, and make their exit with that nerve. The very slight degeneration in the ninth nerve nucleus could not explain the symptoms which were

present and characteristic of disease of this nerve. The left side helped to substantiate this theory, for here we had no pharyngeal symptoms present, but a beginning tongue and vocal-cord affection, together with, toward the end, a complete respiratory failure, and we find only a degeneration in the lower and outer part of the column, in the region of the hypoglossal nucleus. Can this, therefore, as I would suggest, be the course of the vagus fibers in the respiratory column? I do not want to establish this as a fact, for I think we are in need of more autopsies before this view can be confirmed.

The loss of reflex in the palate on the right side, which I also attribute as a loss of function on the part of the glosso-pharyngeal nerve, only took place toward the latter part of the disease. The very slight degeneration of the proper nucleus of the ninth, vago-accessorius, vagus, and twelfth nerves on the right side, together with the beginning degeneration of the twelfth and tenth nerve nuclei on the left side, were signalized by the first attack of respiratory failure. When this degeneration was about to complete itself on the other side, the patient had his second attack and died in a short space of time. Thus, after the first attack of respiratory failure, he had developed symptoms on the part of those nerves whose nuclei became affected in the latter part of the disease—viz., the ninth, tenth, and vago-accessorius on the right side, and the same nerves, together with the twelfth, on the left side.

As regards the glosso-pharyngeal nerve supplying sensory filaments to the front of the soft palate, palatine arches, or back of the tongue, I could not confirm this view, for with disease of this nerve in my own case there was no disturbance of sensation. Taste was likewise normal and not at all affected. It is generally believed that nausea is produced in a reflex manner through this nerve. I could pro-

duce the same in the beginning of the trouble, but later, when the reflex of gagging disappeared, this also vanished on the right side.

As regards the possibility of an accurate anatomical diagnosis, I think there should be no difficulty if we remember that paralysis of parts that functionate together, but have a distinct anatomical innervation, is due to nuclear lesion.

As regards the nature of the lesion, I did not find a tubercle nor did I find any tubercle bacilli in specimens which were stained for that purpose. All that was present consisted of a simple atrophy of the ganglion cells and fibers, motor in function. Whether this may be due to some toxic agent circulating in the blood, probably a ptomaine manufactured by the tubercle bacilli, I will leave for others to decide.

A consideration of the preceding facts in this case and autopsy leads to the following conclusions:

1. That the region of the hypoglossal nucleus gives origin to nerve fibers which supply the tongue, palate, pharynx, and larynx on one side of the body.

2. That the column of nerve fibers known as the respiratory bundle consists of fibers from the glosso-pharyngeal, vagus, and vago-accessorius nerves, and that the lower and outer portion of this column probably serves as the locality for the vagus and vago-accessorius fibers.

3. That the glosso-pharyngeal nerve seems to control the reflexes of nausea and gagging in the soft palate and pharynx, and also to send some of the motor filaments to the pharyngeal muscles. These latter filaments take their origin in the hypoglossal nucleus and ascend in the respiratory column to the nucleus proper, and then make their exit with the glosso-pharyngeal nerve.

4. That the soft palate muscles are not innervated by fibers from the seventh nerve.

Bibliography.

1. Pel. *Klinische Wochenschrift*, No. 29, 1887.

2. Ballet. De l'hémiatrophie du larynx. *Archives de neurologie*, vii, No. 20, 1884.

3. Erb. *Deutsches Archiv für klin. Med.*, xxxvii, p. 265, 1885.

4. Remak. *Berliner klinische Wochenschrift*, 1886, xxiii, 25.

5. Hirt. *Berliner klinische Wochenschrift*, 1885, 20.

6. Joffroy. *Gaz. méd de Paris*, 41, 44, 46, 1872.

7. Jolly. *Archiv für Psychiatrie*, iii, 1872.

8. Lepine. *Revue mensuelle de méd. et de chir.*, 1877.

9. Eisenlohr. *Deutsche med. Wochenschrift*, 1887, 32.

10. Oppenheim and Siemerling. *Charité Annalen*, xii, 1887.

11. Ross. *Brain*, July, 1882.

12. Ballet. *Archives de neurologie*, 1884, vii, 24.

13. Gowers. *Diagnosis of Cerebral Palsies.*

14. B. Sachs. *Multiple Cerebro-spinal Syphilis.*

15. Mackenzie. *British Medical Journal*, March 3, 1883.

16. L. Clarke. *Phil. Trans.*, 1868.

17. Reid. *Physiological, Pathological, and Anatomical Researches*, Edin., 1889.

18. Bischoff. Müller's *Archiv.*

19. Beevor and Horsley. *Proc. of the Roy. Society*, xliv, 1888.

20. Turner. *Journal of Anat. and Physiology*, 1889.

Leyden. *Charité-Annalen*, Berlin, 1885–'86.

Etter. *Corresp.-Blätter für schweizer Aerzte*, 1882, 24.

Remak. *Archiv für Psychiatrie*, Berlin, 1891–'92, xxiii.

Jackson, H. *London Hospital Reports*, 1864, i.

Erb. *Wien. med. Bl.*, March, 1887, 1 and 2.

Erben. *Wien. med. Bl.*, 1887.

Scheiber. *Berlin. klin. Wochenschrift*, 1889, xxvi.

Senator. *Neurol. Centralbl.*, xi, 1892; *Charité-Annalen*, 1891, xvi.

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A WEEKLY REVIEW OF MEDICINE.

EDITED BY

FRANK P. FOSTER, M.D.

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