ARCHIVES OF LARYNGOLOGY.

CLINICAL REMARKS*

ON THE PROCLIVITY OF THE ABDUCTOR FIBRES OF THE RECURRENT LARYNGEAL NERVE TO BECOME AFFECTED SOONER THAN THE ADDUCTOR FIBRES, OR EVEN EXCLUSIVELY, IN CASES OF UNDOUBTED CENTRAL OR PERIPHERAL INJURY OR DISEASE OF THE ROOTS OR TRUNKS OF THE PNEUMOGASTRIC, SPINAL ACCESSORY, OR RECURRENT NERVES.

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THE curious and important fact, which forms the subject of the present communication, is by no means entirely new or unknown. The relatively greater disposition to implication of the abductor filaments was illustrated by Gerhardt[1] as long ago as 1863, and by Morell Mackenzie,[2] in 1868, and alluded to since by Schech,[3] Penzoldt,[4] Burow,[5] again by Mackenzie,[6] by myself,[7] by Bosworth,[8] by Rosenbach,[9] and possibly by others.

With the only exceptions, however, so far as I know, of Bosworth's argument—with which I but very partially

* Dr. Semon's MS. was received in December, 1880. Its publication has been unavoidably so long postponed. This statement is made both as acknowledgment of the fact and as apology.—ED.

[1] Studien u. Beobachtungen über "Kehlkopflähmung." *Virchow's Archiv*, vol. xxvii, p. 88, *et seq.* (Cases 10, 12, 13.)

[2] "Hoarseness and Loss of Voice." Cases 19 and 20.

[3] "Experimentelle Untersuchungen über die Functionen der Muskeln und Nerven des Kehlkopfs." Würzburg, 1873, p. 44.

[4] "Ueber die Paralyse der Glottiserweiterer." *Deutsches Archiv für Klin. Medizin*, vol. xiii, 1874, p. 107, *et seq.*

[5] "Paralyse der Musc. crico-arytænoidei postici, etc." *Berliner Klin. Wochenschrift*, 1879, Nro. 33.

[6] "Diseases of the Throat and Nose," 1880, p. 440.

[7] German edition of Mackenzie's work. Foot-notes on pages 574, 575, 587, 629.

[8] *New York Med. Journal*, Nov. 1880, p. 460.

[9] *Bresl. ärztl. Zeitschrift*, 1, 2, 3, 1880.

agree,[1]—of Rosenbach's paper (which comes to my knowledge only in a short abstract in *Schmidt's Jahrbücher*, vol. 188, 1881, p. 134, while I am correcting the proof-sheets of this communication), and of the remarks in the English and German editions of Mackenzie's recently published "Diseases of the Throat and Nose," all these allusions are merely incidental, and intended rather to explain, by some hypothesis or other, what is considered a pathological curiosity, than to come to certain definite and important general conclusions.

Although this does not apply to Mackenzie's later remarks in his text-book, nor to my foot-notes in the German edition thereof, yet the form in which they had to be couched in a book of this kind, naturally prevented the subject from receiving the more elaborate consideration from a clinical point of view that it certainly deserves.

Its importance is, I hope to show, so great—especially with regard to the diagnosis of diseases of the brain and chest, in which the laryngeal affection plays the rôle of an important, sometimes pathognomonic symptom—that I consider it not only justifiable, but even very desirable, that this question should be once more and separately brought before those who have the opportunity of either corroborating or correcting my statements and conclusions.

The question at issue stands thus:

It is well known that, with the exception of the cricothyroid muscles (the tensors of the glottis), which are supplied by the superior laryngeal nerve, *all* the other laryngeal muscles, adductors as well as abductors, receive their motor innervation from the recurrent laryngeal nerve.[2]

But this fact, simple and natural as it seems to be to all of us who are accustomed to look at it as to a self-understood matter, nevertheless invites a consideration concern-

[1] I think it right to state here that the MS. of this paper was already in the hands of the Editor, when I became acquainted with Dr. Bosworth's important paper. I have preferred to refer to some of his statements which bear upon the question under consideration, by making foot-notes under the original text, instead of interrupting my own argument by a discussion of controversial points.

[2] The thyreo-epiglottici and ary-epiglottici muscles are *perhaps partially* supplied by the superior laryngeal nerve. (Leube, *Deutsches Archiv für Klin. Medizin*, vol. vi, p. 266.)

ing a very interesting, very important, and wholly unsolved question, viz., as to the manner in which this small nerve accomplishes its most complicated and intricate functions.

Quite apart from the delicacy of the mechanism of the *vocal* apparatus, over which the recurrent nerve, in conjunction with the superior laryngeal nerve, has to preside, the natural question must present itself at once to our mind: In what way are the diametrically opposed functions of opening and closing the glottis accomplished, over which, as far as we know at present, the recurrent nerve alone presides? Are we to believe that the nerve is, in fact, a homogeneous one, but that different stimuli, or stimuli coming from different centres, can set up in its root different forces, conducted through the *same* nerve-fibres, and resulting at one time in a general involuntary contraction of the abductor muscles, and at another in the almost always voluntary contraction of their antagonists?[1] Or are we to suppose that, though the nerve is apparently homogeneous,

[1] Bosworth states that the action of the glottis-opening muscles is, "of course, purely involuntary, in that it is entirely beyond the control of the will." This statement seems to me somewhat too absolutely framed. I go so far with him, that I believe that under normal conditions of breathing this action is quite as automatic as that of respiration in general; but at the same time I do not see why the more energetic contractions of the abductor muscles, during *deep* inspiration, should not be occasionally quite as much under the control of the will as the act of deep inspiration itself. Why should the wider opening of the glottis in a voluntary deep inspiration be regarded as an exclusively involuntary and reflex movement, *following* the voluntary act of deep inspiration, and why not as a *part* of this act itself? It must not be forgotten that the posterior crico-arytænoid muscles are striped muscles. This is not the occasion on which to discuss this very interesting physiological question more fully, but I personally am much more inclined to reason as follows:

"In order to understand correctly the movements taking place within the larynx, it should always be remembered that they are not *exclusively* results of the *direct action of the muscles*. Dilatation and constriction of the different laryngeal compartments can, up to a certain degree, be produced as well *by the elastic membrane of the larynx returning to its former state*, after certain muscles, which brought it into a state of tension, have ceased to act. This state of things much resembles that acting upon the thorax during inspiration and expiration." (Luschka, "Der Kehlkopf des Menschen," p. 115. The italics are my own.) Now it seems to me that the state of the glottis, seen with the laryngoscope, in a *quietly-breathing, healthy person* (viz., an opening of middle size, but larger than that seen in the dead body, with but very little dilatation in inspiration and equally insignificant constriction in expiration), is the result of a *permanent automatic* "half-tension" (Luschka, *l. c.*, p. 132) of the abductor muscles, *superadded* to the *natural tension* of the elastic membrane (which tension alone is best illustrated by the "cadaveric position" of the vocal cords, *i. e.*, the state of the glottis seen after death), and that a *further* contraction of the glottis-openers, involving a greater dilatation of the glottis, might, in accordance with the general laws of the mechanism of respiration, be effected *voluntarily* or *involuntarily*, as the case may be.

it consists in reality of a bundle of strictly differentiated fibres, bound together simply by a common nerve-sheath, and actually differentiated throughout their peripheral course, in fact having ganglionic centres of their own?

This physiological question, which is no doubt not only of the greatest interest in itself, but, it will be seen, also of the highest importance for the pathological question, which forms the subject of this paper, has scarcely met with its due share of attention. It is simply taken as granted that the recurrent laryngeal nerve supplies the greater part of the motor innervation of the larynx, and here the curiosity of most observers has ceased.[1] This is not the occasion to attempt to solve the question from the physiological point of view, but I may state at once that the pathological facts to be communicated later on strongly support the greater probability of the second hypothesis, viz.: that the fibres of the recurrent laryngeal nerve are differentiated in the nerve-centre itself, and only surrounded by a common neurilemma.

It will be seen that even this hypothesis is by no means sufficient to explain *all* the pathological phenomena with which we shall have to deal in this paper; but if it be accepted so far, we have at once a *locus standi* for the rejection of some conclusions concerning the effects of a lesion or disease set up in the centres or in the trunks of the spinal accessory, pneumogastric, or recurrent nerves, which might

[1] An incidental allusion to this question will be found in a very interesting annotation in *The Lancet*, 1878, vol. i, p. 584, and in my reply to it, *ibid.*, p. 630. Schech (*l. c.*, p. 42) believes it very probable that the abductor possesses, besides those fibres originating from the pneumogastric and accessory nerves, others, which are in some form of relation with the respiratory centre, and are stimulated from this source, but says that this hypothesis has not been proved up to the present. Merkel ("Stimm- und Sprachorgan," 1863, p. 154) makes a statement similar to that of Bosworth to be presently quoted, but more guarded. He says, after speaking of some other controversial points in relation to the nervous supply of the larynx: "It is not less doubtful, whether the muscles closing the glottis, on the one hand, and the glottis-openers, on the other, are presided over by specifically differentiated nerves (Magendie, H. Ley), although it seems to me personally at least very probable, that the glottis-openers, which are in a state of involuntary tension, must be presided over by nerves of a different kind and of another origin than the glottis-closers, which are almost entirely under the control of the will." Bosworth says (*l. c.*, p. 461): "Reasoning from analogy, we are justified in the conclusion, that this glottis-opening function of the larynx is presided over by an independent ganglionic centre, situated in the brain, but which neither physiological experiment nor pathological investigation has as yet been able to locate."

be very naturally made with regard to the anatomical relations of the single terminal branches of the recurrent laryngeal nerve. These anatomical relations are as follows:

The recurrent nerve is of considerable length. "The *left* is longer than the right in consequence of its being given off much later than the right from the trunk of the pneumogastric nerve. It springs from the latter at a very sharp angle, whilst the pneumogastric passes in front of the end of the arch of the aorta; winds round the transverse part of the arch, outside of the ligamentum arteriosum; passes between the aorta and left bronchus to the posterior part of the former; leaves this between the origin of the carotis primitiva sinistra and the truncus anonymus, and ascends in front of the œsophagus in the groove formed by this part and the trachea." . . . . .

"The *right* recurrent nerve is given off from the trunk of the pneumogastric as the latter descends in front of the origin of the subclavia dextra. It winds round the lower and posterior convexity of this vessel, crosses the carotis dextra behind its origin, and ascends behind this vessel, running toward the middle line in the groove formed between the right lateral wall of the trachea and the œsophagus."[1]

Although during this long course several twigs are given off by the nerve (rami cardiaci inferiores, tracheales and œsophagei superiores), yet it is only *when in close proximity to the larynx itself* that those branches are sent off which serve as motor nerves to the individual muscles of the organ. These are:

a. The posterior crico-arytænoid nerves. As the trunk of the inferior laryngeal nerve is passing close along the posterior crico-arytænoid muscle, it gives off two branches, one of which passes above, the other beneath the crico-arytænoid articulation, under the lateral edge of the muscle. The former of these two branches commences to run obliquely toward the median line at the border of the lower and middle third, the latter at the border of the middle and

[1] This as well as the following anatomical quotations are taken from Luschka, "Der Kehlkopf des Menschen," Tübingen, 1871, pp. 164–166.

upper third of this edge, between the plate of the cricoid cartilage and the substance of the muscle. The first branch having given twigs to the lower half, the second to the upper half of the muscle, both combine and form a small trunk, which becomes visible as the

b. *Nervus arytænoideus transversus* on the upper border of the posterior crico-arytænoid muscle. This nerve crosses the upper edge of the cricoid plate close to the inner end of the crico-arytænoid articulation, and distributes its final branches within the transverse arytænoid muscle, which is thus supplied with nerves from both sides.

c. *The nervus crico-arytænoideus lateralis* is a slender twig, which is given off by the trunk of the recurrent nerve, whilst passing either across the middle of the muscle of the same name, or under some of its bundles.

d. *The nervi thyreo-* and *ary-epiglottici* are given off by the trunk at the upper border of the crico-arytænoid muscle. They send forth their extremely slender terminal twigs into the muscles of the same name.

e. *The nervus thyreo-arytænoideus* is the final branch of the recurrent laryngeal nerve. It descends between the lateral crico-arytænoid and thyreo-arytænoid muscles, and is concealed from view midway between the thyroid cartilage and the processus muscularis of the arytænoid cartilage. In the depth of the gap left between these muscles, the trunk of the nerve splits up, like a brush, into thin twigs, which radiate from below into the substance of the muscle of the vocal cord proper.

This description clearly establishes the previous statement, viz., *that the individual muscular branches are only given off* by the trunk of the inferior laryngeal nerve *when close to the larynx.*

Considering this anatomical fact, and another equally plain one, viz., that the diameter of the trunk of the nerve is a very small one (1 mm. when close to the larynx, according to Luschka), it is very tempting to draw at once the conclusions alluded to above, viz.:

1. That any lesion or disease affecting the nerve from its centre to the spot where it gives off the first branch for

the posterior crico-arytænoid muscle, must of necessity affect *all* the laryngeal muscles (with the exception, of course, of the crico-thyroid muscles, and perhaps of the epiglottidean muscles).

2. That, on the other hand, any impairment of mobility —if not due to mechanical or myopathic influences [1]—of a single one or some of the laryngeal muscles seems to point out clearly that the cause of this impairment must be a *local* one, due to injury or disease of the *respective nerve twigs*.

These conclusions, as sketched here, cannot be rejected *a priori* as unreasonable. The anatomical facts seem to support them strongly, and although it was known at a very early period in laryngological studies that the abductor and adductor muscles could become separately affected,[2] yet the possibility has been emphatically denied that such a *partial* paralysis could be due to either *central* causes or affections of the *main nerve trunks*. Navratil,[3] the champion of this opinion, expresses himself as follows:

"The question whether, under these circumstances, the paralysis be a central or a peripheral one, can be easily decided. Considering that there cannot be any disease-producing cause in the centre which would act upon the adductors alone, without affecting simultaneously the abductors, any central disease can be excluded with certainty. The same is to be said of the pneumogastric and recurrent nerves, and I dispute Gerhardt's view, that any pathological cause could affect just that part of the recurrent nerve (the diameter of the entire nerve is very small) from which (?) the narrowing and the tension (?) are effected.[4]"

I do not know whether this view is still defended by Navratil, or whether he has any followers in the camp of laryn-

[1] See my paper "On Mechanical Impairments of the Functions of the Crico-Arytænoid Articulation," etc. *Med. Times and Gazette*, 1880, vol. ii, Nro. 1577 and *seq.*

[2] Gerhardt, "Studien u. Beobachtungen über Kehlkopflähmung." *Virchow's Archiv*, vol. xxvii, p. 88.

[3] *Berliner Klin. Wochenschrift*, 1869, pag. 383.

[4] It would confuse my subject if I entered upon any discussion of the other very controversial statements made in this quotation and the lines following it in the original.

gologists, but I think it is still held by a great many general practitioners.

Yet it is openly fallacious in consequence of the omission of a very simple but very important consideration, viz., that we must distinguish between a *complete* and an *incomplete* as well as between an *acute* and a *gradually progressive* lesion of the nerve![1]

[1] It is certainly remarkable that the same omission has led Bosworth to make a statement, in part *directly opposed* to that of Navratil, but equally inadmissible, viz.: to argue that *all* cases of what he well terms "primary" paralysis of the abductor muscles must be of a *central* nature (*l. c.*, p. 476.). In his argument on this point he says (*l. c.*, p. 475): "The question arises, does the seat of the original morbid changes which produce the paralysis lie in the nerve-trunks? I do not see how this view of the subject *can be entertained for a moment.* Any disease of the recurrent laryngeal nerve, *which has progressed so far as to destroy its conductivity* (the italics are my own), must destroy and paralyze all the muscles which it supplies." The *conclusion* of this last sentence would be certainly quite correct and unattackable if the *premise* were beyond doubt; but it is just here, in the presumption that the disease in question must be judged from those cases in which it has "progressed so far as to destroy the conductivity of the recurrent laryngeal nerve," that I think Dr. Bosworth's argument is open to criticism. Must a disease affecting a nerve, or a pressure exercised upon a nerve, destroy, *of necessity*, the conductivity of *all* its fibres. If so, I see *no explanation at all* for the cases in which, during life, paralysis of one or of both the abductors *alone* was observed and in which, after death, compression of the *trunk* or *trunks* of the recurrent nerves was found. (See Cases 3, 4, 5, 9, 16, 17, 20, 22 of the following literary retrospect.) But the essential point in which I differ from Dr. Bosworth, is that I believe that in such cases it is *impossible* that *all* fibres of the nerve should have been destroyed. *If* they were, we would simply, as he himself justly observes, find trophic changes in *all* the muscles supplied by the nerve. But here we are in possession of positive facts: During life we find paralysis of the *ab*ductors alone; after death, evidence of compression of the *trunks* of the recurrent nerves, trophic changes of the *abductors* only, *integrity* of the other laryngeal muscles. In other words, clinical observation as well as the results of the *post-mortem* examination *prove*, that none but the abductor fibres *can* have suffered. In the face of such facts it seems to me that any theory which attempts to explain *uniformly* the pathology of this disease by a presumption of *central* changes having taken place in *all* cases entirely collapses. Nor do I think, as is admitted by Dr. Bosworth (*l. c.*, p. 476), that because it is "among the possibilities," that a pressure may "discriminate among the nerve-fibres,"—that he is justified in altogether rejecting the possibility of the original morbid changes, which produce the paralysis, *ever* lying in the nerve-trunks (see above), and this simply on account of the—*theoretically*—small probability that such a discriminating pressure should affect *both* recurrent nerves in a large series of cases. That such a contingency is *rare*, is proved by the small number of cases hitherto recorded; but it certainly has happened *repeatedly*, and as no theory of *central* origin would account for these cases, I believe that a theory of a *uniformly* central origin of bilateral paralysis of the abductors is untenable, though admitting, at the same time, that in a *large proportion* of these cases the original morbid changes, which produce the paralysis, are no doubt to be found in the brain.

I here take leave of Dr. Bosworth's important paper. There are several more points in it which invite discussion, but I am afraid that these foot-notes, which are being added to my paper four months after its completion, give it much more of a polemical character than I could have wished. Still, I feel that, with regard to the importance and comparative novelty of the subject un-

If we have an acute *complete* lesion, such as is produced by, say transverse cutting of the pneumogastric or recurrent nerves, experiments on animals,[1] as well as occasional observations [2] on human beings, have shown beyond doubt that the consequences are such as would be expected theoretically, viz., *total loss of the functions of all the laryngeal muscles* (with the exception of the crico-thyroid muscles), with the usual results and symptoms. So far, therefore, facts and theoretical conclusions quite agree with each other.

Matters, however, become entirely different, if we have to deal with either an *incomplete acute* or an *incomplete gradually progressive* lesion. The former of these two conditions is certainly an extremely rare one in practice, though it can be easily produced by experiment; the latter, on the other hand, is the form which is not only practically the most important, but almost the only one which occurs in reality.

For if we set aside for a moment the rare contingencies in which the nerve centres or trunks suffer from traumatic influences, or in which the centres are suddenly disabled from fulfilling their functions by certain acute poisonous influences, or by the still rarer occurrence of a hemorrhage, or the bursting of an abscess into the floor of the fourth ventricle,[3] almost *all* the other causes which can be thought of as occurring in practice are such as belong to the category of *slowly progressive* and—at any rate *at first—incomplete* lesions. Implication of the roots of the pneumogastric and spinal accessory nerves in central diseases, such as syphilitic processes, progressive bulbar paralysis, disseminated cerebro-spinal sclerosis, locomotor ataxy with final participation of the medulla oblongata; tumors or aneurisms within the

der consideration, it would be wrong to take no notice of so important a paper as that of Dr. Bosworth, and as, unfortunately, our opinions do not agree on some important points, I have considered it a duty to offer the reasons for my objections to some of his ideas.

[1] Schech, *l. c.*, p. 31, and *seq.*

[2] Fano, "*Schmidt's Jahrbücher*," vol. xci, p. 19; Kappeler, *Archiv der Heilkunde*, 1864, p. 271; Mackenzie, *Brit. Med. Journal*, December 24, 1870, and "Diseases of the Throat and Nose," p. 433, etc.

[3] In the strict sense of the term the last two contingencies might as well be said to belong to the class of "traumatic" causes.

skull, pressing on the spinal accessory nerve; tumors of the neck; aneurism of the carotids; goitres, cancers, gummata; aneurisms of the arch of the aorta, or the subclavia dextra; mediastinal tumors pressing on the trunk of the pneumogastric nerve; tumors of a similar nature and position pressing on the trunk of the recurrent nerve; and in addition to these, carcinoma of the œsophagus, pleuritic adhesions implicating the right recurrent nerve close to the apex of the right lung in phthisis, and large pericardial exudation (Bäumler), are all processes, which may lead to lesions of the motor innervation of the larynx, and different as they are from each other in nature and in progress, yet have one point in common, that if they implicate these centres or these trunks, the implication is a comparatively *slow* and *gradual* one. The small size of the nerves does not alter this fact. Of course a smaller nerve will be more quickly implicated by a carcinoma, or suffer earlier from a pressure exercised upon it, than a large one; but at all events there must be some time in which a small nerve as well as a larger one *begins* to experience the effects of the disease-producing cause encroaching upon its functions.

The question now presents itself: What are the early symptoms in such cases?

Here we may consider the two hypotheses introduced at the commencement of this paper.

If we believed that *all* the fibres of the recurrent nerve were identical and not differentiated, but that different stimuli coming from either the same or from different centres, could be transmitted through them, it seems to me that in *every* case in which there is an incomplete impairment affecting the roots or trunks of the spinal accessory, pneumogastric, or recurrent nerves, we could reasonably expect *one and the same* sequence only, viz.: *diminution of* ALL *the functions of* ALL *the laryngeal muscles suppplied with motor fibres by the recurrent laryngeal nerve, and this diminution in proportion to the number and strength of the fibres disabled by the disease-producing cause.* In other words, every paralysis of the recurrent nerve, unless acute and complete from its commencement, ought to begin with

loss of the adductive *as well* as of the abductive power, and this equal impairment should progress *pari passu* with the disablement of the still conducting fibres, until at last, all of them having become devoid of conducting elements by some external cause or internal retrogressive metamorphosis, the stage of complete paralysis of the nerve with immobility of the corresponding vocal cord in the cadaveric position is attained.

Now I must say, that although for several years past this question has interested me very much, and although I have been seeking a case in which the symptoms should develop in this way, I have not been able to find a single one, while during the same time I have seen several cases in which the paralysis developed under my eyes, and in which the progress was different. Yet I am not disposed to dispute, after the fashion of some writers of the day, the possibility of an occurrence, for the sole reason that it never occurred to myself, and I am quite ready to believe that a paralysis of the recurrent nerve which finally becomes complete, can manifest itself during its beginning and progress by the symptoms sketched above.

But what I wish most decidedly to contradict is the probability of the hypothesis first alluded to, by which such an occurrence could be explained, viz.: that the fibres of the nerve are *all identical*, and that *different* stimuli can be transmitted through *all* of them, exciting at one time the action of one set of muscles, at another the action of their antagonists.

No doubt this hypothesis explains why and how in such a hypothetical case of gradually progressive paralysis the action of all the laryngeal muscles should gradually become weaker, and more and more insufficient; but on the other hand its own indefensibility becomes clear from the considerations already alluded to.

If in reality all the fibres of the recurrent nerve were identical, the loss of some of them by any pathological process must, as I have tried to explain, *under all circumstances* be followed by the *same* consequences. The *intensity* of these consequences might vary according to the number of the disabled fibres, and perhaps according to the rapidity

of the pathological process; but in *all* cases the differences *could* only be *quantitative*, never *qualitative*.

In other words, if this hypothesis were correct, it would not admit of a *single* exception to the rule. A single exception, a single good observation, proving that in a case of gradually progressive paralysis of the roots or the trunks of the accessory, pneumogastric, and recurrent nerves one set of laryngeal muscles was either before their antagonists or even exclusively paralyzed, annihilates the entire hypothesis of the homogeneousness of the filaments of the recurrent nerve, for if they were really homogeneous, the laryngeal muscles could *never* be separately affected by a lesion involving the trunk of the nerve itself.

Now we have not one, nor a few, but many cases on record, in which clinical observation and the *post-mortem* examination have actually shown that, although the disease-producing cause acted upon the whole nerve-trunk, yet *one* set of laryngeal muscles only became affected, or one much more than the other, so that it is sufficient to refer to any of the text-books of laryngology or to the medical journals of the last decade; moreover, I have but little doubt, that almost every reader of this paper has observed if not published cases of this sort as occurring in his own practice.

I do not think that there is any possibility of reconciling this positively ascertained fact with the theory of the homogeneousness of the recurrent nerve, and I therefore believe that this theory must be completely given up.

The second hypothesis appears the more probable, and one *a priori* harmonizing much more with the complexity and delicacy of the vocal functions of the laryngeal muscles, viz.: that the filaments of the recurrent nerve are strictly differentiated throughout the course of the pneumogastric nerve and even possess ganglionic centres of their own.

The adoption of this hypothesis at once gives a clue to the explanation of the frequent clinical observations, that in cases of intrinsic nerve disease as well as of external mischief to the nerve, and in cases of central as well as of peripheral lesion, one set of the muscles is earlier affected than the other or even exclusively. If, for instance, a dis-

seminated sclerotic affection of the brain should happen to affect those ganglionic centres only which form the nuclei of the abductor filaments of the recurrent nerve; if a tumor of the neck should happen to press on those nerve-fibres only which supply the crico-arytænoideus lateralis, the thyreo-arytænoideus, and the arytænoideus proprius muscles,[1]—it would not need further explanation to prove that in the first instance we would see, laryngoscopically, the vocal cords not in the cadaveric but in the phonatory position, because the posterior crico-arytænoid muscles only were paralysed, and that, on the other hand, in the second instance we would see the glottis widely open, and meet with complete aphonia in consequence of the inability of the solely paralysed adductor muscles to bring the vocal cords together for the purpose of phonation. In each of the two cases this state of things could remain stationary (viz.: if the pathological process occasioning the paralysis came to a standstill), or could lead to complete paralysis with its consequences (viz.: if the antagonistic centres or fibres, which were left free at the beginning, became also affected later on).

I find myself thus far in complete accord with v. Ziemssen who, although not entering upon any discussion on the *modus operandi* of these gradual and incomplete paralyses, expresses himself as follows:[2]

"Paralysis of individual branches of the recurrent, which go to the laryngeal muscles and to the mucous membrane of the trachea and the larynx, may arise through incomplete lesions of the trunk of the recurrent; for instance, when it is exposed to unequal pressure, or when, for any other cause, the nerve-filaments are affected in an unequal degree by degenerative changes. This generally takes place at the beginning of a severe lesion of a nerve, gradually leading to complete paralysis of conduction—for instance, owing to aneurism or carcinoma—and we can therefore almost always distinguish between an initial stage of incomplete paralysis—now more pronounced in one muscle,

[1] This example has been chosen only for the sake of an illustration. It will be shown hereafter that, so far as I know, no such case has thus far been reported.

[2] Cyclopædia (English edition), vol. vii, p. 948.

again in another,—and the stage of total paralysis." Similar views are held, I believe, by the majority of laryngologists, whilst the question has not met with any special consideration at the hands of the medical profession at large.

It would seem, then, that the earlier symptoms in cases of incomplete, slowly progressing paralysis entirely depended upon the question, *which* fibres have been accidentally first attacked by the pathological process occasioning the paralysis, and the natural conclusion would be that we sometimes should expect early lesion of the abductors, at others of the adductors.

This is, I believe, the general point of view accepted at the present time by laryngologists, and from this point of view it is easily understood, why those who have met with cases of *bilateral* paralysis of the abductor muscles, the cause of which was to be traced not to some *local* or *myopathic*, but to some *central* or nerve-*trunk* lesion, should have looked upon their cases as mere pathological curiosities, and should have tried to explain by more or less ingenious hypotheses, why in their individual case the pathological process, although acting upon the whole of the nerve, should have affected the abductor filaments only.

Now I do not wish to augment the number of these hypotheses by the addition of a new one of my own, but simply to state distinctly and separately once more the following *fact*, previously proclaimed by Morell Mackenzie and myself:

"*The occurrence of an isolated paralysis of the abductor filaments of the recurrent nerve in cases in which the roots or trunks of the spinal accessory, pneumogastric, and recurrent nerves are injured or diseased, is not an isolated pathological curiosity. There is a distinct proclivity of the abductor fibres to become affected, in such cases, either at an earlier period than the adductor fibres, or even exclusively.*"

A statement like this can only be proved in the following way: I must bring forward a comparatively large number of clinical observations and *post-mortem* examinations, showing that isolated paralysis of the posterior crico-arytænoid muscles was the result of disease or injury to the centres

and nerve-trunks, or that at any rate the paralysis of these muscles was earlier, and respectively more developed than that of their antagonists, and this number must not be compensated for by an equal or approximatively equal number of observations proving the occurrence of a primary affection of the adductor fibres under similar circumstances.

I proceed to the first part of my proof and quote some cases belonging to the first category.

CASE 1 (Gerhardt[1]). Chronic *disease of the brain* of doubtful nature. The left vocal cord immovable in the median line.

CASE 2 (Gerhardt[2]). Probably *encephalitic process* in the left half of the brain. Right vocal cord immovable in the median line.

CASE 3 (Gerhardt[3]). *Tuberculosis pulmonum.* Incomplete paralysis of the left posterior crico-arytænoid muscle. *Post-mortem* examination : left pneumogastric nerve completely imbedded in thickened connective tissue and bent backward by some swollen lymphatic glands. The corresponding recurrent nerve intimately connected with a melanotic lymphatic gland. The left posterior crico-arytænoid muscle in a state of fatty degeneration and atrophy. The adductors on the same side also degenerated, but not to such a degree as the abductor. Brain and medulla oblongata healthy.

CASE 4 (Mackenzie[4]). *Paralysis and atrophy of the abductor of the left vocal cord, caused by pressure of a malignant tumor of the thyroid gland on the left recurrent nerve. Duration six years* (!) Left vocal cord fixed in median line. *Post-mortem* examination : Cancerous tumor two inches in breadth, reaching from the arch of the aorta to the cricoid cartilage, which had "*completely incorporated* the left recurrent nerve just where it passes up beneath the upper border of the arch of the aorta. The left crico-arytænoideus posticus was completely atrophied, only a few pale thin fibres could be seen at its lower and inner part, whilst it fellow was large and well nourished."

CASE 5 (Mackenzie[5]). *Paralysis of the abductor of the left vocal cord caused by aneurism of the arch of the aorta pressing on the left recurrent nerve.* Left vocal cord fixed near the median

[1] *L. c.*, p. 307, case x.
[2] *L. c.*, p. 309, case xii.
[3] *L. c.*, p. 310, case xiii., and postscript, p. 318.
[4] "Hoarseness, Loss of Voice," etc. Case xxix, p. 39.
[5] *Ibidem.* Case xx, p. 41.

line. *Post-mortem* examination: Aneurism of the arch of the aorta. "The left recurrent nerve was traced from its origin from the vagus round the arch of the aorta, as far as the sac of the aneurism, with which it became incorporated and could not be followed further." The left posterior crico-arytænoid muscle was completely atrophied, only a very few thin, pale fibres being apparent.

CASE 6 (Semon[1]). Boy, æt. 15. History of *fit and unconsciousness* 7 years previously, after which he stammered for a considerable time. Voice has ever since remained hoarse. Left vocal cord immovable in the median line. After protracted electric treatment slight improvement in mobility.

CASE 7 (Semon[2]). Woman, æt. 55. *Aneurism* of the first part of the arch of the aorta, of the innominate and of the carotis communis dextra. The abductive power of the right vocal cord, which stands nearly in the median line, is much diminished, and the paresis of the right abductor became more complete during the short time the case was under observation. The patient soon gave up attending the hospital.

CASE 8 (Semon[3]). Woman, æt. 40. *Fibrous nodulated struma.* The abductive power of the left vocal cord much diminished. It stands close to the median line. Injections of tinct. iodi into the goître produce a considerable diminution in the size of the tumor, but the mobility of the left vocal cord does not improve, although later on the local applications, electricity, and subcutaneous injections of strychnia are made use of.

CASE 9 (Riegel[4]). *Chronic pneumonia, bilateral paralysis of the abductors.* Boy, æt. 6. Duration of the laryngeal symptoms, from the beginning until the death of the patient, *three* years. Result of the *post-mortem* examination: Both recurrent nerves imbedded in dense connective tissue; only the posterior crico-arytænoid muscles in a state of atrophy and fatty degeneration, the other laryngeal muscles *healthy.* Microscopic examination of the recurrent nerves shows, above the points of adhesion, atrophy and fatty degeneration of the majority of the filaments; but on both sides still a number of *normal, well-preserved* fibres is found.

CASE 10 (Penzoldt[5]). *Tertiary syphilis. Repeated apoplectic attacks. Bilateral paralysis of the abductors.* Woman, æt. 61.

[1] Unpublished. [2] Unpublished. [3] Unpublished.

[4] Berlin. Klin. Wochenschrift, 1872. Nos. 20 and 21, and 1873, No. 7.

[5] "*Deutsches Archiv für Klin. Medizin,*" Bd. xiii, p. 107.

Duration of the disease, 1½ months. Death. Result of *post-mortem* examination: *Roots of both pneumogastric and accessory nerves strangely thin and in a state of gray discoloration.* (Similar changes in other cerebral nerves.) *Both posterior crico-arytænoid muscles pale, brownish-red.* The left pneumogastric and recurrent nerves somewhat *slender and of a grayish color.* Microscopic examination of these nerves shows the majority of the primitive fibres as normal, but between them there are filaments which are distinctly broader than normal, and the neurilemma of which is in a state of distinct fatty degeneration. The interstitial connective tissue is augmented and contains a great quantity of fat corpuscles.

CASE 11 (Paul Koch[1]). *Compression of both recurrent nerves by enlarged bronchial glands. Bilateral paralysis of the abductors.* Girl, æt. 24. Diagnosis *per exclusionem.*

CASE 12 (Mackenzie[2]). *General paralytic symptoms. Bilateral paralysis of the abductors.* Man, æt. 44. General paralysis affecting both extremities 16 months before admission. Bilateral paralysis of the posterior crico-arytænoid muscles. No change in the laryngeal muscles during the three months he was under observation.

CASE 13 (Mackenzie[3]). *Cerebro-spinal symptoms. Bilateral paralysis of the abductors.* Repeated epileptic attacks, involuntary motions, and oozing away of the urine. Nearly complete immobility of the vocal cords in the phonatory position. Duration of the laryngeal symptoms not distinctly ascertained. Death. No *post-mortem* examination.

CASE 14 (Mackenzie[4]). *Syphilitic disease of the brain. Bilateral paralysis of the abductors.* Manifold evidence of central disturbance. Seizures, nausea, vomiting, pains, and loss of coördinative muscular power in legs, loss of eyesight, vertigo. Partial paralysis of the posterior crico-arytænoid muscles. Fifteen years previously primary venereal sore, followed by eruption. Iodide of potash speedily improved all the symptoms.

CASE 15 (Mackenzie[5]). *Imperfect paralysis of the right leg. Bilateral paralysis of the abductors.* Dyspnœa for several years. Sudden death. No *post-mortem* examination allowed.

CASE 16 (Mackenzie[6]). *Pressure on both recurrent nerves by aneurisms. Bilateral paralysis of the abductors.* Man, æt. 51.

[1] "*Annales des Maladies de l'Oreille et du Larynx*," 1878, Nos. 6 and 7.

[2] "Diseases of the Throat and Nose," vol. 1, page 428.

[3] *Ibidem*, p. 429.

[4] *Ibidem*, p. 429.

[5] *Ibidem*, p. 429.

[6] *Ibidem*, p. 443.

Death. Result of *post-mortem* examination: "One very large aneurism, commencing in the ascending aorta and involving the innominate and right subclavian artery, pressed, at its upper and outer part, on the right recurrent nerve and slightly on the right pneumogastric nerve. The second smaller aneurism involved the under and posterior surface of the descending portion of the arch of the aorta, and slightly pressed on the left recurrent nerve." Both posterior crico-arytænoid muscles in a state of atrophy and fatty degeneration. The other laryngeal muscles healthy.

CASE 17 (Mackenzie[1]). *Pressure on both recurrent nerves by cancer of the œsophagus. Bilateral paralysis of the abductors.* Man, æt. 67. Death. *Post-mortem* examination: Both recurrent nerves passed into a cancerous growth, originating from the œsophagus. Their exit from this mass could not be traced. The abductor muscles were found to be greatly reduced in size and presented signs of fatty degeneration. The other muscles of the larynx were healthy with the exception of the left thyro-arytænoid muscle, which showed signs of molecular transformation.

CASE 18 (Mackenzie[2]). *Pressure on both recurrent nerves by an enlarged thyroid gland. Bilateral paralysis of the abductors.* Boy, æt. 15. Duration of the laryngeal symptoms, four months. The treatment of the bronchocele resulted not only in a cure of this affection, but also in restoration of the function of the paralyzed abductor muscles.

CASE 19 (Mackenzie[3] and Semon[4]). *Pressure of an aneurism on both recurrents, and compression of the nerves by dense connective tissue. Bilateral paralysis of the abductors.* Man, æt. 60. Duration of the laryngeal symptoms, till death, six months. *Post-mortem* examination: Hypertrophy of the heart, aneurismal dilatation of the first part of the aorta, chronic pneumonia, and enlargement of the bronchial glands, which were enveloped in abundant firm connective tissue which compressed both recurrent nerves. Atrophy of both abductors; the other laryngeal muscles were apparently healthy.

CASE 20 (Semon[5]). *Aneurism of the arch of the aorta pressing on both recurrent nerves. Incomplete but progressive paralysis of the abductors.* Man, æt. 51. Only complaint dyspnœa and pe-

[1] *Ibid.* p. 443. [2] *Ibid.*, p. 444. [3] *Ibid.*, p. 444.

[4] "Transactions of the Clinical Society," vol. xi, 1878, p. 149.

[5] Alluded to in "German edition of Mackenzie's work," foot-notes, pp. 587 and 720, and by Dr. W. M. Ord, in "St. Thomas' Hospital Reports," vol. x, 1880, p. 131.

culiar alteration of voice beginning three months before he came under observation. Incomplete bilateral paralysis of the abductors, more marked on the left than on the right side, progressing, during the two months which elapsed before the death of the patient took place, *almost daily under my eyes.* At the time of the patient's death the left vocal cord remained immovable in the median line, while the right still moved *slightly* outward. [At the same time the level of the left vocal cord was *lower* than that of the right, and its inner border slightly excavated.] Death from rupture of the aneurism into the œsophagus. *Post-mortem* examination: Large, curiously formed aneurism of the upper and posterior part of the arch of the aorta, beginning at the commencement of the convexity formed by the vessel when passing from the ascending into the transverse part, and extending over two-thirds of the transverse part. The left recurrent nerve is firmly implicated for a considerable distance by the wall of the aneurism. It does not, however, to the naked eye[1] seem to be atrophied in its parts situated above the aneurism, but, on the contrary, somewhat swollen and congested. The right recurrent nerve is pressed upon at the point where it is given off by the right pneumogastric nerve, and in the very beginning of its course by that part of the aneurism projecting to the right. The nerve is apparently not changed. The left posterior crico-arytænoid muscle is so completely atrophied that but a few thin, pale, and yellowish fibres are seen at its inner insertion; the right is also atrophied, but to a considerably less degree. The other laryngeal muscles are apparently normal, with the exception of the left crico-thyroid muscle, which is considerably discolored and atrophied. The left superior laryngeal nerve is intact.[2]

Case 21 (Semon[3]). *Disseminated cerebro-spinal sclerosis. Bilateral paralysis of the abductors.* Gait of the character of locomotor ataxy; numbness of (now) both legs; incontinence of urine; slight left facial paralysis; slight nystagmus; inequality of the pupils,—of late; numbness of the mucous membrane of the upper lip. The laryngeal paralysis, *which has remained unchanged ever since the beginning of the observation, i. e., 2¾ years ago,* preceded *all* the other symptoms for *nearly two years!*

[1] Unfortunately no microscopical examination was made.

[2] Was, in this case, the motor innervation of this muscle derived from the recurrent nerve instead of the superior laryngeal? (v. Ziemssen, *l. c.*, German edition, p. 445.)

[3] "Transactions of the Clin. Soc.," 1878, vol. xi, p. 146.

These two facts, viz., the permanence of the *bilateral* paralysis of the *abductors* only, and the *long interval* between the appearance of this and any of the other symptoms, made me, when I first published this case, nearly three years ago, disinclined to believe in a *causal* connection between the laryngeal and the other symptoms.[1]

But the case has ever since been before my mind, and has, in fact, led me to the considerations of which the present paper is the outcome. Having convinced myself that *bilateral* paralysis of the abductors *only* might be the result of a central lesion affecting the roots of the accessory and pneumogastric nerves (Cases 10, 12, 13, 14), and that a lesion affecting the roots or trunks of these nerves and of the recurrent nerve, might result in such an *isolated* paralysis, which remains stationary *for a long time* and does not necessarily lead to retrogressive changes of the other laryngeal muscles, I cannot refrain longer from admitting that this is *most probably* a very unusual case of multiple cerebro-spinal sclerosis, in which the first and for a long time the only symptom was bilateral paralysis of the abductors.[2]

CASE 22 (L. Weber[3]). *Bilateral paralysis of the abductors after typhoid fever.* Tracheotomy. 2½ months later, pneumonia and nephritis. Death. *Post-mortem* examination: Besides other changes in different parts of the body, tracheitis and purulent peri-tracheitis were noticed. *Both recurrent nerves were found to have been embedded entirely in this purulent infiltration.* The nerves did not show any considerable changes to the naked eye, with the exception of appearing somewhat flattened; microscopically, the only change to be detected was the *diminished volume of some of the axe-cylinders.* The abductor muscles themselves appeared perfectly healthy to the naked eye and under the microscope.

[1] See *The Lancet*, vol. i, 1878, p. 630.

[2] Whilst correcting the proof sheets of this paper, I find that Dr. Hering, of Warsaw, related at the International Congress of Laryngology, held last year at Milan, a case of paralysis of the posterior crico-arytænoid muscles, "followed nine months afterward by symptoms of labio-glosso-pharyngeal paralysis of bulbar origin, which there was every reason to believe was due to syphilis." (These ARCHIVES, vol. i, 1880, p. 388.) The shortness of the communication does not, of course, permit of any definite conclusions being drawn; but even from this short report the case appears to belong to the category under consideration, and to be very similar to the one just referred to.

[3] *Berl. Klin. Wochenschrift*, Nro. 29, 1880, p. 412.

We have here then a considerable number of clinical observations, many of them authenticated by the discovery on the *post-mortem* table of anatomical changes corresponding to the symptoms observed during life, and proving that pathological processes, implicating the nerve-centres or the nerve-trunks themselves, are frequently manifested by actual changes in the abductor fibres and muscles, either exclusively or better developed than in the antagonistic muscles. I need not say that the above list by no means claims to be complete. It seemed to me that it was only necessary to show that isolated paralysis of the abductors in consequence of causes affecting centres or trunks was *not* an accidental curiosity, but occurred comparatively *often*. I am sure that it would have been easy to augment considerably the above list,—especially as far as *unilateral* paralysis of a posterior crico-arytænoid muscle from central or nerve-trunk lesions was concerned,[1]—had I made an extensive search through the literature of laryngeal paralysis. But I considered this the less necessary, because the number of 22 cases becomes important and considerable, if compared with the number of cases in which a central or trunk lesion of the nerves under consideration was found to have led to isolated paralysis of the *adductors*.

For it must not be forgotten that I have thus far only fulfilled *one* part of my task: I have shown that in many cases disease of the centres or pressure upon the trunks *can* lead to isolated paralysis of the *abductors*. The question now is: Is the number of these cases covered by a similar number, in which, under the same circumstances, the *adductors* only became affected?

The answer to this question is simple and will, no doubt, be surprising to many readers of my paper:

Not only have I *never* seen such a case, but in the whole range of laryngeal literature, which is known to me, I have been unable to find a SINGLE case, in which primary organic[2]

[1] I remember in my own hospital practice, several, at least three more cases of aneurism of the aorta with *phonatory* position of the left cord, of which I have no notes at hand.

[2] I purposely say "organic" disease of the brain, or nerve-trunks, because it is a most remarkable fact, *that the so-called "functional" neuroses show, on the*

disease of the brain or of the nerve-trunks was proved by clinical observation or the result of the *post-mortem* examination to have been the cause of *isolated* paralysis of the *adductors*![1]

I must say that I was not a little astonished, when I reached this result. The laryngeal literature, however, is already so large that it is not impossible that a few cases of this sort might have been described of which I have no knowledge;[2] but even if this were so, I think that I have proved the proclivity of the abductor fibres of the recurrent nerve to become affected sooner than the adductor fibres, or even exclusively, in cases of undoubted central or peripheral injury or disease of the roots or trunks of the pneumogastric, spinal accessory, or recurrent nerves.

The next question—if the fact has been established—would naturally be as to the *cause* of this curious proclivity. To this question it is very difficult, at the present, to give an answer satisfactorily explaining *all* the conditions under which an isolated paralysis of the abductor muscles can take place.

If the theory of the *uniformly central* origin of this paralysis, as supported by Bosworth, were correct, the presumption of the existence of an *independent ganglionic centre for the abductors* (as suggested by Mackenzie, myself, Bosworth, and others) would be sufficient to explain its cause. *Any* degenerative change, or, in a few cases, sudden functional disturbance taking place in the brain, under different influences, if limited to these ganglia, would clearly lead to iso-

other hand, quite as strange a predilection for affecting the AD*ductors only, as the "organic" lesions for the* AB*ductors!* Although there are a few cases of hysterical paralysis of the *ab*ductors on record (Fränkel, Guttmann, Biermer, Burow, Schreiber, Mackenzie-Semon), yet the *immense majority* of cases of "hysterical paralysis," hitherto recorded, concern the *ad*ductors (and tensors) only.

[1] To avoid all possible mistakes, I beg to state distinctly that I speak of such cases only in which isolated paralysis of the *ad*ductors could be traced to a "*primary*" lesion of the *roots or trunks* of the accessory, pneumogastric, and recurrent nerves. Such cases are not known to me. But I have no doubt that cases have occurred (or could be imagined), in which an originally local affection (such as a carcinoma) affected, in consequence of its anatomical situation, at an early period, the adductor fibres and muscles only, and, implicating in its later stages the entire recurrent nerve, led to paralysis of *all* the laryngeal muscles.

[2] I earnestly hope that this paper will serve in eliciting contributions corroborating or rectifying, as the case may be, my statements, from those who have had and have the opportunity of seeing cases in point.

lated paralysis and atrophic changes of the abductors. Of course there would still remain the question why the centres for the abductors are so often the seat of such degenerative processes, *in preference* to the common centre or to the probably-connected centres of the adductors (if it be supposed that each of those, as well, has a ganglionic centre of its own).

Several answers might be given to this question, contributing to, if not affording, a solution.

In the first place, the very fact just mentioned, viz.: the *mutual coöperation* and probably existing anatomical connection of the adductor centres, offers an explanation why they should be longer and more effectually protected against disease-producing influences, to which the unsupported and isolated centres of the abductors would earlier succumb. (See also Penzoldt, *l. c.*, p. 120.) Even if one or some of these adductor centres were diseased, it would by no means be improbable that the remaining healthy ones would act *vicariously* for them. That this suggestion is not a purely theoretical one, but that the mechanism of adduction of the vocal cords is actually endowed with a sort of *compensatory* tendency, is shown by the fact that, in paralysis of an entire recurrent nerve, with consequent immobility of the affected cord, in the cadaveric position, the contraction of the adductors on the healthy side, in very many cases, is so excessive, when phonation is attempted, that the healthy vocal cord *crosses* the median line to join its diseased fellow.

Another explanation might perhaps be found in the fact that the activity of the abductors, although, I believe, not *entirely* beyond the control of the will, is certainly much more automatic than that of the antagonists, and that, hence, perhaps, its power of resistance against disease-producing causes is less.

Finally, attention is to be drawn to the very remarkable fact, that this proclivity of the *abductor* centres to succumb to central causes of disease is quite *analogous* to the similar proclivity of the *extensor* muscles of the extremities to become sooner affected than the flexors, or even exclusively, in diseases of central origin, *e. g.*, in lead paralysis. (See also Rosenbach, *l. c.*)

But as I have shown that the theory of a *uniformly central* origin of the disease is untenable in the face of the authenticated cases of paralysis and degeneration of the muscles in consequence of disease of, or pressure upon, the nerve-*trunks*, even the existence of an independent centre would not account for the origin of this latter class of cases.

What, then, is the explanation of these cases?

There would be no difficulty, certainly, in explaining the phonatory position of the vocal cord or cords, in cases of undoubted nerve-trunk lesion, if this phenomenon were limited to a comparatively *short and early* period of the primary disease, in cases of *pressure* on the nerves. Before any pressure *disables* any fibres of a nerve, it acts in the manner of a mechanical *irritation*. Schech's beautiful experiments on animals have proved[1] the following fact:

"If the recurrent nerve or the pneumogastric nerve is irritated, the result is: a position of the vocal cords in the median line (phonatory position) in consequence of the preponderance of the adductor muscles." In other words, we have the same phenomenon as if the adductors alone were irritated.

But this explanation holds good only for the *earliest* stages of a pathological process encroaching upon the nerve-trunks. Why does the *phonatory* position of the vocal cords continue in the later stages? Why do we see during life, or find at the *post-mortem* table, evidence of retrogressive changes in the posterior crico-arytænoid muscles *only*, in cases of *long* duration (Cases 3, 4, 6, 9, 15, 21) and of undoubted *grave* implication of the nerve-trunks (Cases 3, 4, 5, 9, 10, 16, 17, 19, 20, 22)?

Under *all* circumstances, we must, in order to *understand* these cases, keep *one indispensable premise* before our minds, although we may, in the present state of our knowledge, not always be in the position of actually *proving* it, viz.: that in *all* these cases it is *impossible* that the conductivity of *all* fibres of the nerve-trunk should have been destroyed, however complete and long-existing the complication of the

[1] *L. c.*, p. 33.

nerve-trunk, in the morbid process, seemed to be! Riegel's case of involvement of both recurrent nerves, in dense connective tissue (No. 9 of the retrospect), is an excellent proof that, if the microscopic examination of the compressed nerves is made with sufficient care, it will be found, doubtless in *all* such cases, that a number of normal, well-preserved fibres still exist. And yet in his case the duration of the laryngeal disease was *three years!* It is thus conclusively shown that neither *intensity of the morbid process* nor *long duration* of the disease involve, *of necessity*, *total destruction* of the nerve-trunk, and we can fairly draw the inverse conclusion that, if we see, during life, isolated paralysis of the abductors, or find them, after death, in a state of atrophy and fatty degeneration, in cases of undoubted affection of the nerve-trunks themselves, the very limitation of the morbid changes to this one set of muscles, proves that the conductivity of the nerve *cannot* have been destroyed *in its entirety.* (See Cases 4 and 17 of the retrospect.)

Again, if this explanation be correct, we find ourselves confronted by the same question which we considered after the explanation of the cases of *central* origin, viz.: What is the *cause* of this curious proclivity of the *abductor filaments* to become affected sooner than the adductor fibres, or even exclusively, in cases of affection of the whole nerve-*trunk*?

Several hypotheses might be made and have been made to explain this peculiar proclivity, viz.:

1. That the anatomical distribution of the fibres of the recurrent nerve may be a *concentrical* one, and that the abductor fibres may be situated in the *periphery* of the nerve, *i. e.*, *most* exposed to all external injuries.

2. That there may be a specific vulnerability of the abductor filaments, or—what would amount *practically* to the same thing—that even in cases of partial disablement of the *ad*ductor filaments, the remaining healthy adductor fibres might conduct *all* the nerve-force emanating from the adductor centres, to the adductor muscles.

3. That possibly the adductors receive an increment of nerve-force from the superior laryngeal nerve.

However, all these hypotheses are open to certain ob-

jections, and none of them seems to me to offer a really satisfactory and plausible explanation. But at the same time I do not believe that an *explanation* is the thing we want at present.

In the face of the comparative novelty of the subject and of all the difficulties connected with this question, I would rather believe that the time has not yet come when we can venture to give an *explanation*, but that, before all, by numerous further contributions, the *fact* should receive further corroborative evidence, that: *There is a proclivity of the* AB*ductor fibres of the recurrent nerve to succumb to pathological influences affecting the roots and trunks of the motor nerves of the larynx.*

Quite apart from the *great intrinsic anatomical and physiological* interest of the question, its affirmative reply would entitle us to lay down the following practical rule:

Immobility of one or of both the vocal cords in the *phonatory* position—if not occasioned by mechanical impairments, or myopathic affection—invites to a consideration of *all* the possibilities which may produce paralysis of the *entire* recurrent nerve; immobility in the position of *deep inspiration*—if not occasioned by mechanical impairments, or myopathic affection—is much more likely to be due to either a *functional* or a *local* neurosis, *i. e.*, an affection produced by *local* disease of the *adductor twigs* of the recurrent nerve.