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CLINICAL LECTURE

ON

THE DIAGNOSTIC SIGNIFICANCE OF LARYNGEAL ABDUCTOR PARALYSIS.

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GENTLEMEN,—Before entering upon the subject proper of my lecture, I think it desirable to recall to your memory a few anatomical and physiological facts which are essential for the understanding of the curious and fascinating phenomenon with which we have to deal. You all remember that the motor nerve *par excellence* of the larynx is the recurrent laryngeal nerve. With the only exception of the tensor of the vocal cords, the crico-thyroid muscle, which is supplied by the external branch of the superior laryngeal nerve, it innervates all the laryngeal muscles proper, that is, the antagonistic groups of the abductor and the adductor muscles of the vocal cords. The former is represented by the posterior crico-arytenoid muscle only, the latter by the lateral crico-arytenoid, the external and internal thyro-arytenoids, and the inter-arytenoid muscle. The statements of Exner and of his pupils Mandelstamm and Weinzeig, according to which the superior laryngeal nerve takes part in the motor innervation of all the laryngeal muscles proper, though in varying degree, have never met with general acceptance, and have been strongly opposed by later observers. A warmly contested question, on the other hand, at the present time is that of the ultimate derivation of the recurrent laryngeal nerve. Since the beginning of this century this question has formed a bone of contention. Whilst a good many anatomists and pathologists considered the spinal accessory nerve the source of motor innervation of the larynx, others assigned the same rôle to the pneumogastric, and Claude Bernard was of opinion that the accessory supplied the phonatory, the vagus the respiratory, fibres of the laryngeal innervation. At last in 1873 the question seemed to be settled by the experiments of Schech according to which the spinal accessory was the only source of motor innervation of the larynx. This view held good until quite recently, when almost simultaneously various workers in different countries, notably Grabower, Grossmann, and Walter Spencer, concluded from their experiments and dissections that the spinal accessory had nothing whatever to do with the innervation of the larynx, and that the true source of the latter was the lower bundle of the vagus roots. In my own opinion the question is not definitively settled. Without in any way wishing to oppose the statements of the last named observers, there are certain clinical facts which still prevent me from giving them my unconditional adherence, and I refer to these discrepancies here only because later on we shall find that to some degree they enter into the subject of to-day's lecture. Finally I have to mention in this connection that Dr. Risien Russell has succeeded in splitting up the recurrent laryngeal nerve throughout its peripheral length into three different bundles of fibres, one of which supplies the abductor and another the adductor muscles, whilst from the third no motor effect can be produced in the larynx. We therefore now know definitely that the fibres going to the antagonistic groups of laryngeal muscles are differentiated throughout their peripheral course. Those fibres which ultimately supply the abductor of the vocal cords

are situated on the inner side of the recurrent laryngeal nerve.

We now come to the consideration of some physiological points. In many textbooks of physiology, even quite modern ones, you read that the vocal cords during respiration make very marked excursions synchronous with the respiratory movements of the thorax, so that during each inspiration the glottis very perceptibly widens, and is equally narrowed during expiration. In a series of investigations which I undertook some years ago, and which I laid before the Royal Society in 1890,¹ I have shown that this view is quite incorrect so far as the great majority of healthy adults of the human species is concerned. If you examine healthy adults accustomed to the introduction of the laryngoscope and not knowing that you are going to observe their respiration, you find in fully four-fifths of all cases examined that instead of these excursions the glottis, during both inspiration and expiration, forms an isosceles triangle, the sides of which move very little or not at all during the two phases of respiration. This triangle, which represents what is called the "common respiratory position" of the vocal cords, is very perceptibly larger than the triangle formed by the vocal cords and the posterior wall of the larynx after death, when the cords occupy the so-called "cadaveric position." The differences are so considerable, that the average width of the glottis in a healthy adult man during quiet respiration is $13\frac{1}{2}$ millimetres, whereas after death the maximum width ever seen so far is 6 mm. in a man and 5 mm. in a woman; the average in a man is 5, in a woman 4. The minimum cadaveric width of the glottis in both sexes I have ever seen is 2 millimetres. A graphic illustration of these conditions will show you at once that the difference referred to is anything but a *quantité négligeable*.

Fig. 1.—Ordinary respiratory position of vocal cords.

Fig. 2.—Cadaveric position of vocal cords.

The question naturally arises, What is this greater width of the glottis during life due to? The reply is this: The abductor muscles of the vocal cords are endowed with a special reflex tonus, by means of which the glottis during life is kept open during both phases of respiration to such a degree, that that type of respiration which we call the "ordinary," that is, the one in which we breathe with the diaphragm and intercostal

muscles only, is rendered possible. As soon as during life the glottis is narrowed to the same degree as we see it after death, we find that with every unusual muscular exertion dyspnoea begins, manifested either by very quick and shallow, or by very deep and laboured inspirations, accompanied by audible inspiratory stridor. At the same time, whilst the action of the diaphragm and the intercostal muscles thus becomes intensified, the accessory muscles of respiration also come into play. The reason of all this is simple. We know that of the whole respiratory apparatus the larynx itself is by far the narrowest part, and the calibre of the larynx again is still very much further narrowed by the fact that interpolated in it is the organ of phonation, namely, that reduplication of folds of mucous membrane which forms the vocal cords. By this interpolation of the phonatory in the narrowest part of the breathing apparatus, the space for the entry of air is, as I have shown by exact measurement,² reduced to less than one-third of its natural area. The result, if this narrowing were not counterbalanced by some compensatory arrangement of Nature, would be that no sufficient space would exist for the entrance of air into the lower air passages as soon as any extra demand were made on the breathing powers. In order to obviate this, Nature has endowed the abductor muscles of the larynx with the tonus before referred to, by means of which the glottis is kept sufficiently open for ordinary breathing. This tonus is produced by certain centripetal fibres contained mainly, but not exclusively, in the trunk of the pneumogastric nerve, which are stimulated by the interchange of gases in the lungs during the process of respiration, and act rhythmically upon certain centres in the medulla oblongata, where they are changed into tonic impulses, which travel downwards again along the fibres, which ultimately form the recurrent laryngeal nerve and keep the glottis open to a degree sufficient for ordinary respiration. These cursory anatomical and physiological observations are indispensable for an understanding of the pathological phenomenon with which we have to deal.³

I begin the pathological part of my lecture by reminding you of the following fact. If you cut any motor nerve which supplies various muscles horizontally across, all these muscles become completely paralysed, unless, of course, they are simultaneously innervated from other nerve sources. If, however, the lesion, instead of being a complete and sudden one, acts in a slow and progressive fashion, one of two possibilities may occur: either all the nerve fibres contained in that nerve may suffer simultaneously and in equal degree, in which event the stage of complete paralysis of the muscles will be preceded by a condition of paresis equally developed in all muscles supplied by the nerve in question; or, on the other hand, the pathological cause may act in an unequal degree upon the nerve fibres contained in the nerve trunk, when the paralytic changes may be more pronounced in one set of the muscles supplied by that nerve than in another.

These two considerations are necessary to understand the phenomenon with which we have to deal. Up to 1876 the general belief with regard to the recurrent laryngeal nerve corresponded to the two alternatives just sketched. That is to say, it was taught that if the nerve were totally disabled all the muscles supplied by it would, of course, be completely paralysed, whilst if the lesion were a slow and progressive one, there would be either general paresis of all the muscles supplied by the recurrent and equally developed in all those muscles, or, according to the particular circumstances of individual cases, this paresis might be more marked in certain of these muscles than in others, or it might even affect some muscles exclusively at first and be absent in others.

With regard to the first of these two alternatives there can be no doubt that if you cut the recurrent laryngeal nerve across there will be complete paralysis of all the muscles supplied by it, and the corresponding vocal cord which previously during quiet respiration stood in the position characteristic for this phase would, because the abductor tonus would be completely lost, recede directly into the position seen after death, and called the "cadaveric" position. Recently a great deal has been made of the fact that what I may call the "clinical" cadaveric position is not quite

identical with the "genuine" cadaveric position as seen after death, because in the former case the action of the tensor of the vocal cords, the crico-thyroid muscle, which is supplied by the superior laryngeal nerve, is not abolished, whilst in the latter eventually it is. Without denying that there may be a very slight difference between the two positions, I feel justified in saying that it is so insignificant that for all practical purposes it may be safely left out of consideration.

With regard to the second alternative, on the other hand, it was supposed until 1876 that in one case the fibres supplying the adductor, in another those innervating the abductor might be damaged first, and that according to this the one group of muscles might show a greater degree of paresis in one case, whilst in another their antagonist would be the more paretic one. The practical application of this would be that in a case in which the group of adductors was more attacked at first, on attempted phonation the vocal cord on the affected side would not quite come up to the median line but remain a little apart from it, whereby a certain degree of aphonia would be caused, whilst in another case in which the abductor was first paralysed, the vocal cord on the affected side would not be able to go quite so far out as its healthy fellow on attempted deep inspiration. Statements to this effect are found in many of the leading textbooks up to 1876. In the years 1876-78 I had an opportunity of observing at the Throat Hospital six cases of organic bilateral nervous affections of the vocal cords, in which beyond any doubt the disease-producing cause was such as to act either upon the whole of the centres or upon the whole of the trunks of the motor nerves of the larynx. Yet in all these cases the phenomena seen during life were those of paralysis of the abductors of the vocal cords only, and not once in these six cases did I see what I should have anticipated would occur with almost equal frequency, that is, a primary paralysis of the adductors or a more marked paresis of the adductors than of the abductors. During the same time I saw an even larger number of cases in which a similar process did not act bilaterally but upon one recurrent laryngeal or one pneumogastric only. Yet in all these cases again it was always the abductor which exhibited the first or even the only signs of palsy. This fact, as you see, went flat against the, at that time, universally accepted doctrine, that it was merely an accident in every individual case which group of fibres should be the one first attacked. It struck me that my experience was a very uncommon one, and I therefore searched through all the laryngological literature at that time existing in order to find whether other observers had also met with similar experiences. The result of that search was very surprising to myself. I did not find a single really authenticated case in which in an organic progressive lesion of the motor nerves of the larynx the adductors had been the first muscles to be affected. Unless the paralysis was complete from the beginning of the observation it was always the abductor which was the first affected muscle, and hence it appeared that the doctrine found in the books had been theoretically constructed. With a view of ascertaining this beyond doubt I wrote a paper⁴ in which I was very careful not to pronounce the fact which I had observed as an invariable law, but in which I merely spoke of a "proclivity" of the abductors of the vocal cords to become affected sooner or even exclusively in all cases in which an organic progressive lesion affected the centres or trunks of the motor laryngeal nerves, the spinal accessory, the pneumogastric, and the recurrent laryngeal nerve.

The publication of this paper⁵ was at first followed by a number of communications corroborating my statements; then came some cases which were supposed to show that the greater vulnerability of the abductors was not unexceptional. These cases could be easily disposed of as being faulty in observation, the more so as none of them was corroborated by the evidence of a *post-mortem* examination. On the other hand, I demonstrated a number of specimens before the Pathological and other London Societies, and before the Laryngological Section of the Deutsche Naturforscherversammlung, 1886, which showed that whilst during life there had been progressive organic disease affecting the whole of the centres or the trunks of the motor laryngeal nerves, there was after death either isolated atrophy and fatty

degeneration, with increase of interstitial connective tissue of the abductor muscles, or that at any rate the degenerative and atrophic changes were considerably more advanced in the abductor than in the adductor muscles supplied by the same nerve. Indeed, this part of the question may be briefly summarised as follows: Whilst there is not a single authenticated case on record in which it has been shown by *post-mortem* examination that in a slowly progressive organic lesion of the motor nerves of the larynx the adductors had been primarily or exclusively affected, we are now in possession of quite a number of well-observed cases demonstrating the opposite order of events.

Fig. 3.—Left abductor (posterior crico-arytenoid) muscle, exhibiting very advanced atrophy and degeneration with increase of interstitial connective tissue.

Fig. 4.—Right abductor (posterior crico-arytenoid) muscle, normal.

I only yesterday received a number of very interesting photographs, which I show you here, from Dr. Friedrich, of Leipzig, who had a case of recurrent laryngeal paralysis from the pressure of an aneurysm of the aorta under observation for three years, and who after the patient's death made a very thorough examination of all the muscles of the larynx on

both sides. It was the left recurrent which was affected. What you will see here (Fig. 3)^e is that there is a simply enormous amount of atrophy and degeneration, with a large amount of increase of interstitial connective tissue in the left posterior crico-arytenoid muscle; and it is very interesting to compare this with the right side (Fig. 4), which is absolutely normally preserved. In this connection I may mention at once that I later on was led to conclude from further clinical observations that of the adductors the first to show signs of giving way in these cases of slowly progressive organic paralysis is the internal thyreo-arytenoid muscle. You will find that here (Fig. 5),

Fig. 5.—Left internal thyreo-arytenoid muscle, showing considerable atrophy and degeneration, but less developed than in the left abductor muscle.

Fig. 6.—Left external thyreo-arytenoid muscle, also showing considerable atrophy and degeneration, but again less developed than in the corresponding internal thyreo-arytenoid muscle.

too, is a very considerable amount of atrophy and degeneration in the left internal thyreo-arytenoid muscle, but on comparing it with the left posterior crico-arytenoid you will see that the degeneration is infinitely more advanced in the latter muscle than in the internal thyreo-arytenoid. Finally,

you will see here in the left external thyreo-arytenoid muscle (Fig. 6) an amount of degeneration which must still be called considerable, though it is again considerably inferior to that seen in the internal thyreo-arytenoid, and much more so to that seen in the posterior crico-arytenoid muscle.

What you see here, I repeat, is by no means an isolated fact; on the contrary, in all cases of this kind in which a searching examination has been made, it has always been found that the posterior crico-arytenoid muscle was either the only one affected, or that at any rate the atrophic and degenerative changes were much more marked in it than in any of the adductor muscles supplied by the same nerve. In short, abductor paralysis in most cases merely represents the initial stages of complete paralysis of the recurrent laryngeal nerve. This, however, of course does not preclude that in some central diseases, such as *tabes dorsalis*, which preferentially single out certain nerve tracts, the affection should attack the laryngeal abductor apparatus only. Here is perhaps the occasion to make the plea that if ever such cases should come in future under your own observation, you should if possible obtain a *post-mortem* examination, and make such a thorough investigation of the individual muscles as has been done in Dr. Friedrich's case.

I have also to mention here one additional fact which is of considerable importance, and which in a curious way corroborates the fact of the greater vulnerability of the abductor fibres; that is that, whilst the abductor is the first to succumb, the adductors are the first to recover. That also has been shown in a number of very striking cases. For instance, I have described in 1891 the case of a distinguished tenor who suffered from a syphilitic tumour in his neck pressing upon the left recurrent laryngeal nerve, and completely paralysing the left vocal cord, which stood motionless in the cadaveric position. Under appropriate treatment, the tumour almost disappeared, and with its disappearance the adductor fibres of the left recurrent recovered, whilst the abductor fibres had been irretrievably damaged. The result was that the adductor fibres after their recovery got into the condition of which you will presently hear more, namely, that of paralytic contracture. The left vocal cord was in consequence of this process gradually dragged into the phonatory position, in which ever since it has remained, and the very unexpected but very pleasing outcome of this partial recovery of the nerve fibres is that the patient not merely regained his speaking voice, which had been completely lost, but also his singing powers, so that at present he again exercises his profession, being able to sing high chest as well as falsetto notes, and the voice, according to the statements of persons who have heard him sing before and after his illness, not showing any difference in character, power, and compass. Similar cases have been described by others, and a case has recently come under my own notice in which, after an unintentional division of the vagus nerve in the course of a surgical operation, the adductors after suture of the nerve recovered their function, whilst those of the abductor were permanently lost.

I already mentioned incidentally that a periodical controversy has been going on over this whole question. It is raging as violently at the present moment as ever it was, and the reason of all this is that the ultimate cause of the greater vulnerability of the abductors is still unknown.

Unfortunately not a few members of our profession doubt the very existence of a new fact, unless it be accompanied by what they consider an adequate explanation. I confess I regret such an attitude. It necessarily gives rise to the construction of wild hypotheses, which are but too likely to lead us astray from calmly and dispassionately following the true, even though slow, way to the ultimate solution of the problem; however, to conclude from the multitude of hypotheses framed to explain the fact of the greater vulnerability of the abductors, I am afraid that many people think otherwise. I forbear to enter upon or even merely enumerate all these hypotheses, none of which have met with general acceptance. Instead of this I prefer showing you that the pathological fact of which I am speaking does not stand isolated, and that on the contrary in the course of this controversy a number of other facts have been ascertained, all of which point in the same direction, namely, that there are biochemical differences

between the two antagonistic groups of laryngeal muscles. These facts are as follows:

First, if you take the larynx of an animal which has just been killed, and lay the laryngeal muscles bare, stimulate all these muscles with a weak faradic current, and watch the time during which they respond to the stimulus, you find without exception in the most different species of animals, that is, in dogs, monkeys, cats, rabbits—and it has been shown even in human beings by Jeanselme and Lermoyez, who thus examined the bodies of people who died from cholera shortly after death—that the posterior crico-arytenoid muscle, although by far the largest of the laryngeal muscles proper, loses its electric excitability long before any of the adductors. It is not a question of a few seconds or a few minutes, but so great is the difference that sometimes it amounts to half an hour, or even an hour, when the adductor muscles still respond to the stimulus, and the abductor has long ceased to be excitable.

Secondly, parallel conditions have been shown to exist during life by Dr. Risien Russell, who, on splitting up the recurrent laryngeal nerve into its individual bundles, found that the bundle supplying the abductor lost its electric excitability long before that supplying the adductors.

Thirdly, a further experiment, also of a very interesting kind, was made by Professors B. Fraenkel and Gad, of Berlin. They gradually disabled the recurrent laryngeal nerve in a living animal by putting the nerve on a freezing mixture (the animal, of course, being under an anæsthetic). It was found that the function of the vocal cord, which was lost first, always was that of abduction.

Fourthly, within the last few weeks another most interesting fact has come to light, of which one can only hope that it will prove to be a regular phenomenon, although its discoverer, Dr. Grabower of Berlin, is most anxious for it to be known that his communication is only of a preliminary nature. He examined the nerve endings of the recurrent laryngeal nerve in the various laryngeal muscles, and found that the nerve endings in the lateral crico-arytenoid and internal thyreo-arytenoid are of a much more complicated character than those seen in the posterior crico-arytenoid muscle. In the latter one usually finds the nerve endings to be of a "simple buttonlike character of an either roundish or oval form" (Fig. 7). In the adductors, on the other hand, one

Fig. 7.—Nerve endings in the abductor (posterior crico-arytenoid muscle).

sees a "flat plate (*Plattensohle*) into which the axis cylinder opens, and round the latter additional differently formed end organs, round, or more frequently oval, bud-like structures, which extend over a larger space, and on the whole represent more voluminous and firm formations." Having had the opportunity of seeing Dr. Grabower's original preparations, I can testify to the accuracy of the above description. Through Dr. Grabower's courtesy I am privileged to republish here the illustrations which accompanied his original paper in the *Archiv für Laryngologie*, and may add that the differences are even more striking in the specimens themselves than in the illustrations.

Fifthly, the following fact which, in my opinion, is the most important one, and which actually shows that there are biochemical differences between the two antagonistic groups,

was ascertained about eleven years ago by the late Dr. Franklin Hooper of Boston. He stated in a paper read before the American Laryngological Association in 1885 that stimulation of the recurrent laryngeal nerve, uncut or cut, with an electric current, which throughout was kept equally strong, resulted in adduction of the corresponding vocal cord when the narcosis—he always employed ether—was slight, whilst, when the anæsthetic was pushed to the condition of very deep narcosis, stimulation with a current of the same intensity resulted in decided abduction of the corresponding cord. This very extraordinary fact has, I am afraid, never met with the attention it deserves, although Mr. Horsley and I¹⁰ have specially pointed out its importance after having convinced ourselves by repetition of Dr. Hooper's experiments of the correctness of his statements. The fact that the result of electrical stimulation—currents of the same strength being used throughout—entirely differs according to the depth of the narcosis, not only when the entire re-

Fig. 8.—Nerve endings in the internal thyreo-arytenoid muscle.

Fig. 9.—Nerve endings in the lateral crico-arytenoid muscle.

current, but also when after its section its peripheral end is stimulated, clearly points to the conclusion that the difference cannot be due to any differentiating influence of the anæsthetic agent upon the nerve centres, but that there must be a specific peripheral and differential action on its part, through the agency of the circulation, upon either the nerve fibres, the nerve endings, or the muscular substance itself. In other words, there must be biochemical differences between the two antagonistic apparatus, which in all probability some day will explain the fact of the greater vulnerability of the abductors as compared with the adductors.

Having so fully treated the various points so far enumerated, you may well ask me whether the subject proper of this lecture was really of such importance as to deserve their consideration at such a length. This you will be able to judge in one moment, because I now come to the clinical phenomenon which has given the title to my lecture, namely, the diagnostic significance of the greater vulnerability of the abductor fibres in cases of progressive organic

paralysis of the motor nerves of the larynx. I shall, perhaps, best be able to illustrate its importance by going with you through the various phases of abductor paralysis in a concrete case. Take, for instance, the case of an aneurysm of the transverse part of the aorta pressing upon the left recurrent laryngeal nerve—a contingency which is the most fertile peripheral source of unilateral paralysis of the nerve. According to the law of the greater vulnerability of the abductor fibres, first of all the abductor tonus of the left posterior crico-arytenoid muscle will be gradually lost. This means that during quiet respiration the left vocal cord will no longer be in the position the cords ordinarily occupy, but will recede into the cadaveric position. No trace of this change will become objectively apparent during respiration, because the space left between the two cords is still fully sufficient to allow of free ingress and egress of air into and from the lower air passages; nor will the voice show any change, because, as the adductors of the left vocal cord are still acting, the left vocal cord as well as the right will on intended phonation go into the median line, and by the action of the crico-thyroids the necessary tension of the vocal cords will be effected. Thus neither during phonation nor during respiration will any objective symptom draw your attention to the fact that there is already something seriously amiss with the larynx.

This stage, however—that is, the one in which the affected vocal cord on attempted deep inspiration does not go further to the side of the larynx than to the cadaveric position—is only a transitory one. After a little while the same element comes into play which we see everywhere in the body when, of two antagonistic groups of muscles, one only is paralysed—namely, the paralytic contracture of the antagonists. We find such paralytic contracture, for instance, in traumatic paralysis, in spinal paralysis of children, in diseases of the joints, lead poisoning, non-use of certain parts on one side of the body only, etc. These contractures are due, according to Erb,¹¹ to “repeated or persistent mechanical approximation of the points of attachment of the muscles. This might be produced in different ways, and often is occasioned by the active voluntary contraction of the antagonists of the paralysed muscles, which shorten themselves with each voluntary effort, the paralysed muscles being incapable of again effecting their extension; thus the shortening becomes permanent.”

This description entirely applies to the subject now under consideration. The adductors, which shorten themselves each time phonation is intended, at first are somewhat released from this shortening by the natural elasticity of the elastic membrane of the larynx, and the cord recedes into the cadaveric position. Later on, however, as no sufficient counter-extension is effected by the paralysed abductor, they remain in the condition of permanent shortening. Slow as this process is, its final result is invariably the same; the paralysed vocal cord is drawn into the median line and remains immovable in this position (Fig. 10). Now the point

Fig. 10.—Complete abductor paralysis of left vocal cord.

to which I wish to prominently draw your attention, and which is of the greatest importance so far as the diagnostic value of this form of paralysis is concerned is this: that even when this stage has been reached there is—in pure unilateral

cases—still absolutely no sign which would draw attention to the larynx. Experience has shown that the opening of the glottis on inspiration, which is still effected by the healthy abductor of the other side, is sufficient to carry on respiration without any subjective or objective dyspnoea and without stridor being heard.¹² Should dyspnoea be present in such cases, it is much more likely to be due to a simultaneous compression of the lower air passages by the same tumour which compresses the recurrent laryngeal nerve—for example, an aneurysm or other mediastinal tumour, a goitre, malignant disease of the oesophagus, etc. But in pure cases—that is, in such in which the nerve disease is unaccompanied by such mechanical compression of the trachea or bronchi—under ordinary circumstances no respiratory symptoms of any kind are present. Again, with regard to phonation, the paralysed cord, as I have just explained, stands motionless in the middle line—that is, in the proper position for phonation. Its tension, which is necessary for the act of voice production, is mainly though not exclusively supplied by the crico-thyroid muscle, and this, as you know, is innervated by the external branch of the superior laryngeal nerve, which only in the rarest of cases of this kind is affected simultaneously with the recurrent laryngeal nerve. There is, therefore, in the large majority of cases no cause why the tension of the paralysed vocal cord should not be properly effected by the crico-thyroid, and thus the final factor necessary for normal voice production be supplied. As a matter of fact, indeed, the voice in a good many of these cases, though not in all, is perfectly normal for ordinary speaking purposes, and even singing, as in the case of the tenor I mentioned to you, may still be possible with a vocal cord immovably fixed in the middle line.

To summarise: The stage of complete abductor paralysis, which frequently extends over many months, not at all rarely over several years, may be present without a single symptom drawing your attention to the larynx.

Before I proceed further I wish to demonstrate to you the actuality of the statements just made.

[Here two patients were introduced, one a woman suffering from bulbar paralysis with bilateral paresis of the abductor muscles, the other a man afflicted with a new growth affecting the left side of the medulla and cerebellum, and suffering from complete left-sided laryngeal abductor paralysis. It was shown that in the case of the first patient dyspnoea only occurred on exertion, whilst the voice was quite normal, and that in the case of the second patient neither voice nor respiration showed any abnormality, the patient being still able to produce high chest notes.]

Having thus shown you the actuality of the fact, it remains for me to demonstrate its clinical importance. This importance arises from three circumstances: first, that the motor nerves of the larynx have so long and tortuous a course that from their medullary origin to their endings in the laryngeal muscles they are exposed to an enormous number of various pathological influences; secondly, that the laryngeal abductor paralysis caused by any of these influences may, and in a good many cases does, remain for a long time the only positive sign of these various pathological processes; thirdly, that it in no way proclaims its existence, but must be sought for, if one does not wish to miss the opportunity of making an early diagnosis in a good many of these cases. With regard to the first point, I need only remind you of the course of these motor fibres; whether they ultimately are derived from the spinal accessory or from the pneumogastric nerve, there is no doubt that, at any rate, after the juncture of these two nerves they run from their passage through the jugular foramen in the paths of the pneumogastric nerve. With this nerve they descend along the root of the neck into the thorax, where the recurrent laryngeal nerves are given off. The right recurrent laryngeal, which starts about the height of the right subclavian artery, winds round that vessel and ascends close to the apex of the right lung to the posterior aspect of the trachea. The left recurrent is given off by the vagus at the level of the convexity of the arch of the aorta, winds round that part of the vessel, and ascends, like the right, in the furrow between the trachea and the oesophagus up to the larynx, in the close proximity of which both nerves are split up into the twigs which end in the various laryngeal muscles.

In this long course the motor laryngeal fibres are exposed to the following bulbar and bulbo-spinal as well as peripheral diseases:

I. BULBAR AND BULBO-SPINAL AFFECTIONS.

1. Hæmorrhage and softening.
2. Syphilitic processes.
3. Tumours.
4. Diphtheria.
5. Progressive bulbar paralysis.
6. That curious form of systemic central nervous disease first described by Hughlings Jackson and Morell Mackenzie, in which one-half of the tongue, the corresponding half of the palate, the corresponding vocal cord, and, in a number of cases, the corresponding trapezius and sterno-mastoid muscles are affected.
7. Amyotrophic lateral sclerosis.
8. Disseminated cerebro-spinal sclerosis.
9. Syringomyelia.
10. Tabes dorsalis.

II. PERIPHERAL AFFECTIONS.

1. Acute rheumatic influences.
2. Catarrhal neuritis.
3. Toxic influences (lead, arsenic, etc.).
4. Tumours in the posterior cavity of the skull or in the foramen lacrum or foramen jugulare.
5. Pachymeningitis.
6. Traumatism (unintentional ligation of nerves, injection of iodine into a goitre, cut throat, stabbing, injury during extirpation of goitre, etc.).
7. Tumours of neck (goitre, peritracheal glands, etc.).
8. Aneurysms of the arch of the aorta, innominate, subclavian, carotid.
9. Mediastinal tumours (malignant, tuberculous, calcification of bronchial glands, etc.).
10. Pericarditis.
11. Pleurisy.
12. Tuberculosis and pleuritic thickening of apex of right lung.
13. Chronic pulmonary affections (chronic pneumonia, anthracosis, etc.).
14. Infectious fevers (typhoid, etc.).
15. Oesophageal carcinoma.

This list, as you see, is a very formidable one; yet it is not theoretically constructed, and of each of the contingencies named examples have been published in which laryngeal abductor paralysis was a prominent if not the first symptom. This is particularly true of such affections as bulbo-spinal syphilis, progressive bulbar paralysis, tabes, goitre, aneurysms of the aorta, and cancer of the oesophagus.

But what makes the sign so important from a diagnostic point of view is that in a considerable number of all of these cases it may be, and not rarely is, for a long time not only the first but the only symptom of the serious affection with which the patient is threatened. There is a number of well-authenticated cases on record, and I myself have seen several instances in point, in which it was the casual discovery of a laryngeal abductor paralysis, and this alone, which led to the diagnosis of such serious organic diseases as I have just enumerated. Always remember, too, that the symptom does not obtrude itself on your notice, but, like optic neuritis, must be sought for on account of the, in many cases, complete absence of all objective signs and subjective complaints pointing to the larynx. Let me give you one case in point in illustration of what I have just said:

Some years ago the head master of a large public school consulted me on account of fatigue of the voice, which he had observed during the preceding term. He was apparently a hale and hearty man of about 55, and the vocal weakness was the only symptom complained of. His voice, however, when he told me his history, was perfectly natural, and there was not the least stridor or other respiratory symptom. I was therefore not a little surprised when, on laryngoscopic examination, I found the left vocal cord motionless in the phonatory position, and, as there was no local explanation, made a careful general examination. The result of this may be summarised thus: that I found not the least evidence of any central nervous disease, and that the only suspicious signs noticeable were, first, a somewhat doubtful slight dullness over the middle part of the sternum, and, secondly, the fact that the heart sounds were audible over a larger area than they normally ought to have been. At the same time they were perfectly normal in character, there was no pulsation, no thrill, no inequality of pulses, of pupils, etc. Nevertheless, on account of the laryngeal abductor paralysis, I suspected an aneurysm of the aorta, and, as the patient did not seem willing to realise that the matter was graver than he had anticipated, proposed a consultation with Dr. William M. Ord. This was accepted, with the result that Dr. Ord

shared my suspicion, although he too found no further evidence of the existence of aneurysm. Five weeks afterwards the patient, who, instead of adopting our advice—namely, to undergo Tufnell's treatment—started for a journey to Switzerland, died from rupture of an aortic aneurysm.

I need not say that this is an exceptionally drastic illustration of the value of the symptom in question; but in many other cases it is of no less importance because the issue is not so speedily fatal. Thus, quite a number of cases are now on record in which bilateral or unilateral paralysis of the abductor muscle was for a long time the first symptom of tabes dorsalis, and the rule recommended as far back as 1881 by Dr. Buzzard—namely, that in all cases of organic laryngeal paralysis of doubtful etiology the patellar reflexes ought to be examined—deserves to be warmly supported, though, as a recent observation of my own has shown, advanced abductor paralysis may be present in tabes, even though the patellar reflexes are not quite abolished.

The very multitude of causes, however, to which laryngeal abductor paralysis may be due should caution one not to commit oneself to too rash a diagnosis of its ultimate cause on a discovery of this symptom, and I think I cannot better illustrate the necessity of this advice than by quoting to you the most extraordinary diagnostic mistake which I think I ever made.¹³

Some years ago a woman, aged about 48, came to the throat department of St. Thomas's Hospital. Her only complaint was difficulty of swallowing, and she stated that she felt an obstacle about the level of the top of the sternum. The patient looked sallow and cachectic. A bougie easily passed down the œsophagus. Though the symptoms did not point to any laryngeal affection, the left vocal cord was found to be fixed immovably in the median position; otherwise the larynx, as well as all other organs, were normal. The patient's age, the apparently normal state of all other organs, the absence of a syphilitic history, led one to think of an infiltrating carcinoma of the œsophagus, which had not yet obstructed the canal itself, but had already to some extent involved the left recurrent laryngeal nerve. Iodide of potassium was prescribed without any effect. A fortnight later there occurred ptosis of the left eyelid, then paralysis of the left rectus externus, followed shortly afterwards by complete left ophthalmoplegia. Sensory troubles in the mucous membrane of the left cheek, left-sided deafness, left-sided paralysis of the facial nerve, made successively their appearance.

By this time, of course, we knew we had to deal not with a peripheral but with a central lesion on the left side, gradually involving one cerebral nerve after another. The patient was now admitted into the hospital and subjected, under the care of Dr. Bristowe, to an energetic treatment by inunction and large doses of iodide of potassium. Her condition within the next few months improved so much that, though not cured, she could leave the hospital. The left vocal cord remained all along immobile in the median position. Syphilitic pachymeningitis was diagnosed at the time. A few months later the patient, having become much worse, came back to the hospital. The paralysis had returned with greater intensity: the left vocal cord was now fixed in the cadaveric position, and the voice, which had been formerly quite normal, had become almost aphonic. Signs of incipient paralysis were found in some of the right cranial nerves. The patient was submitted to the former treatment but without success. The paralytic phenomena on the right side progressed, and after a while included diminished outward mobility of the right vocal cord. This paresis steadily increased, but before the stage of complete right abductor paralysis had been reached the patient died comatose.

The necropsy corroborated the diagnosis of syphilitic pachymeningitis. All the affected cranial nerves were found to be imbedded near their points of emergence from the cranium in a half-gelatinous, half-fibroid mass, which had partly destroyed, partly compressed them. The vagos-accessory nerves on both sides were involved.

This brings me to my concluding remarks. The multitude of possibilities which ought to occur to your minds when discovering paralysis of both and even more so of one abductor is so great that unless there be plain collateral evidence of a definite form of organic disease, you ought to look

upon that symptom as a silent storm signal, hoisted for the benefit of the physician, but not justifying you in pronouncing the death warrant of the patient. Undoubtedly a number of cases of abductor paralysis occur, in which that sign is present for a long number of years without any other symptoms ever developing. In such cases it is possible that there may be some trivial local lesion, such as an enlarged gland compressing to a certain degree the motor laryngeal nerves at any point of their long course, and causing the persistent abductor paralysis owing to the greater vulnerability of the abductor fibres. Several such cases are known to myself, and I therefore consider it my duty to warn you not to frighten the patient in whom you may discover the laryngeal symptom by hinting at all sorts of contingencies, none of which may ultimately come true. But at the same time you ought to keep a patient who presents this sign under careful periodical observation. In the great majority of cases after some months, if not years, you are likely to discover further symptoms of the fundamental disease. In the case which has given rise to the whole doctrine of the greater vulnerability of the abductor apparatus, two years elapsed between the occurrence of bilateral abductor paralysis and that of the other symptoms of tabes, to which the patient seven years afterwards succumbed. And, in conclusion, you must also keep before your minds the possibility that the fixation of a vocal cord in the middle line may be due to an ankylosis of the crico-arytenoid articulation. Such ankylosis may occur in the course of a number of the same diseases in which also unilateral or bilateral abductor paralysis may be produced, and whilst signs of previous ulcerative diseases, absolute immobility of the arytenoid cartilage in question and the corresponding vocal cord, abnormal destruction of folds of mucous membrane, and especially tumefaction at the basis of the fixed arytenoid, will favour the diagnosis of a mechanical fixation, yet all these signs may be absent and nevertheless a joint disease exist.¹⁴

It behoves, therefore, the observer who discovers fixation of one or both the vocal cords in the phonatory position to be very guarded in the diagnosis of the disease to which such fixation owes its origin. But even with every possible limitation, there can be no doubt that the sign is one of very great diagnostic significance in a number of cases of the most serious bulbo-spinal and thoracic organic diseases.

[At the conclusion of the lecture a number of patients suffering from various central organic affections who exhibited unilateral or bilateral laryngeal abductor paralysis were demonstrated.]

NOTES AND REFERENCES.

- ¹ On the Position of the Vocal Cords in Quiet Respiration in Man and on the Reflex Tonus of their Abductor Fibres, *Proc. Roy. Soc.*, vol. xlviii, 1890.
- ² *Loc. cit.*
- ³ Fuller information on the physiological aspect of the question will be found in my paper published in *Proc. Roy. Soc.*, 1890.
- ⁴ Clinical Remarks on the Proclivity, etc., *Archives of Laryngology*, vol. iii, 1881.
- ⁵ Concerning the controversy between Professor Rosenbach, of Breslau, and myself as to the priority of the discovery, I beg to refer those interested in the question to my contribution to the *Virchow Festschrift*, vol. iii, pp. 400 et seq., and to my chapter on the Nervous Affections of the Larynx in Heymann's *Handbuch*, vol. i, pp. 685 et seq.
- ⁶ Dr. Friedrich has kindly permitted the reproduction of his photographs in this lecture. From them I have selected the photographs of the left posterior crico-arytenoid, the left internal thyreo-arytenoid, and the left external thyreo-arytenoid muscles, because the progressive degree of degeneration in these three muscles, all of which are supplied by the left recurrent, can be well seen, and I have added, for comparison sake, the photograph of the right posterior crico-arytenoid muscle, which shows the normal condition in the same individual.
- ⁷ On the Culture of the Singing Voice, *Proc. Roy. Soc.*, 1891.
- ⁸ Mr. G. H. Makins, Secondary Epithelioma of Neck, etc., *Clin. Soc. Trans.*, vol. xxix, 1896, pp. 193 et seq.
- ⁹ Those particularly interested in the question will find full details on these points in my papers in the *Virchow Festschrift*, vol. iii, pp. 400 et seq., in Heymann's *Handbuch*, vol. i, p. 684 et seq., and in the *Archiv für Laryngologie*, vol. vi, Heft 3.
- ¹⁰ On an apparently Peripheral and Differential Action of Ether upon the Laryngeal Muscles, *BRITISH MEDICAL JOURNAL*, 1888.
- ¹¹ *Von Ziemssen's Cyclopaedia*, vol. ii, p. 378.
- ¹² Matters are quite different of course in the bilateral form. If the process so far sketched develops at the same time on both sides the glottis is gradually reduced not merely to the cadaveric width, but finally to a very narrow slit, so that its appearance is that of the phonatory position of the vocal cords. Long, however, before this stage has been reached increasing difficulty in breathing occurs, manifested by inspiratory stridor, which is particularly loud and sonorous whilst the patient is asleep. Expiration at the same time is perfectly free and easy, and the voice in many cases remains normal or nearly so. It is this coexistence of considerable inspiratory dyspnoea with free inspiration and normal or almost normal voice which is so characteristic that it is sometimes possible to diagnose from its presence, without the aid of the laryngoscope, bilateral paralysis of the abductors of the vocal cords. It ought to be noted that the subjective dyspnoea of the patient is the more violent the

more rapidly the laryngeal stenosis has been developed, whilst if this process has taken place slowly—as, for instance, in many cases of *tuberculosis*—it is remarkable how great an adaptability the organism shows to the altered conditions. Degrees of laryngeal stenosis are borne under such circumstances with comparative ease, which on laryngoscopic examination would seem to be almost incompatible with the continuation of life. ¹³ This case which is important from various points of view has been described by me previously, in the *Virchow Festschrift*, and in *Brain*, Parts 59-60, 1892. ¹⁴ Fuller information on this important question will be found in my paper on Mechanical Impairments of the Functions of the Crico-arytenoid Articulation, etc., *Medical Times and Gazette*, vol. ii, 1880.

ON A SECOND CASE OF REMOVAL OF A "PRESSURE POUCH" OF THE OESOPHAGUS.

By HENRY T. BUTLIN, F.R.C.S., D.C.L.,
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In the year 1890 I saw, I believe for the first time, a patient with symptoms of a "pressure pouch" of the oesophagus, and in June, 1892, I removed the pouch. At that time there was, so far as I know, only one published case of operation for the removal of a pressure pouch of the oesophagus (von Bergmann's) but I think the operation had been performed in two, if not three, other cases, once by Billroth, and once or twice by Kocher. A full account of my case and of the operation was brought before the Royal Medical and Chirurgical Society, and was published in the *Transactions of the Society* in 1893 (vol. lxxvi, p. 269). The paper is illustrated by drawings of all the pouches, which are true or apparent "pressure pouches" in the museums of all the schools of medicine in London at that time. The small number of them, my own experience, and the statements of all writers on the subject led me to regard the condition as one of very great rarity, and I concluded my paper by saying: "I can scarcely hope to perform this operation again, for the affection is so rare that it falls to the lot of but few surgeons to meet with two of these cases in the course of a long surgical life." Since that time, my experience has been so peculiar, that I am anxious to bring the question of these oesophageal pouches prominently before the profession.

CASE I.—Captain X., aged 47. Symptoms of four years' duration. Seen first in 1890. Pouch removed in 1892. Case published in the *Transactions of the Royal Medical and Chirurgical Society* (vol. lxxvi).

CASE II.—A gentleman, aged 57, was sent to me by Mr. Critchett in September, 1894. For ten years he had suffered from slight difficulty in swallowing his food, and the difficulty had very slowly increased. He found it necessary to gulp his food down, and was then often able to swallow large pieces of solid food. But, in spite of every device and care, portions of food seemed to stick in his throat, and came up unchanged many hours after they had been swallowed. He had not lost flesh, and his general health was good. I had not the opportunity of passing an instrument for him. It is more than three years since I saw him, but I have just received a letter from him telling me that his difficulty of swallowing has very slowly grown worse, so that he is often unable to swallow liquids without some of them passing into the pouch. In the morning, after an early breakfast, he often coughs or chokes up fragments of meat, undigested, which he took on the previous evening. He is able by a movement of his neck to bring up these fragments of food. In all other respects he is perfectly well. No bulging has been noticed in his neck.

CASE III.—A gentleman, aged 73, tall, and a remarkably fine man, was sent to me by Dr. Ewart (Eastbourne) in August, 1895. He was thought to be suffering from a stricture of the oesophagus, which had been diagnosed by a medical man in large practice in London. The stricture was said to be due to varicose veins in his throat, and the veins had been burnt and the uvula shortened by a specialist in throat diseases in London, who is also in large practice. My opinion was sought on the question of the desirability of dilating his stricture. The difficulty of swallowing had commenced four years previously. It consisted, not only in a difficulty of swallowing, but in the return of fragments of undigested food long after they had been taken. On hearing this I went very thoroughly into his case, and came to the conclu-

sion that his symptoms were those of a pouch, not of a stricture. I suggested to him the possibility of a successful removal of the pouch, but told Dr. Ewart that the operation ought not to be performed unless it could be reasonably proved, immediately before the operation, under an anæsthetic, that no stricture was present. I also begged that swelling of the neck should be sought for directly after the taking of food, particularly of liquid food.

In November, 1895, the patient decided to be examined under an anæsthetic, and to undergo the operation if I could assure myself of the existence of a pouch.

On November 29th Dr. Ewart came up from Eastbourne, and the operation was performed. But, immediately before it, I succeeded in passing a very large bulbous bougie down into the stomach, and in passing a curved sound into the pouch and feeling the end of it distinctly in the posterior inferior triangle on the left side. While Mr. Gill was administering the anæsthetic, I learned for the first time that both Dr. Ewart and the patient had noticed swelling of the neck after the taking of food. The demonstration of pouch was therefore perfect, and the operation was at once proceeded with. A long incision was made on the anterior border of the left sterno-mastoid, with its centre opposite the cricoid cartilage. The dissection was carried down between the trachea and oesophagus on the inner side and the great vessels on the outer side. The superior thyroid artery was tied, the omo-hyoid muscle divided, and the pouch was discovered lying behind the oesophagus, at its junction with the pharynx, and projecting a little towards the left side. It was cut away from above downwards, while the edges of the wound were brought together with fine silk sutures. The operation was precisely similar to that described in the *Transactions of the Royal Medical and Chirurgical Society*, and the only difference in the treatment was that no attempt was made to keep a tube in the oesophagus for the purpose of feeding, and a drainage tube was inserted in the wound of the neck instead of long strips of gauze.

Fig. 1.—From a specimen in the Museum of the Royal College of Surgeons, No. 2,291 D.

The pouch was so like that removed in my first case, that one could not be told from the other. They were practically identical in size, in shape, in structure.

On December 4th milk came for the first time through the wound in the neck; and for several days large quantities of the liquids he took passed out through the wound. But by December 12th this had almost ceased; and on December 20th he returned to Eastbourne, able to swallow perfectly well, and the wound healed.

CASE IV.—A gentleman, aged 65, was brought to me in December, 1895, by Dr. Morcom, of Dunstable. For more than a year he had experienced difficulty in swallowing, but he had not wasted, and his health was excellent, with the exception that he suffered a good deal from gout. He, like the other three patients, was able to swallow liquids with greater facility than solids; and, like them, he coughed up or choked up fragments of undigested food which he had taken long previously. He swallowed a little water in my consulting-room, and, although I was not able to discover any swelling in the neck, I was able to cause the return of some of the water into his mouth by pressing on the neck. In January of 1896 I saw him again, when his symptoms were unchanged. I passed a sound, which was arrested nine inches from the teeth, and could not be passed any further, although he was able to swallow tolerably large fragments of solid food.

I have just received a letter from Dr. Morcom, dated November 24th, 1897, saying that his patient is in much the