Part First.

ORIGINAL COMMUNICATIONS.

I.—THE DISORDERS OF SPEECH.

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(Concluded from page 881.)

Dysarthric and Anarthric Disturbances of Speech due to Lesions affecting the Motor Speech-Tracts—Continued.

Lesions in the Second Trophic Realm.

In the foregoing notes upon lesions in the first trophic realm of the speech-tract, I have taken the part of the tract for the vocal mechanism and the part for the oral articulative mechanism together; because they lie in juxtaposition throughout their course, and when affected by disease are almost necessarily involved together. But in now treating of the second trophic realm, it will be best for us to take the two parts of the tract separately; because each part, springing from its own root-cells in the medulla, is continued to its muscular distribution in the form of separate and distinct motor nerves, which can easily be affected by disease independently of each other. I shall first consider the lesions of the nerve trunks, and then the lesions of the medullary nuclei from which they spring; and I shall begin with the motor nerves of the Vocal Mechanism.

1. Lesions in the Second Trophic Realm of the Motor Tract for the Vocal Mechanism.—The reader will remember that the motor nerves for the larynx take their origin in the medulla from the nerve-cells of the spinal-accessory nucleus; that the root-fibres proceeding from this nucleus soon join themselves to the trunk of the vagus; and that, after joining the vagus, they are continued down in the neck as part of its trunk, until—(1), a small number of them leave the vagus to form the motor portion of the superior laryngeal nerve; and (2), the bulk of them leave the vagus at a much lower level, to constitute the great motor nerve of the larynx, viz., the recurrent laryngeal.

As to the Superior Laryngeal nerves, it will be remembered that the muscles of the larynx deriving their motor innervation from
them are the crico-thyroid muscles and the small muscles which depress the epiglottis during deglutition. According to some authorities, filaments from them are also supplied to the interarytenoid muscle, and to the lateral crico-arytenoid muscles; but this is doubted by others, and seems to be contradicted by the evidence obtained from the study of paralysis due to lesions of the recurrent nerve. The crico-thyroid muscles, undoubtedly supplied by the superior laryngeal nerves, help to fix the thyroid cartilage during phonation, and to tighten the cords during the production of high notes. They act by approximating the cricoid cartilage to the thyroid, in front. Bilateral paralysis of the superior laryngeal nerves is said to be of not uncommon occurrence as an accompaniment or a sequela of Diphtheria, and Sir Morell Mackenzie states that in such cases the chink of the glottis, in phonation, instead of being straight, is bent in zigzag or sinuous fashion, owing to the undue slackening of the cords. The patient, it would appear, may be either voiceless or merely husky. But the chief symptom of paralysis of the superior laryngeal nerve is paralysis of sensation within the larynx. It will be remembered that, besides containing the motor fibres above mentioned, the superior laryngeal is also the sensory nerve for the larynx. Within the nerve, and within the trunk of the vagus, its sensory fibres course along with the motor; but in the medulla the two sets of fibres are separated from each other, the sensory fibres being connected with the nucleus of the vagus, and the motor with the nucleus of the spinal-accessory. It will be readily understood that the sensory and motor symptoms characteristic of paralysis of the superior laryngeal nerve may be produced either by a lesion of the nerve itself or by a lesion of the trunk of the vagus above the point at which the nerve branches from it.

The Recurrent Laryngeal nerve is, of course, the great motor nerve of the larynx. With the exception of the crico-thyroid muscles above mentioned, it supplies all the muscles which act upon the vocal cords. It thus supplies both the adductors which close and the abductor which opens the glottis.

Careful observation within recent years has brought out some interesting facts about the two sets of fibres—adductor and abductor—that go to make up the recurrent laryngeal nerve. On the one hand, it has been found that when the whole nerve is stimulated with electricity, the effect is not an equally balanced contraction of adductor and abductor muscles, resulting in absence of movement in either one direction or the other, but a movement of adduction, which closes the glottis. And, on the other hand, it has been found that when the whole nerve is gradually compressed from without by a tumour, the result is not a slowly increasing paresis equally marked in adductors and abductor, but a paresis which shows itself first in the abductor movement, and goes on to paralysis of that movement before there is any distinct paresis in the
movement of adduction. How are these two facts to be explained? There has been much controversy about their explanation, and it would take a great deal of space to make a statement of all the hypotheses that have been advanced. I shall content myself with making a brief note of two different explanations, which are each supported by high authority.

Dr Gowers\(^1\) holds that there may be no inherent difference in the properties of the two sets of fibres, and that both of the phenomena above noted may be explained if the bulk and strength of the adductor muscles are—as they seem to be—greater than those of the abductor; or if, as he thinks is the case, the weaker abductor acts at a disadvantage as compared with its opponents, owing to the nature of its attachment to the arytenoid cartilages, and the angle at which it pulls. If we grant that, in the muscular and mechanical arrangements within the larynx, it is thus provided that adduction has a distinct preponderance of power over abduction, then we can understand how it is that stimulation of the whole nerve trunk may cause closure of the glottis, owing to this preponderance of the adductor power; and how, again, when the whole nerve trunk is being slowly weakened by pressure, paresis may show itself first in the movement that is originally the weaker.

Dr Semon\(^2\), however, explains the phenomena in a very different manner. He holds that, in the two sets of fibres composing the nerve trunk, there are inherent physiological differences. To the adductor fibres, he attributes a greater inherent excitability; and to this, he thinks, is due the closure of the glottis which occurs when the whole trunk is stimulated. To the abductor fibres, on the other hand, he attributes a greater vulnerability; and in this he finds the explanation of the fact that paralysis of abduction precedes paralysis of adduction, when the whole trunk is being slowly disabled by pressure from without. If, hereafter, it should be thoroughly established that, in these cases of pressure on the nerve, there is distinctly greater wasting in the abductor muscle than in the adductors, then Dr Semon’s explanation must be accepted; but if, on the other hand, the degree of wasting is equal in abductor and adductors, then Dr Gowers’s explanation will have the advantage.

A third explanation of the early appearance of paresis of abduction in these cases of pressure on the nerve trunk was offered by Sir Morell Mackenzie, in his work on Diseases of the Larynx. He suggested that, in the trunk of the nerve, the abductor fibres might be arranged peripherally in the outer zone of the nerve-trunk; whereas the adductor fibres might be collected in the centre. This would account, he thought, for the earlier

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\(^1\) Diseases of the Nervous System, vol. ii. p. 262.

\(^2\) "Geschichte der Lehre von den motorischen Kehlkopflähmungen," a contribution to Virchow’s Festschrift, vol. iii. p. 407.
appearance of abductor paresis. But experimental stimulation of
the nerve fibres in the trunk has not confirmed this suggestion.
Dr Risien Russell, especially, has made a series of conclusive
experiments upon the nerves of living animals. Dissecting out
the nerve bundles from each other, and stimulating them indi-
vidually with electricity, he has been able to distinguish the
adductor from the abductor bundles, and has found them not to
be arranged as suggested by Mackenzie, but to be mixed up
indifferently throughout the thickness of the trunk.

Let us now, from a more strictly clinical and practical point of
view, consider, for a moment, the effects of pressure upon the
trunk of the recurrent nerve. As a typical example, let us take
a case of Aneurism of the Arch of the Aorta, with pressure upon
the left recurrent nerve. In the course of such a case, there is
apparently always a first stage, during which the paralysis betrays
itself by no symptom whatever, except, it may be, a very slight
alteration of the voice. This is the stage at which, as yet, there is
only paralysis of the abductor muscle, without paralysis of the
adductors.

If the patient be examined with the laryngoscope at
this stage, the left cord is found fixed near the median line—in
the position it ought, normally, to occupy only during phona-
tion. It remains there even when, on the patient taking a long
breath, the opposite cord is abducted to its full extent. It seems
to be the tone of the unopposed adductor muscles that thus
keeps the cord of the affected side fixed permanently in the position for
phonation. In this stage, the voice is normal, or only very
slightly altered; and there is no difficulty of breathing, because
the other "gate of the glottis" is open during respiration, and can
be swung open to the fullest extent when a long breath is taken.
In fact, this stage of recurrent paralysis can only be diagnosed
with the aid of the laryngoscope.

With continued and increasing pressure upon the nerve trunk,
there is, by-and-by, developed the second stage, characterized by
the addition of adductor paralysis to the already existing para-
lysis of abduction. In this stage, the cord of the affected
side is no longer stretched near the median line—in the position
for phonation,—as it was in the first stage. The tone of the
adductor muscles which kept it there being now lost, the cord
falls back from the middle line into the "cadaveric position"—a
position midway between that of adduction and that of abduction;
—and there it lies immobile, both during attempted phonation
and during deep inspiration. When the patient attempts to
phonate, the cord of the opposite side is strongly adducted; and,
in its effort to meet its paralyzed neighbour, it even crosses the
middle line, so as to leave but a narrow interval between itself
and the paralyzed cord. The interval is sufficiently narrow to
enable the patient to whisper; and, in the whisper, there is

1 Proceedings of the Royal Society, vol. li. p. 102.
generally a feeble vocal element due to the vocal vibration of the sound cord. The symptoms and the laryngoscopic appearances of this, the second stage of recurrent paralysis due to aneurism, are familiar to every physician; but the first stage is not so familiarly known, as there may be no symptoms betraying its presence, and as it can only be diagnosed with the aid of the laryngoscope.

Unilateral paralysis of the recurrent nerve, such as is thus so often due to aneurism of the aorta, may also be due to other causes, and occur in connexion either with the left or with the right recurrent. Thus the pressure from a tumour in the mediastinum, or from a glandular tumour in the neck, or from the enlarged lateral lobe of a goitrous thyroid body, or, again, the involvement of the nerve in cancerous disease of the oesophagus, or its compression, on the right side, by the newly-formed connective tissue of a pleuritic thickening at the apex of the right lung,—all of these conditions may produce paralysis of the recurrent nerve on one or other side; and the paralysis may present a first and a second stage, as when it is due to the pressure of an aortic aneurism.

Further, it should be remembered that recurrent paralysis is sometimes due to pressure upon the trunk of the vagus, at some point above the level at which the recurrent nerve is given off. If the point pressed against be so high in the neck as to be above even the point at which the superior laryngeal is given off, then, along with recurrent paralysis, there will be also paralysis of the superior laryngeal nerve, with its characteristic anaesthesia of the larynx.

In some rare cases, Bilateral Paralysis of the recurrent nerves has been found to be due to pressure, either upon the two recurrent nerves or upon the two pneumogastrics. This bilateral paralysis presents the same two stages in its progress as have already been described as occurring in unilateral recurrent paralysis. First, in the stage of simple abductor paralysis, the cords lie in the position of adduction, close to each other in the middle line; and then, in the stage of combined abductor and adductor paralysis, the cords have retired from the middle line, and lie in the cadaveric position at some distance from each other. It will be readily understood that in the first stage there is great danger of suffocation. The cords lie so close to each other that there results that crowing stridulous inspiration so characteristic of bilateral abductor paralysis. The noise is loudest when the patient makes any exertion, and when he is asleep; and during sleep a paroxysm of laryngeal dyspnoea may set in, and may end fatally. Tracheotomy is thus often advisable, in bilateral paralysis of the abductor. Though inspiration is noisy and difficult, expiration is easy; and the voice, in speech, may, in this stage, be natural. When, in the second stage, the cords retire into the cadaveric position, breathing becomes easy, all stridor
disappearing; but, in this stage, the voice is lost, because the adductors are now paralyzed, as well as the abductor.

Before leaving the laryngeal nerves, I may refer, in a word, to the occasional occurrence of neuritis in one or other of their branches of distribution, and to the resulting paralysis of one or other of the individual muscles of the larynx. I have already, in the second paper of this series, made some brief reference to the loss of voice which may be caused in this way, by the paralysis of some single adductor muscle.

It still remains for us to consider the Nuclear Cells in the Medulla, from which the motor nerves of the larynx spring. As already indicated, these cells form part of the nucleus of the spinal-accessory division of the eighth cranial nerve. It has been asserted that some of them are also contained within the nucleus of the vagus, but this opinion still requires confirmation.

There are many lesions, coarse and fine, which may affect the motor and sensory nuclei in the medulla; but, as some of these affect, indifferently, the cells of either the oral articulative mechanism or the vocal mechanism, or affect both sets of cells simultaneously, and as others, such as the lesion in glosso-labio-laryngeal paralysis, are primarily developed in the cells of the oral articulative mechanism, I think it will be well to reserve any enumeration of them until we come to look at the nuclear origins of the nerves for the oral articulative mechanism. But there is one lesion which is of special interest in the present connexion, as, on invading the nuclei of the medulla, it seems to have a special proclivity to affect the motor cells of the vocal mechanism,—I mean the spread of degeneration into the medulla in cases of tabes dorsalis (locomotor ataxy).

It is now well ascertained that, in the course of ordinary tabes dorsalis, two forms of laryngeal complication may be developed, in consequence of this invasion of the spinal-accessory nucleus. These are—(1), Temporary paroxysms of laryngeal dyspnœa ("laryngeal crises"), which by some authorities are attributed to temporary spasm of the adductor muscles, and by others to temporary paralysis of the abductor; (2), permanent paralysis of laryngeal muscles. This paralysis seems always to affect the abductor in the first instance. It may be confined to this throughout, or, in course of time, may involve also the adductors.

Often, especially at first, the paralysis is unilateral; and then, so long as it is purely a unilateral abductor paralysis, it can only be detected with the laryngoscope,—there being no symptoms, such as loss of voice, or laryngeal dyspnœa. When, however, the adductors are also affected, and the cord has fallen back from the median into the cadaveric position, then there is loss of voice, as in the advanced stage of unilateral recurrent paralysis in cases of aneurism.

If there is bilateral paralysis of the abductor muscle alone—as,
in tabes dorsalis, there often is,—then the patient suffers from the marked and dangerous laryngeal dyspnoea which has been already noted as characteristic of that condition. This will disappear, but the voice will be lost, if the paralysis ultimately involves also the adductors.

These laryngeal complications of tabes dorsalis must be commoner than is usually imagined. Dr Semon says that in the first twelve cases of tabes examined by him in the "National Hospital for Epilepsy and Paralysis," he found no fewer than seven to be affected either with unilateral paralysis of the abductor, or with unilateral paralysis of both the abductor and the adductors, or with severe bilateral paresis of the abductor. But he adds that in the next fifty or sixty cases not a single case presented any symptom of laryngeal paralysis.¹

Seeing that in so many of these cases the abductor is affected alone, or is affected first, before the adductors are also involved, Dr Semon has been naturally led to the conclusion that in the medullary nuclei of the laryngeal nerves there is the same comparative vulnerability in the abductor nerve-cells that he believes to exist in the adductor nerve-fibres of the recurrent nerve. And this conclusion would be irresistible, if it were proved that all degenerative conditions invading the medullary nuclei singled out the abductor cells in similar fashion. But this is not proved. The leading degenerative disease of the medullary nuclei is Progressive Bulbar Paralysis (glosso-labio-laryngeal paralysis); and when that disease, in its latter stage, spreads from the nuclear cells of the oral articulative mechanism into those of the vocal mechanism, it attacks first the adductor cells,—not the cells for abduction. It does not, however, often cause complete paralysis of the adductors. "The laryngeal palsy," says Dr Gowers, "rarely becomes complete, and it is still rarer for the power of abduction to be specially lost, common as abductor palsy is in some other forms of central degeneration."

Some explanation other than that offered by Dr Semon may yet be found—possibly one connected with the anatomical position of the abductor cells,—to account for the frequent occurrence of abductor paralysis as a complication of tabes dorsalis.

In dealing with any case of laryngeal paresis or paralysis, a very important question for us to determine is, whether the paralysis is of functional or of organic origin. As recent investigation has thrown considerable light on this question, perhaps it may be well for us to devote here a little attention to it.

If we take first the Bilateral forms of paresis or paralysis, we may make note of the following general conclusions:—

1. That a bilateral paresis of the Adductor muscles, without any affection of the abductor, is almost always of functional origin,

¹ Op. cit., p. 444.
and is commonly due to hysteria. When it is slightly pronounced, it causes "hysterical aphonia." When it is better marked, it causes the common variety of hysterical mutism. This subject has already been fully discussed in the second paper.

We must, however, admit that in a few cases this paresis of the adductors may be of organic origin,—if it is true that when progressive bulbar paralysis invades the nuclear cells of the vocal mechanism the adductor cells are the first to be affected.

2. That a bilateral paresis or paralysis of the Abductor, without apparent affection of the adductors, is very generally of organic origin,—being most commonly the result of lesion in the bulbar nuclei, as well as being also, in rare cases, the first effect of pressure upon both recurrent nerves or both pneumogastrics.

It is believed by some authorities, however, that this paralysis may in a few cases be of functional causation. Gowers, for example, states that temporary attacks of it, attended with the characteristic dyspnœa, have been known to follow an ordinary laryngeal catarrh; and he expresses his belief that the laryngeal paroxysms of dyspnœa which occur occasionally in some cases of hysteria may sometimes be due to temporary paralysis of the abductor, and not always to spasm of the adductors, as is usually supposed.

3. That a bilateral paralysis involving both Abductor and Adductor muscles is always of organic origin. There seems to be no exception to this rule.

If we now pass to the Unilateral forms of paralysis, we can say in brief about them, that whether they affect adductors or abductor, they are practically all of organic origin.

Possibly it may yet be found that there are rare cases of Hysteria in which there is adductor paresis of one cord only. This possibility I shall discuss presently.

As to the Situation of the lesions accountable for these various forms of paralysis when they are of organic origin, we can say that in the vast majority of cases, if not in every case, it is somewhere in the second trophic realm of the motor tract. It is either in the medullary nuclei, or in the trunks of the nerves, or in their branches of distribution. Being due to lesion in the second trophic realm, these paralyses are attended with the characteristic wasting of the paralyzed muscles.

The rare exceptions to this law—that the organic lesions capable of producing paralysis in the larynx are situated in some part of the second trophic realm of the motor tract—are furnished by bilateral lesions involving the first trophic realm, in both hemispheres or in both crura; and by central lesions in the pons which disable both tracts. In such cases, as we have seen, there is pseudo-bulbar paralysis—a paralysis always bilateral;—and all
voluntary control over the larynx, as well as over the muscles of the tongue and lips, is lost.

If the motor representation of the vocal cords in the cerebral cortex is perfectly bilateral (as Semon and Horsley believe it to be), then it is not possible for any lesion in the first trophic realm—whether in one cortical centre or in its tract—to produce paralysis of one vocal cord (unilateral paralysis in the larynx); because the remaining cortical centre and its tract continue to innervate both cords. Nor, à fortiori, is it possible for any disablement there from functional causes, such as hysteria, to produce unilateral laryngeal paralysis.

But if further investigation should show that Masini is right in stating that the motor representation of the vocal cords in the cortex is not perfectly, but is only imperfectly, bilateral—just as is the motor representation of the tongue, lips, etc.,—it would then appear that it is possible for a unilateral lesion in the first trophic realm (either in one cortical centre or in its motor tract) to produce paresis of the opposite cord,—just as a lesion in the first trophic realm of one tract for the oral articulative mechanism produces a slight paresis in the opposite side of the mouth and of the tongue, as is seen in ordinary hemiplegia. And if an organic lesion on one side can thus produce a unilateral paresis in the opposite cord, so perhaps may a functional disablement on one side, due to hysteria.

As we saw in a former paper, cases of cerebral lesion in one or other of the hemispheres attended with motor paralysis in the opposite cord have already been published. It is, however, suggested by Dr Semon and others, that in these cases some other lesion, in what I have termed the second trophic realm of the tract, must have been present, though overlooked. We must wait for further evidence, before we can be quite sure which party in this controversy is right. If cases of hysterical hemiplegia should be met with in which there is loss of voice from adductor paresis of the vocal cord on the same side as the hemiplegia, they would lend support to the opinion of Masini that the motor representation of the vocal cords is only imperfectly bilateral; since they would tend to show that even a functional unilateral disturbance of the cerebral cortex is capable of producing a unilateral paresis of the opposite cord. The weight of evidence, however, at the present time, is strongly in favour of the conclusion of Semon and Horsley, that the representation of the vocal cords is perfectly bilateral, and that it is, therefore, impossible for any organic lesion or functional disturbance in the first trophic realm to produce unilateral paralysis or paresis in the larynx.

The common bilateral paresis of the adductors in hysteria is commonly believed to be due to functional disturbance of motor power, or, shall we say, the power of will, in both hemispheres.1

1 For fuller information regarding the Paralyses of the Larynx, see the chapter on the subject by Dr Gowers in his work on Diseases of the Nervous System.
2. Lesions in the Second Trophic Realm of the Motor Tract for the Oral Articulative Mechanism.—This realm begins in the motor nuclei of the medulla, and extends, in the form of various motor nerves, to the muscles of oral articulation. The nuclei from which the nerves spring are the motor nuclei of the seventh pair, the hypoglossal nuclei, and portions of the nuclei of the spinal accessory. The nerves are the portio dura of the seventh, the hypoglossal, and the nerves from the spinal-accessory nucleus which supply the soft palate and the muscular wall of the pharynx.

As I do not think it would be of much advantage to attempt here an elaborate description of the lesions of the nerve trunks, I shall content myself with reminding the reader of a few leading facts about these lesions.

First, about the portio dura of the Seventh, I need scarcely say that unilateral paralysis, due to neuritis of the nerve trunk (Bell's paralysis), is one of the commonest of all the forms of local paralysis. Though the mouth is pulled towards the opposite side, and the lips on the paralyzed side are flaccid, the articulation of the labials and the labio-dentals is only slightly interfered with. In rare cases, both nerves are paralyzed; and then the whole face is expressionless, and the lips are flaccid on both sides. Commonly, these cases are due to the pressure of a tumour at the base of the brain upon the nerve roots. But, some years ago, I had, in my wards, a case in which this bilateral paralysis had been caused by injury of the nerve trunks, at or about their exit from the stylo-mastoid foramina. In the bilateral cases, the enunciation of the labials and labio-dentals is impossible; but, as the other parts of the articulative mechanism are intact, speech is not very seriously damaged, and is always easily intelligible. It is most striking to hear and see a patient with double facial paralysis laugh: there is something so incongruous between the sad, flaccid, and immobile countenance, and the merry sounds that come, as it were, from behind it. The patient just referred to, whose double paralysis was due to injury, used sometimes to laugh heartily; and she always produced a strong impression on her hearers when she did so.

Paralysis of the Hypoglossal nerve from lesion of the nerve trunk is so rare, that I think we may pass it over. It would, of course, produce paralysis of the tongue on the same side, with very marked wasting; but would probably not interfere very seriously with articulation.

Paralysis of the fibres from the Spinal-accessory nucleus which supply the soft palate is of importance, because it is a frequent complication or sequela of diphtheria. In such cases, it is usually System, vol. ii. p. 256; or the article by Dr Semon referred to in the footnote to p. 865; or the chapter by Dr M'Bride in his work on Diseases of the Throat, Nose, and Ear, and an article by the same author in the Edinburgh Medical Journal, July 1885.
bilateral. It causes the soft palate to droop, and to remain drooping even when the patient takes a long breath or sings a high note. During deglutition, it permits the escape of fluid through the nose. In speech, it produces the peculiar and characteristic nasal snuffle. It is one of the advantages of studying the function of speech with close attention, that such an alteration of speech as is produced by the presence of even a slight nasal snuffle is at once detected by the trained ear. I can recall, at the present moment, a case in which the presence of a slight nasal snuffle in speech led to the immediate detection of a diphtheria, though the patient was making no complaint about the throat; and another case—one of suppression of urine from blocking of both ureters—in which it led to the examination of the throat, and the detection of an oedema of the soft palate and uvula,—the beginning, as it proved to be, of an acutely advancing dropsy, which caused death in a few hours, by invasion of the lungs. Many other conditions besides paralysis of the soft palate, and edema of it, may produce a nasal snuffle in speech—as, for example, perforating ulcer of the soft palate, and split palate;—and some people snuffle in speech either from habit and carelessness, or from not having learned in childhood how to manage the soft palate in speaking.

More important than the lesions of the nerve trunks, are lesions of the medullary nuclei from which they spring. Affections of these nuclei produce the type of paralysis known as Bulbar Paralysis.

There are a few cases in which this type of paralysis is suddenly or rapidly produced by such gross lesions as apoplexy, or acute inflammatory softening, in the medulla; but such cases are comparatively rare, and usually prove rapidly fatal. More common and important are those in which disease is of the degenerative and sclerotic type. Such disease slowly involves the nuclei more and more, until there is total paralysis of the muscles supplied from them. This slow degeneration of the bulbar nuclei is sometimes a complication of certain diseases of the spinal cord, such as chronic progressive poliomyelitis (progressive muscular atrophy), or tabes dorsalis, or multiple sclerosis. Sometimes it is a disease *per se*, being, throughout its course, confined to the nuclei of the medulla. It is then known as Progressive Bulbar Paralysis, or Glosso-Labio-Laryngeal Paralysis. This is the most important of the varieties of Bulbar Paralysis; and I think it will suffice for the purposes of this paper if I make a very few notes about it.

Progressive bulbar paralysis is always bilateral; and the nuclei first affected are usually those for the innervation of the tongue. Thence the disease spreads to the nuclear cells for the lips, and to those for the soft palate and the pharynx. It is only, as a rule, when all these parts have, in course of time, become totally, or almost totally, paralyzed, that the disease overflows, as it were, into the nuclei for the innervation of the larynx.
I need not, in detail, describe the well-known symptoms of progressive bulbar paralysis, but I may remind the reader that, in the advanced stage of the disease, the lower part of the face becomes expressionless as a mask, the lips being entirely paralyzed and remaining helplessly apart, so that the patient cannot prevent the dribbling of saliva from the open mouth; that the tongue not only lies paralyzed in the mouth, but generally presents a wrinkled and shrunken or shrivelled appearance, due to marked wasting of its muscular substance, and often in its wasting fibres presents fine fibrillar movements like those in the wasting muscles of a patient with progressive muscular atrophy; that the palate hangs drooping and paralyzed; and that the paralysis of the pharynx renders deglutition impossible.

It will be readily understood that as the paralysis extends from tongue to lips, and from lips to soft palate and pharynx, corresponding deteriorations of articulation appear in the patient's speech. These, however, need not be described; as the best key to them is to be obtained from the study of the Physiological Alphabet. Sometimes the lips are affected before the tongue; and then it is the labials and the labio-dentals that first suffer. When the palate becomes affected, the characteristic nasal snuffle appears. In the advanced stage of the disease, if the larynx be yet unparalyzed, the patient, though totally unable to articulate, may still, when he tries to speak, be able to emit monotonous vocal sounds of a grunting character.

As to the final invasion of the larynx by the paralysis, it seems certain that, in the great majority of cases of this disease, the first muscles affected are the adductors,—not, as in tabes dorsalis, the abductor. It is rare for the patient to exhibit that laryngeal dyspnoea which is so characteristic of bilateral abductor paralysis. Usually he first exhibits a want of explosiveness in his cough; and afterwards the voice becomes enfeebled, though it is rarely altogether lost. Anaesthesia of the mucous membrane, from involvement of the sensory nuclei, is sometimes added to the motor paralysis. Owing to the combined motor and sensory paralysis of the larynx, the air-passages are imperfectly protected from the entrance of food or fluid, if the patient attempts to swallow; and it therefore becomes necessary to feed him with the stomach-tube.

I would here make a special note regarding the loss of explosiveness in the patient's cough, which often forms the first indication that, in the spread of bulbar paralysis, the larynx is beginning to be invaded. Some years ago, I noticed it in two cases of my own; and I made a note of it in a paper on "Extra-auscultation," recently contributed to the first volume of the Edinburgh Hospital Reports. I now find, however, that Dr Gowers had already made careful note of it, in his work on Diseases of
the Nervous System. But I should like to direct attention to the explanation I have suggested for this want of explosiveness in the cough, as it is different from the explanation offered by Dr Gowers. Dr Gowers evidently holds the common opinion that in the closure of the glottis which is preliminary to the act of coughing, and which attends upon the act of straining, the glottis is closed efficiently against the exit of air by the co-aptation of the true vocal cords,—that, in short, it is closed in the same manner as it is for phonation, only with greater strength and firmness. He therefore regards the loss of explosiveness in the cough as simply a first indication of adductor paresis. But, as I have already explained in a former paper, the closure of the glottis for coughing or straining is a very different thing from its closure for phonation; because in phonation only the true cords are co-aptated, whereas in the preliminary stage of coughing and in straining the false cords are co-aptated as well as the true; and it is, I believe, the false cords, with the Ventricles of Morgagni, which, in valve-like fashion, hold in the imprisoned air during the preliminary or compressive stage of a cough. I do not know that the innervation of this important closure of the false cords has ever yet been adequately investigated. It may be that the muscular fibres which effect the movement are innervated, like those which depress the epiglottis, by the superior laryngeal nerve. But whether this be so or not, I think it highly probable that the nuclear cells for the movement are, in the course of progressive bulbar paralysis, invaded before the nerve-cells for the ordinary adductors of the larynx, and that thence results the early loss of explosiveness in the cough.

In my paper in the Edinburgh Hospital Reports I have termed the unexplosive cough above mentioned the "Bovine Cough," because an ox has no Ventricles of Morgagni or false cords, and its cough is therefore an unexplosive grunt or wheeze.

In a normal cough, there are both an explosive element, due to the action of the false cords, and a vocal element, due to the action of the true cords. In the Bovine cough, the explosive element is lost, but the vocal may be retained. In severe cases of hysterical aphonia, on the other hand, the vocal element is often lost, while the explosive is retained; though in mild cases both elements are retained. I have at present, in my wards, a severe case of this kind, in which the aphonia amounts almost to complete mutism, and has hitherto resisted treatment, even by our skilled specialists; and in this case the absence of the vocal element of the cough and retention of the explosive element are well exhibited. I think this condition of things shows that there may be adductor paresis of the true without adductor paresis of the false cords. Further, we can, each of us, in our own persons, cough at will, either—(1), in the normal way, with both elements in the cough; or (2), like the patient in the above-mentioned case.
of hysterical aphonia, with the initial explosive element, but without the accompanying vocal element; or (3), after the manner of the ox, with the accompanying vocal element, but without the initial explosive element. Does not all this show that there are two laryngeal mechanisms employed in the act of coughing, viz., that of the false cords and that of the true? It is now close upon thirty years since I first demonstrated the valvular action of the false cords and ventricles of Morgagni during the act of straining, and at the initial stage of coughing; and it is eleven years since my conclusions were confirmed by the joint investigations of Dr Lauder Brunton and Dr Cash, which I have already referred to. When will physiologists and physicians recognise that the subject is worthy of attention?

Alongside of these notes regarding the probable spread of degeneration, in the second trophic realm, from the nuclear cells of the oral articulation mechanism into the nuclear cells for the closure of the false cords, I should like to put a note reminding the reader of a point discussed in the first paper of this series, viz., the occurrence, a rather rare variety of stammering, of an overflow of energy, from the over-stimulated centres of the oral articulative mechanism into the centre for the closure of the false cords. There results, it will be remembered, when the patient attempts to speak, a closure of the "upper glottis," which gives its special feature to the "gutturo-tetanic" variety of stammering. I think it probable that this overflow occurs in the cortical cells of the first trophic realm.

In concluding this sketch as to the tract for the oral articulative mechanism, it may be well for us to ask ourselves what general conclusion it tends to lead up to regarding the diagnostic value of the dysarthric disturbances of speech that are produced by lesions of the speech-tracts.

I think, on the whole, it should teach us that in diagnosing the seat of any lesion involving the tract, either in its first or in its second realm, we should not rely exclusively, or even mainly, upon the specific speech-disturbances present. There is too much sameness in the alterations of speech, whatever may be the part of the tract involved, to warrant an exclusive reliance upon them. A thick, slurring, and more or less laborious articulation is the leading characteristic in nearly all such cases.

Yet there are varieties in the degree of the dysarthria which may sometimes help us to diagnose the seat of lesion. A total anarthria is more common in lesions of the pons or of the medulla than in lesions of the hemispheres or of the crura. A special slurring upon linguals or labial may suggest the early stage of bulbar paralysis; and a nasal snuffling may suggest a paralysis of the soft palate, perhaps of diphtheritic origin. Again, predominance of the staccato element in the speech ought to
suggest multiple sclerosis, though something of the staccato element may, as we have seen, be present in other conditions.

On the whole, however, we shall do well, in trying to diagnose the nature and locality of disease in any case of dysarthria or anarthria, not to trust too much to the specific alterations of the speech, but, whilst giving these their due weight, to arrive at our conclusions only after having taken fully into account the associated symptoms of paralysis, and all the other symptoms of the case.

My task is now finished. I began these papers by considering the nature of Stammering, a disorder which, by the disruption it exhibits in the harmonious action of the vocal and oral articulative mechanisms, is admirably fitted to illustrate the semi-independence of each of these mechanisms, and to enable us to realize the beautiful coördination with which, in normal speech, they work together. I have now finished by tracing down the motor tracts of the two mechanisms to their terminations in the executive muscles, and by noting the leading conditions of disease that may interfere with motor conduction in them. In the course of the papers, we have been led to consider many matters of great interest and importance; for it is a great subject we have been discussing,—a subject full of interest in all its relations. I hope that these papers, as a contribution to the study of the subject, will be found practically useful. There are few studies, I think, so well calculated to give material help in the diagnosis of nervous diseases as the careful study of the Disorders of Speech. If these papers should prove of practical value to others, as an aid in the prosecution of this important study, it will be to me, I need scarcely say, a lasting pleasure and satisfaction.

I beg respectfully to tender my thanks to the Editor and Publishers of the Edinburgh Medical Journal, for allowing me to publish consecutively in the Journal so many papers on this subject; and to the Staff of the Journal, for much courteous help and attention.

II.—MORISON LECTURES ON INSANITY.

By John Batty Tuke, M.D., F.R.C.P.E.

(Delivered before the Royal College of Physicians of Edinburgh.)

(Continued from page 898.)

Lecture IV.

We must now turn our attention to a very important series of symptoms produced by morbid hyperaemia and congestion of the Rolandic area,—the impairment of the health of the general