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A LECTURE

ON

SOME POINTS IN RELATION TO THE DIAGNOSTIC SIGNIFICANCE AND THERAPEUTIC INDICATIONS OF LARYNGEAL SYMPTOMS RESULTING FROM PRESSURE OF ANEURYSMS UPON THE VAGUS AND RE- CURRENT LARYNGEAL NERVES.

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It will be my endeavour to show by reference to cases of aortic and innominate aneurysm: First, that aneurysm of aorta and innominate artery may exist and give rise to laryngeal symptoms only, but in most instances, on critical physical examination, certain collateral signs may be made out sufficient to warrant one in forming a positive diagnosis, or to give rise to a very strong suspicion of an intra-thoracic tumour. Second, that in the early stage pressure may cause paroxysms of most urgent dyspnoea, accompanied by laryngeal stridor and paroxysmal cough. Third, that at a later stage paralysis occurs usually, but not always, limited to one side, characterised by phonative waste of breath and imperfect cough, but without dyspnoea, except when reflex spasm is indicated on the opposite side, or when pressure-stenosis is caused by the aneurysm. Fourth, that in certain cases tracheotomy should be performed not only to prevent impending death from asphyxia, but also as a remedial measure.

In this paper it is not my intention to consider the various causes of paralysis of the vagus or recurrent laryngeal nerves, but it may be mentioned that paralysis results from a multitude of conditions, for example, enlargement of the thyroid body or of the bronchial glands, intra-thoracic tumours, growths in the oesophagus, pleuritic adhesions at or fibroid induration of the apices of the lungs, and certain diseases of the heart which lead to displacement, such as hypertrophy or great pericardial exudation. These and other rarer lesions have come to be recognised as possible causes of unilateral or bilateral recurrent paralysis, and must therefore be held in view in forming an opinion respecting the pathology of a case. On account of the relationship of the left recurrent laryngeal nerve to the arch of the aorta, it is especially exposed to pressure by aneurysms, enlarged bronchial glands, or tumours within the mediastinum, while, on the other hand, the nerve on the right side, because of its proximity to the apex of the right lung and to the innominate artery, is more apt to be included in fibroid induration of the apex, pleuritic adhesions, or aneurysm of the innominate artery. The disturbances which may lead to paralysis cannot be considered in detail, but we must now endeavour to answer the first question proposed for discussion, namely, the fact that an aneurysm of the aorta may give rise to laryngeal symptoms only. The word "symptoms" is used in contra-distinction to "physical signs," for this reason. It is because of some particular disturbance of function that a patient seeks medical advice, and it has been found at the Out-door Department for Diseases of the Throat at the Glasgow Royal Infirmary, that patients have been sent by their medical attendants, or have come of their own accord, seeking relief from what they regarded to be a throat affection and nothing else, but which ultimately

proved to be disturbance of the voice as a consequence of impairment of the function of the muscles supplied by the recurrent laryngeal nerve.

The purpose of this paper is to represent the general and important facts, and indicate the conclusions which may be arrived at respecting what may be described as the gross characteristics of the lesion, the finer distinctions being left out of account, or only briefly alluded to, when they have a direct bearing upon the question under discussion.

With these preliminary remarks the history of the cases from which deductions have been drawn may now be given.

CASE 1. Aneurysm of Innominate Artery and Descending Aorta: Paroxysmal Dyspnoea from Unilateral Spasm of Right Vocal Cord, followed three months after by Bilateral Spasm of Adductors, and at a more remote date by Bilateral Paralysis of Adductors: Dysphagia and Laryngeal Symptoms only early Evidences of Aneurysm: Appropriate Treatment recommended but not carried out: Increase in Size of Aneurysm: Death from direct Pressure of Aneurysm upon Trachea.—J. K., aged 45, is a particularly interesting case in many respects, but especially from the fact that there was first unilateral spasm, followed two months after by bilateral spasm of the intrinsic muscles of the larynx, and at a more remote date by bilateral paralysis. The patient came first to the out-door department for diseases of the throat in December, 1885, on account of paroxysmal difficulty in breathing and a harsh, dry cough, the voice being practically unimpaired excepting during the paroxysms, and on laryngoscopic examination (which was very difficult to accomplish on account of the unusually sensitive condition of the fauces) the larynx was seen to be healthy, and the movements of cartilages and cords normal, but if the mirror was held in position for even a short time the right vocal cord would be drawn across the middle line and fixed there, and immediately thereafter the patient would suffer from a paroxysm of dyspnoea. The patient had slight difficulty in swallowing solids. The laryngeal symptoms and the difficulty in swallowing excited the suspicion of an aneurysm, or tumour, pressing upon the right recurrent laryngeal nerve, but a minute examination failed to reveal any other evidences of such diseases. The patient was, however, treated as for aneurysm. He was again seen in February, 1886, when he stated that the difficulty in breathing had become greater, and the paroxysms more frequent. The laryngeal mucous membrane was seen to be injected, but on account of the liability to spasm a satisfactory inspection could not be made even after the application of cocaine.

The paroxysms closely resembled an attack of laryngismus stridulus, as seen in children. Physical examination of the chest showed a slight bulging of the sternal end of the right clavicle, and over an area of about an inch and a-half in diameter the percussion note was more resonant than on the opposite side; this area was situated immediately to the right of the sternum, and between the clavicle and the second rib. No pulsation was detectable over the dull area, but there were evidences of hypertrophy of the left ventricle. With these symptoms and physical signs the suspicion of aneurysm was greatly increased, and the patient was directed to come into hospital immediately. This, however, he did not do till the 23rd of March, when he was admitted into Dr. J. W. Anderson's ward. By this time, as recorded in the ward journal, the patient only suffered a little from inspiratory dyspnoea, and the voice was altered in quality and strength. There was no difference in the pulses, but the other evidences of aneurysm were distinct. The dull area had increased in size, and become pulsatile, but without *bruit*. At this time the patient only remained in hospital twenty days, but improved greatly during that period. He was re-admitted on the 20th of April in a worse condition than ever, the dyspnoea being then greater and the aneurysm increased in size. On the 22nd of May Dr. Anderson asked me to see the patient on account of urgent paroxysmal dyspnoea. On laryngoscopic examination both cords were found to be paralysed, and during attempted phonation remained apart, and the dyspnoea was found to be due to the direct pressure of the aneurysm upon the trachea. The question, therefore, of tracheotomy was not raised, and the patient died in the evening.

A *post mortem* examination was made on the 26th of May, when the

following report was made:—"Heart weighs ten ounces, left ventricle dilated. The aorta is atheromatous, and when cut open measures about $4\frac{1}{2}$ inches across. The left subclavian and the left carotid arteries are normal, but at the root of the innominate artery there is a large globular aneurysm about $3\frac{1}{2}$ inches in diameter, and firmly adherent to the sternum. Posteriorly the aneurysm presses upon the trachea, and has pushed it so far to the left that the trachea forms an obtuse angle with the cartilages of the larynx. On dissection, the right recurrent laryngeal nerve was found to be completely obliterated, and the right vagus pressed upon by the aneurysm. The left nerves not involved. A second aneurysm found on a level with sixth and upper part of seventh dorsal vertebrae."

CASE II. *History and Evidence of Syphilis: when first seen the only Symptom was Dysphonia, from Pressure of the Left Recurrent Laryngeal Nerve; and only other Physical Sign, beyond Laryngeal Indications, supporting Diagnosis of Aneurysm, was a Difference in Strength and Rhythm of Radial Pulses: within short interval Development of Paroxysmal Tracheal Dyspnoea, Partial Suppression of Respiration, Murmur on Left Side, and Detection of Dull Areas in Course of Ascending and Descending Aorta: Hypertrophy of Left Ventricle: during six months' residence in ward great Amelioration of Symptoms and Diminution in Size of Aneurysm.*—The patient, E. M. D., aged 50, came first under my observation on account of laryngeal symptoms on the 6th September, 1886, and, on inquiry, it was found that his voice began to fail about three months previously. For this he was unable to assign any cause, but on questioning him the circumstance that he had syphilis many years ago was ascertained, and on careful examination evidences of the disease were found. Inspection of the larynx showed that the left vocal cord remained stationary in the cadaveric position during attempted phonation, and the apex of the arytenoid cartilage was observed to be drawn so far forward that the posterior third of the left vocal cord was not visible. There was no change in the laryngeal mucous membrane, except over the left arytenoid cartilage, where it was thickened slightly, but this induration did not prevent passive movement of the cartilage.

A minute examination of the chest failed to reveal any abnormality in respiration, or even any comparative difference in the two sides. The heart's sounds and the cardiac dulness were practically normal, and there was no appreciable nor abnormal dulness in the course of the aorta. In fact, at this time, the only sign, beyond the laryngeal indications, which supported the presumption of aneurysm, was a difference in the strength of the pulses. On the left side the pulsation of the radial, but especially of the brachial artery, was smaller, and behind the right in rhythm.

At the beginning of September the patient was treated in the out-door department, and on the assumption that the case was one of aneurysm, he was recommended to give up his work (that of a labourer), and moderate doses of iodide of potassium were prescribed. Ten days after his first visit he returned, and stated that during the interval he had followed his usual occupation, and was now in a much worse condition, both in respect to voice and respiration. Since last seen he had suffered, on exertion, for the first time from paroxysmal dyspnoea. He was, therefore, sent to the ward, and on arriving there was seized with a paroxysm of dyspnoea. Inspiration was associated with a whizzing noise, while expiration was free and noiseless; the head was bent forwards, and the larynx remained practically stationary during respiration. There was no cough, and the patient was able to allow a rapid laryngoscopic examination to be made, thereby permitting the position of the vocal cords to be observed. As they were found to occupy the same position as at other times, I therefore concluded that the dyspnoea was tracheal rather than laryngeal, and this opinion was supported by subsequent observations which will be referred to presently. After the patient had been in the ward for a few days I again examined his chest, and although satisfied that some change had taken place since the last examination, I was not confident as to the significance of the newly developed signs. Consequently, I deemed it advisable to have my observations checked by a physician, and asked my friend, Dr. Middleton, to make an examination. He found the pulmonary percussion generally normal, but on the left side posteriorly, at the level of the third dorsal vertebra, there was a distinct but limited, and not very sharply defined, dulness. A dull area also existed on the left side, anteriorly, at the union of the sternum and second rib. Over these areas a v. s. murmur was heard. The heart was hypertrophied, and the aortic second sound accentuated. Throughout the left side the vesicular murmur was now somewhat suppressed; expiration was slightly prolonged, but no râles were heard. As the patient was one of our medical treatment he was transferred to Dr. J. W. Anderson's ward, where he has now (March 18th, 1887) been for six months. During that time he has greatly improved in

every respect; the physical signs of aneurysm, both in front and behind, have become less apparent; the v. s. murmur has disappeared, while the second sound remains accentuated, and other evidences of hypertrophy of the left ventricle still exist, but the difference in the radial pulses and in the respiratory murmur on the two sides continues, and the patient complains of pain on a level with the upper three dorsal vertebrae.

CASE III. *Aneurysm of Descending Portion of Arch of the Aorta: Dyspnoea and Dry Cough, without Alteration in Voice: Physical Examination in Early Stage revealed only Hypertrophy of Left Ventricle, without Bruit: Treatment neglected: Increase in Severity of Symptoms: Physical Evidence of Aneurysm: Continued Dyspnoea, with Paroxysmal Exacerbations.*—J. F., aged 48. The patient was first seen in the beginning of July, 1886, and at that time he stated that occasionally he had considerable difficulty in breathing, and a severe cough, without expectoration; during the intervals between the attacks of dyspnoea the voice was unaltered. Examination of the larynx showed that the approximation of the cords during phonation was complete, but that the left cord did not respond so actively as the right. Otherwise the larynx was normal. The use of the laryngeal mirror was apt to bring on a paroxysm of dyspnoea, especially when the patient endeavoured to take a full inspiration; when he breathed naturally, with care, a good view of the larynx could be obtained. A physical examination of the chest failed to discover any abnormality beyond slight hypertrophy of the left ventricle, without murmur either in the cardiac area or in the course of the aorta. The patient, who was treated at the out-door department, was asked to return in a week. This he failed to do, and was not again seen till the middle of September. He then stated that during the interval the dyspnoea and cough had greatly increased in severity, and an examination of the chest demonstrated a very marked change for the worse. The heart was now found to be displaced downwards, and a circumscribed area, an inch and a half in diameter, giving a dull note on percussion, was found to the left of the sternum on a level with the third rib, and posteriorly a dull area was also detected on the left side on a level with the body of the fourth dorsal vertebra. Over the heart and in the course of the aorta a distinct v. s. murmur was audible. Pulmonary percussion was apparently normal, but on account of the noise produced in the larynx auscultation was unreliable. The paroxysms were more severe than formerly, and were readily induced by any exertion on the part of the patient, but over and above the paroxysmal dyspnoea there was a constant impairment of the inspiratory murmur, most apparent on the right side of the chest.

CASE IV. *Paralysis of the Left Vocal Cord, with Aphonia, and, on exertion, Dyspnoea: Suspicion of Aneurysm at Bifurcation of the Trachea: No increase of Symptoms during six months: Improvement in Voice by Compensatory Action of the Right Cord: Latterly slight Obstruction to Deglutition.*—J. N., aged 68. When first seen, the patient stated that he had always enjoyed good health till the beginning of 1886, when he supposed that he had "caught cold," and after being hoarse for a few days, his voice entirely left him. This was the only symptom complained of till July. About the beginning of that month he experienced unusual difficulty in breathing on exertion, but when at rest respiration was free. When I saw him in September, the only complaint he made, even when questioned, was of loss of voice. On laryngoscopic examination the left vocal cord, during attempted phonation, was found to remain in the cadaveric position, but on direct pressure being applied with a probe to the left arytenoid cartilage, it moved freely. The right cord moved to the middle line only. Physical examination of the chest revealed slight hypertrophy of the left ventricle, and a deficiency in the respiratory murmur, and diminished resonance in the inter-scapular space on the right side, with tubular breathing over the same area on the opposite side. No dysphagia or alteration in the sounds of deglutition.

Taking into consideration all the facts of the case, I was disposed to suspect aneurysm, and directed treatment accordingly. After remaining in Glasgow for a few weeks, during which time I saw the patient occasionally, he went home, but returned again at the end of January, when the following note was made: "Since he left Glasgow, the patient has taken almost complete rest, and has followed carefully the directions given to him. He states that now he can speak much better, but that he is more troubled with his breathing than formerly. On closely questioning him, this dyspnoea is found to be present on exertion, and then amounts only to a sense of oppression. The physical examination of the chest reveals practically the same condition as in September, with this exception, that, whereas then there was no impediment to deglutition of either solid or fluid food, now, although the patient does not appreciate any difficulty in swallowing, auscultation of the oesophagus reveals slight obstruction

to the passage of semi-fluid food at a point over the third dorsal vertebra, and therefore corresponding to the bifurcation of the trachea. The improvement in the voice is accounted for by the circumstance that in place of the right vocal cord coming only to the middle line during phonation, as observed in September, it now crosses, and, to some extent, compensates for the immobility of the left cord, which remains precisely the same as when last examined.

The cases just described may now briefly be reviewed, in order to bring out the main fact which they are intended to demonstrate. They have been selected for the purpose of showing that an aneurysm of the aorta or of the innominate artery may exist, and give rise to laryngeal symptoms for which the patient seeks advice. The apparent laryngeal disease may with the aid of the laryngoscope lead to the discovery of an aneurysm, which might not otherwise have been suspected; but the suspicion once having been raised, a critical physical examination usually reveals one or more circumstances which demonstrate the presence of an aneurysm. The most obscure case is the one last described (No. 4). The age of the patient is in favour both of malignant tumour and of aneurysm, and there are no symptoms to be observed which might not be present in both; but the situation of the disease, the slow development of the symptoms, and, with rest and iodide of potassium, the practically unaltered condition during the last six months, favours the opinion that the paralysis is due to the pressure of an aneurysm.

But while the true nature of this case cannot be definitely decided, it serves to show the difficulties that may arise in certain circumstances. During the early observation of the other cases, in one of them, dysphagia and the laryngeal symptoms were the only evidences of aneurysm, and in the other two, although it cannot be said that there was an entire absence of any of the physical signs or symptoms of aneurysm, yet it may be asserted that the symptoms and signs, other than the laryngeal, if taken alone, would not have raised the suspicion of disease of the great vessels. But while, during the early state, this was so, it must be admitted that the phenomena which developed later on fully justified the diagnosis of aneurysm.

Injury of the recurrent laryngeal nerve was recognised as a cause of aphonia by Galen, who reported a case of a child having lost its voice because of the nerve having been divided by a charlatan, who treated the child for cervical adenitis; and another patient whose voice was permanently lost on account of division of both recurrent laryngeal nerves. Between the time of Galen and that of Stoerk, who appears first to have observed the paralysis laryngoscopically, many cases of laryngeal paralysis consequent upon pressure on the inferior laryngeal nerve were recorded, and long before the laryngoscope came into general use the pathology and symptoms of the lesion were carefully described and understood.

Professor Gairdner, in his *Clinical Medicine*, published before the laryngoscope had practically come into use, points out the importance of laryngeal symptoms, such as vocal disturbance, spasmodic dyspnoea, and imperfect cough, in the diagnosis of aneurysm. By the careful and educated use of the finger, he was able to distinguish when these symptoms were due to a primary laryngeal affection, and when they were the result of pressure upon the laryngeal nerves by an intra-thoracic tumour.

In cases of recurrent laryngeal paralysis, the voice becomes altered in pitch, impure, and wanting in tone, and on straining to speak loudly the voice readily breaks into a falsetto. Other symptoms may also be present, according to the stage at which the case is seen, and may be described either under spasm, consequent upon partial or slight pressure on the nerve, or paralysis, due to greater disorganisation of the nerve-trunk.

Spasm of the larynx as an accompaniment of aneurysm is most frequently unilateral. It is characterised by urgent paroxysmal dyspnoea and laryngeal stridor, accompanied by tremulous action of the heart, not infrequently terminating in syncope, from which the patient may or may not recover. During the intervals the voice may be comparatively little altered, and there may be an almost total abeyance of the symptoms; but, if the patient exerts himself, if he becomes excited, a paroxysm of dyspnoea is likely to be induced.

That death occurs, in cases of aortic aneurysm, as a direct result of laryngeal suffocation, has been attested by numerous cases, and is not now the subject of doubt. The question now to be considered is, what is the cause of these paroxysms of dyspnoea? About the beginning of this century Legallois gave an explanation of the cause of dyspnoea after section of the recurrent nerves, and his observations were extended by very careful and satisfactory experiments by Dr. John Reid, in an investigation into the functions of the eighth pair of nerves.¹ Dr. Reid says: "From the experiments we have detailed,

it is, however, apparent that severe dyspnoea, amounting to suffocation, may arise both from irritation and compression of the inferior laryngeal nerves or the trunks of the pneumogastrics. For when both, or even one, recurrent nerve was irritated, the arytenoid cartilages were approximated, so as in some cases to shut completely the superior aperture of the glottis; and we have already explained at some length how paralysis of this nerve, occasioned by compression or any other cause, should produce this effect by arresting all the movements of the muscles of the glottis."

By experiments performed by Rosenthal,² Rutherford,³ and by Waller and Prevost,⁴ it has been demonstrated that, if the superior laryngeal nerve be divided, and its central end stimulated, bilateral spasm of the glottis will follow. The impression being conveyed by the superior laryngeal nerve to the central nerve nuclei, and on account of the close connection between the nerve nuclei on the two sides, the impression becomes reflected through the efferent nerves (recurrent laryngeals) to the muscles on both sides. We can therefore understand why pressure upon the superior laryngeal or on the trunk of the vagus may reflexly cause either bilateral spasm or bilateral paralysis. It is otherwise, however, when the recurrent laryngeal nerve alone is stimulated. A stimulus acting on the efferent fibres generally causes unilateral adduction or abduction of the vocal cord. You will see from these experiments that pressure upon one recurrent laryngeal nerve is not likely to lead to serious dyspnoea; while, on the other hand, pressure on the vagus, by inducing spasm of the adductor muscles on both sides, or of paralysis on one side and spasm on the other, frequently leads to serious and sometimes fatal laryngeal obstruction.

At this stage, two points in diagnosis may be legitimately raised. Is the obstruction to respiration the result of interference with the function of the larynx, or is it due to diminution in the lumen of the larger air-tubes? and, how are we to determine when the dyspnoea is due to paralysis of the abductors, and when to spasm of the adductors? The first question is of great importance, especially when tracheotomy is contemplated.

Supposing that the patient is suffering from marked obstruction to respiration, his medical attendant must satisfy himself whether the dyspnoea is due to laryngeal obstruction, or to diminution in the diameter of the trachea. In the former event, the difficulty in respiration will be relieved by tracheotomy; in the latter this operation will be of no avail. When the lumen of the trachea is diminished by the pressure of an aneurysm, the dyspnoea is present in a direct ratio to the amount of obstruction; consequently, any transitory enlargement of the aneurysmal sac, from increased action of the heart, resulting from undue exertion, excitement, or passion, may cause a paroxysm. When the dyspnoea is purely tracheal (as in Case II at later stage), the impediment to inspiration is greater than to expiration. The head is kept in its natural position or thrown forwards, the larynx remains practically stationary, the stridor is associated with a whizzing or sipping noise; the voice is weak, but not otherwise altered in character; cough is not necessarily present, nor does it possess laryngeal peculiarities; expiration is, as a rule, easy and noiseless, and may have communicated to it a wavy movement synchronous with the heart's impulses. Tracheal stridor is heard most distinctly at the root of the neck, and over the upper dorsal spines, and if the pressure be greater towards one side the circumstance will be evinced by an alteration in the respiratory murmur on the affected side. On the other hand, in laryngeal dyspnoea the head is thrown backwards, the larynx descends during inspiration, the stridor is associated with a metallic ring caused by vibration of the cords or mucous membrane of the larynx; the voice is greatly altered, or may be suppressed; cough is a frequent symptom, and is usually imperfect; laryngeal stridor is heard most distinctly over the larynx or at the back in the middle cervical region, and, unless there is some other lesion to account for it, the respiratory murmur is equal on both sides. Laryngoscopic inspection is necessary to determine the seat of the lesion, and its importance cannot be over-rated.

Without it needless tracheotomy may be performed. Compressive stenosis, involving the trachea, at or above its bifurcation, may be recognised laryngoscopically, and cases have been recorded where the pulsation communicated to the trachea from the aneurysm was recognised by the laryngeal mirror. In tracheal obstruction the movements of the larynx are not interfered with, unless the recurrent laryngeal nerves are also involved. While, on the other hand, if the obstruction be due to spasm or paralysis of the laryngeal muscles, the appearances already described will be present. In paralysis of the

¹ *Die Athembewegungen und ihre Beziehungen zum Nerven Vagus*. Berlin. 1862.

² Dr. George Johnson, *Med. Chir. Trans.*, vol. lviii.

³ *Arch. de Phys.*, Tome iii, 1870.

⁴ *Phys., Anat., and Path. Researches*, p. 120.

abductors of one, or both, sides the dyspnoea may be explained in two ways, either from sucking in of the flaccid cords, or from accumulation of mucus in the air-tubes on account of efficient coughing being impossible. When due to paralysis, dyspnoea is seldom severe, although persistently present in a greater or lesser degree. On account of the partial closure of the glottis the obstruction is constant, but incomplete, and auscultation over the larynx indicates more or less hindrance to the entrance of air, while expiration is usually unimpeded.

Dyspnoea, although seldom urgent in cases of uncomplicated, unilateral, and abductor paralysis, may become severe from temporary spasm of the adductors on the opposite side. Aphonia and dysphonia are not common symptoms, because of the power of the muscles on the healthy side to compensate for the inefficient action of those on the paralysed side. The voice is impure, and during phonation, in order to throw the cords into tone-producing vibration, the air in the chest is subjected to an abnormal pressure, and, as a consequence, if prolonged, this over-exertion speedily produces exhaustion of the patient's strength and inability to phonate, except in a very imperfect way.

When the paralysis involves the muscles of both vocal cords, inspiratory dyspnoea, much increased by exertion or the recumbent posture, is a marked feature in the case; while the voice, although feeble, may remain almost normal in other respects, and expiration may be free. Some light has been thrown on this subject by physiological experiment.

Dr. John Reid, whom I have already quoted in his physiological and anatomical researches, demonstrates the function of the vagus and inferior laryngeal nerves, and says, "I have witnessed, after the division of the vagi in the middle of the neck, and the consequent arrestment of the movements of the muscles of the arytenoid cartilages, sudden and violent attacks of dyspnoea, which generally went off in the course of a very few minutes, when they did not terminate in suffocation—phenomena which have been considered by some as affording an insurmountable objection to the theory of Dr. Ley, as it supposes the exciting cause of the disease to be permanent and not occasional. In these animals the respirations were performed with ease after section of both vagi, as long as they remained at rest, or made moderate exertion; while, as soon as they began to struggle—in other words, when the respiratory movements were performed with greater force—symptoms of suffocation presented themselves, and these in some cases shortly subsided, occasionally rather rapidly, on the animals refraining from the violent exertion. We have also, in some of these animals, designedly brought on a paroxysm of dyspnoea by causing them to struggle."

"I have lately had frequent occasion to experiment upon the inferior laryngeal nerves, and have observed that very few dogs give any indications of suffering when these nerves are irritated or cut. We are not, however, to conclude that they are entirely motor, for it must be remembered that many dogs remain perfectly quiescent, if previously well secured, during the incisions necessary to expose the nerve. Besides, the anatomical distribution of some of the filaments of the nerve, and the fact that animals do occasionally give indications of suffering on irritating it, are sufficient to prove that it does contain some sensitive filaments."

Whether abduction or adduction follows upon electrical stimulation of the recurrent laryngeal nerve seems to depend upon the relative power of the antagonistic muscles of the larynx, for we find that, while in one animal stimulation of the recurrent laryngeal leads to approximation of the vocal cords, in another it produces exactly the opposite effect. In spasm of the adductors the closure of the glottis, being complete though transitory, gives rise to paroxysms of violent dyspnoea, which come on and pass off with equal suddenness. In the intervals between the paroxysms the voice is unaltered, and auscultation over the larynx fails to detect any impediment to respiration. The amount of pressure to which the nerve is submitted seems to my mind to determine whether or not the patient suffers from dyspnoea. If the pressure be slight, the irritation of the excito-motor nerve calls into action the muscles of the larynx, and the constrictors being the most powerful, closure of the glottis results; whereas, in a later stage, the function of the nerve is impaired or destroyed, and consequently diminished power is observed in the movements of the muscles on the affected side, while reflected spasm may be induced on the opposite side.

The reason why dyspnoea should occur in the later as well as in the earlier stages is explained by the experiments of Dr. John Reid. Let us suppose that the abductors on both sides are paralysed, or that the abductors of the left vocal cord are paralysed, and the right adductors liable to spasm. In both instances the mechanism of the stenosis is practically the same.

In normal inspiration, the vocal cords are drawn widely apart by the abductors, and free admission is permitted to air; but if these muscles be weakened, the opening is diminished in size. Supposing that an unusual effort be made to inspire, the force of the current of air passing to the lungs causes the sides of the glottis to come together, like a valve, which stops the breath till the effort to inspire ceases. When expiration is again attempted, the glottis at once opens.

In animals, when the recurrent laryngeal nerves are cut, the aperture of the glottis is diminished, though still wide enough to allow quiet inspiration to be carried on; but, if the inspiratory movement be violent, the air, rushing through the diminished glottis, carries the passive sides of the paralysed cords inwards, and so the aperture is further reduced. To prevent immediate suffocation, it is necessary to hold the vocal cords apart.

The occurrence or non-occurrence of dyspnoea therefore depends, to some extent, upon the size of the larynx and the violence of the respiratory movements at the time.

Respiration, while quiet, may be unobstructed; but if the patient makes muscular effort, more air is required, the capacity of the thorax is suddenly and greatly enlarged, and the air is made to rush through the diminished aperture of the glottis, causing the characteristic crowing sound, and if the struggle be prolonged, apnoea results from closure of the glottis in the way just described, continuing till the inspirating effort abates. When paralysis of the abductors exists on the left side only, a paroxysm of dyspnoea may be brought on by spasm of the adductors on the opposite side.

Laryngeal obstruction may therefore be explained in various ways, according to the amount of pressure to which the nerves are subjected. But while dyspnoea may very frequently be explained in the way just mentioned, the fact must be borne in mind that the nerves with which the vagus and recurrent laryngeal are intimately connected have a great influence over the bronchial muscles. If the pulmonary plexus of nerves be pressed upon by the aneurysmal sac, spasm of the bronchial muscles may be induced, and symptoms resembling those of spasmodic asthma may appear.

In view of the facts just mentioned, the case first described will now be seen to be of great interest. During the various stages of the disease, the dyspnoea was clearly due to different causes. When first seen, the patient on exertion complained of paroxysmal difficulty in breathing, with a harsh dry cough; but, during quiet respiration there was no interference with the entrance or exit of air. On inspection, the larynx appeared normal, but the slightest local irritation induced a spasm of the right cord. The obstruction to inspiration was considerable though not complete—probably on account of the left cord not being yet involved. The aneurysm was then so small as not to give rise to other evidences of its existence. At this period, the right vagus was probably not compressed, the recurrent laryngeal being the only nerve involved. Two months thereafter, the sac had so increased as to cause a slight bulging at the sternal end of the clavicle, and at the same time the closure of the glottis being more complete, the paroxysms resembled an attack of laryngismus stridulus. This change was in all likelihood caused by pressure upon the trunk of the right vagus, leading to the temporary spasm of the adductors on both sides. This bilateral spasm was doubtless excited by the peripheral irritation of the right vagus, causing an exalted excitability of the nerve-centre; so that the slightest local stimulation, such as the introduction of the laryngeal mirror, induced a paroxysm. A month later, when the patient was admitted into Dr. Anderson's ward, the dyspnoea had greatly diminished, although the aneurysm had increased in size. The protracted irritation of the vagus had then induced structural changes in nerve-centres, and, in place of bilateral spasm of the adductor being easily provoked by local irritation, on attempted phonation, the cords failed to come together. As far as the action of the intrinsic muscles of the larynx was concerned, the patient was now safe; notwithstanding, he was doomed to die from dyspnoea, due to pressure of the aneurysm upon the trachea. The trachea was so displaced to the left, that it formed an obtuse angle with the cartilages of the larynx.

I may conclude this paper by some practical observations. It has been my endeavour to show that the laryngoscopic image or laryngeal symptoms may reveal the presence of an intra-thoracic aneurysm before it is likely to be otherwise suspected, and that even after a critical physical examination of the thorax, no signs may be found, in the early stage, to confirm the diagnosis; but that, as a rule, collateral signs, such as swelling, dull area on percussion, pulsation, thrill, *bruit*, variation in the pulses, inequality in the pupils, or difficulty in deglutition, either warrant one in forming a positive diagnosis, or strengthen the suspicion of aneurysm.

The cases quoted demonstrate that disturbance of the voice due to pressure upon the laryngeal nerves is apt to be mistaken for a primary laryngeal affection. This is an accident which occurs in practice more frequently than is generally believed; it cannot, therefore, be too strongly urged that in all cases where the voice is altered and imperfect without structural disease in the larynx, but especially when the dysphonia is associated with imperfect cough and dyspnoea, aneurysm should be suspected. The absence of other symptoms and of the physical signs of aneurysm is not sufficient to justify one in abandoning the suspicion that the laryngeal disturbance is the result of pressure upon the laryngeal nerves, for it is an acknowledged fact that very small aneurysms may give rise to symptoms of laryngeal obstruction. The laryngoscopist may be led astray by relying upon the absence of symptoms, and be induced to pronounce the paralysis neurotic in its origin. When the laryngoscope reveals, or the symptoms indicate, the characteristic lesion of paralysis, due to pressure on the vagus or recurrent laryngeal nerves, in absence of the phenomena usually associated with aneurysm, the case should be treated as one of aneurysm, and kept under close observation until the diagnosis is complete.

Paroxysmal or constant dyspnoea, attended by other laryngeal symptoms, but without structural disease of the larynx, cardiac or pulmonary lesions, to account for the symptom, should always lead one to suspect the presence of aneurysm, involving either the innominate artery or the posterior and inferior aspect of the arch. When the spasm or paralysis of the vocal cord is limited to one side, the probability is that the recurrent laryngeal on that side is alone compressed, and in neither of these instances is dyspnoea a dangerous symptom, although, in the former, it may be a prominent one. If spasm be present, the pressure is probably slight and of short duration; if paralysis, the pressure is either great or prolonged. When the paralysis or spasm is bilateral, one of two conditions may exist: (1) pressure upon both recurrent laryngeal nerves, or (2) pressure upon the trunk of one vagus. In both of these cases, but especially in the latter, dangerous dyspnoea is to be looked for.

As regards treatment, it will be acceded by all that early diagnosis and successful treatment go hand in hand. Therefore any, even the smallest, indication which will lead to the suspicion of an aneurysm, and thereby cause a critical examination to be made of the facts of the case, must be a great assistance to the practical physician. Cases III and IV exemplify this.

Spasm or paralysis involving only one side of the larynx very rarely causes serious difficulty in breathing in adults, but by direct pressure the original disease may diminish the lumen of the larger air-passages, and give rise to tracheal dyspnoea. It is bilateral paralysis of the abductors, or spasm of the adductors, that causes serious impairment to respiration, for which surgical interference may be required.

There is only one point in the treatment to which I shall allude. It is with reference to the question of tracheotomy in cases of aortic or innominate aneurysm. It is impossible to deny that the paroxysms of dyspnoea not only act very deleteriously upon the aneurysm by causing increased strain upon the walls of the sac, but are also an immediate danger to life. Therefore placing a tube in the trachea will perform a double service—it will permit of rest, and relieve the patient of the danger of death from asphyxia or rupture of the aneurysm during a paroxysm. It may be said that such a proceeding can only give temporary relief; but when the dyspnoea is a consequence of laryngeal obstruction, and not a result of direct pressure of the aneurysm upon the trachea or the lungs, under these circumstances it would be very difficult to draw a limit to the advantages which might accrue from enabling the patient to breathe freely.

If, on examination, the lungs are found to admit air abundantly, and to be free from physical signs of disease, the sooner tracheotomy is performed the better, should the laryngeal obstruction become threatening. The question may be asked—What is the benefit of performing tracheotomy on a patient suffering from thoracic aneurysm, a disease in which the tenure of life is very precarious? In reply, I may say that, as a clinical observer, and as pathologist to the Glasgow Royal Infirmary, experience has taught me that in a large number of cases of aneurysm death from hæmoptysis is preceded by threatenings of laryngeal suffocation; and in many cases I have been convinced that the rupture of the sac has been directly caused by the spasmodic attacks of dyspnoea. It is not only when the patient is suffering from suffocating agony, and all the circumstances indicate that no time is to be lost, that tracheotomy should be performed. The operation, which, in itself, is not attended with danger, should be resorted to whenever it is evident that the patient's life may be placed in jeopardy by laryngeal dyspnoea, or when the laryngeal impediment to respiration

is clearly causing enlargement of the sac by undue strain upon the circulation.

The tracheotomy tube should have an opening on its convex aspect, and be provided with a valve so as to enable the patient to breathe by the mouth and speak with occluding the tube with his finger. Such a tube may be worn without danger, or even much inconvenience. Intubation of the larynx is not applicable in such cases, but should be reserved for cases of laryngeal stenosis arising in the course of acute disease, and the removal of the paralysed cord, as proposed by Dr. G. B. Hope in cases of paralysis of the abductor muscles, is only mentioned, not recommended.

LECTURES

ON

CERTAIN DISEASES OF THE JAWS.

Delivered at the Royal College of Surgeons of England, June, 1887.

By CHRISTOPHER HEATH, F.R.C.S.,
Hunterian Professor of Surgery and Pathology.

LECTURE II.—TUMOURS OF THE JAWS.

(Concluded from Vol. i, page 1579.)

Osteoma.—The simplest form of osseous tumour of the upper jaw is an hypertrophy of the whole or of some portion of the bone. In October, 1883, I had under my care in University College Hospital a young woman, aged 25, in whom a painless enlargement of the right upper jaw had been noticed for ten years, encroaching upon the palate and bulging out the cheek. I successfully removed the whole upper jaw, and on section the tumour was found to be simple bone, very dense, but otherwise healthy. One half of the specimen is in University College, and the other in the College of Surgeons' Museum (2211 A).

Besides this form of bony tumour, due apparently to an increase of the cancellous structure of the bone, specimens of tumour as hard as ivory have from time to time been met with. Perhaps the most remarkable of these is one described by Mr. Hilton in the *Guy's Hospital Reports*, vol. i, p. 493, from the fact that the tumour separated spontaneously from the face. The patient was a man, aged 36, who, twenty-three years before Mr. Hilton saw him, noticed a pimple below the left eye, close to the nose, which he irritated, and from that spot the tumour appears to have originated. The tumour, in its growth, displaced the eyeball, giving rise to excruciating pain, which subsided

Fig. 13.

on the bursting of the ball. It began to loosen by a process of ulceration around its margin six years before it fell out, which event was unattended by either bleeding or pain; the tumour weighed 14½ ounces. It was tuberculated externally, and an irregular cavity existed at the posterior part. A section presented a very hard polished surface resembling ivory, and exhibited lines in concentric curves, enlarging as they were traced from the posterior part. The huge cavity left by the tumour was bounded below by the floor of