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President—W. M. MOLLISON, M.Chir.

Discussion on the Laryngeal Paralyses

ARCHIBALD DURWARD: I propose to confine my remarks to a brief account of the accepted central connections within the medulla of the nerves which are distributed to the muscles and the mucosa of the larynx. The nerves involved are the vagus and the bulbar portion of the accessory. The classification of these nerves may be unfortunate and confusing; the bulbar portion of the accessory must be regarded functionally as a part of the vagus complex, concerned in the innervation of certain striped muscles along the course of the vagus in the neck. The association of the bulbar and spinal parts of the accessory is brief; from the viewpoint both of peripheral distribution and of central nuclear connections the two are independent.

Of the various branches of the vagus complex in the neck, only two concern us in the present discussion, namely, the superior laryngeal nerve and the inferior or recurrent laryngeal nerve, and some points about the so-called anastomosis between them in the laryngeal region will bear discussion. The actual pattern of the anastomosing rami of the two nerves varies, but such a connection is to be regarded as constant. It has been suggested that some superior laryngeal fibres might be distributed in the field of the recurrent nerve, and that the superior laryngeal nerve might supply or assist in the supply of the muscles other than the cricothyroid. Certain specific muscles have even been given a definite supply from the superior laryngeal, e.g. the interarytenoid, muscle. There is no good or sufficient evidence for this view. Experimental work on animals* lends no support to the view that the internal branch of the superior laryngeal nerve is motor to laryngeal muscle. If fibres can be traced into some muscles in man from the superior

* Lemere, F., *Amer. Journ. Anat.*, 1932, li., 417.

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laryngeal nerve or from the anastomosis between it and the recurrent, this need not mean that any such muscle is receiving a motor supply from the nerve concerned; such fibres may be sensory to muscle and mucosa. The older view that the recurrent nerve supplies all the muscles except the cricothyroid is the more acceptable to-day. The recurrent nerve is predominantly motor, but contains sensory fibres from the lower larynx as well. The superior is predominantly sensory and supplies only one muscle of the larynx.

A section of the medulla shows the nucleus ambiguus as a long column of cells deeply placed and adjacent to the spinal tract of the trigeminal. From this cell column fibres pass into the ninth, tenth and the bulbar portion of the eleventh nerve. The caudal part of the nucleus ambiguus is the centre for laryngeal movements and from the cells here the fibres travel towards their distribution in the larynx, *viâ* the bulbar portion of the eleventh nerve. The blood-supply of the nucleus ambiguus generally comes within the field supplied by the posterior inferior cerebellar artery. Sensory fibres from laryngeal mucosa travel in both laryngeal nerves to the tractus solitarius. Cell bodies are located in the vagal nuclei. The tractus solitarius also receives afferents from the seventh and ninth nerves (taste). The nucleus of the tractus solitarius is placed immediately adjacent and its cells can deflect the impulse to the cord and brain stem nuclei for reflex purposes or towards the thalamus, probably *viâ* the medial fillet.

Cell bodies may be found in and about the laryngeal muscles and along the course of the laryngeal nerves. Their nature is a matter of dispute; they may be aberrant receptor cell bodies or, again, peripherally placed parasympathetic neurones—most probably the latter.*

C. A. Joll: As early as 1885, when operations on the thyroid gland were still unusual, Jankowski[4] gave a description of laryngeal paralysis following total thyroidectomy, and Wölfler[8] in 1890 recorded six bilateral cases with four deaths. These publications induced Billroth[11] and Kocher[18] to systematize the operation of hemithyroidectomy, but this proved advantageous only because it resulted in the substitution of unilateral for bilateral paralysis. Socin and Reverdin avoided some of the dangers by resecting only the more prominent masses of thyroid tissue. Mikulicz's[17] melon-slice resection of the thyroid, though more successful in avoiding damage to the recurrent laryngeal nerves, proved even less satisfactory as a means of extirpating diseased thyroid tissue; Kocher's[16] method of resection-enucleation for thyroid adenomata fulfils most of the desiderata for avoiding nerve damage, though it is not applicable to all types of goitre.

* Lemere, F. *Anat. Record*, 1932, liv., 389.

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Anatomy.—Either or both of the laryngeal nerves, superior and inferior (recurrent), may be damaged through thyroid operations. The superior laryngeal nerve often lies close to the inner side of the trunk of the superior thyroid artery, particularly at a point a short distance above the superior pole of the gland. In ligating the main artery, remote from the pole, the nerve may be inadvertently included. I have often identified it during preliminary operations for ligation of the superior thyroid artery—if touched, it gives a sharp painful sensation—and this itself may provide sufficient warning to enable one to avoid it. The best course, however, is to ligate the artery as close as possible to the pole of the gland.

The recurrent laryngeal nerve is comparatively seldom exposed in routine operations of the thyroid gland, because in most cases both lobes of the gland can be dislodged without disturbing the loose cellular tissue in which the nerve lies. I estimate that I expose the nerve sufficiently to recognize it, in not more than 10 per cent. of all goitre operations and then usually at the stage when the inferior thyroid artery is to be ligated in continuity. It is only when the artery happens to be a little difficult to identify, or in a slightly unusual position, that the dissection necessary to expose the artery reveals the nerve also. It is of little practical value to know that in 65 per cent. the nerve lies behind the artery and in 26 per cent. in front of it, that in 9 per cent. it passes between its branches, or that it is inclined to be a little deeper on the left than right; it is sufficient to be aware that it may lie in any of these positions and to be alert particularly when approaching *the middle third of the lateral border of the gland.* By pulling the lateral lobes forward, no essential change occurs in the relationship of the nerve to the inferior artery or the thyroid gland, and therefore this manœuvre, if properly carried out, is perfectly safe, even though Crile[7] and others advise against it. The recurrent nerve lies at first outside the surgical or false capsule of the gland and is a little further from the trachea on the right than on the left; as it passes upwards it approaches the gland and penetrates the posterior layer only, of the false capsule; the thin anterior layer of the capsule which is apposed to the postero-internal aspect of the gland is not perforated. Thus it follows that the recurrent nerve lies between the two layers into which the surgical capsule is divided posteriorly, while the inferior artery or its branches must perforce perforate both layers of this capsule. In all but a few exceptionally lowly placed inferior arteries, the nerve is at least half to three-quarters of an inch medial to the trunk of the artery at the point where the latter emerges from behind the carotid sheath. There is a rare anomaly of the recurrent nerve worthy of note which I have seen once only; similar cases have been described by Pemberton

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and Beaver,[19] Milianitch,[28] and others: the vagus may give off the recurrent nerve relatively high in the neck so that instead of hooking upwards behind the right subclavian it merely crosses the common carotid to reach the posterior border of the thyroid gland rather more transversely than usual. It is for this reason more vulnerable.

Physiology.—The opinion held most widely in this country is that the intrinsic laryngeal muscles are supplied by the recurrent laryngeal nerve and that the crico-thyroid is supplied by the external branch of the superior laryngeal. On the other hand Dilworth,[13] Berlin and Lahey,[12] New,[29] Nordland,[26] and others, claim that the arytenoideus and possibly other laryngeal muscles have a double nerve supply, i.e. derived from both the superior and inferior laryngeal nerves. None of them go so far as Exner[14], who believed that all the intrinsic muscles have a double nerve supply, though Berlin and Lahey[12] suggest that the internal laryngeal is usually the exclusive motor nerve to the arytenoideus.

A number of recent writers, New and Childrey,[24] also Smith, Lambert and Wallace,[23] have cast doubt on Semon's law[5] mainly because in none of the cases reported by the former and in only one reported by the latter investigators did the vocal cord pass from the median to the cadaveric position with the lapse of time. The real crux of the discrepancy appears to me to lie in the essential difference between a slowly progressive lesion of the nerve, such as is produced by an infiltrating neoplasm, and the sudden trauma inflicted during an operation. The latter must nearly always fail to provide the necessary conditions for a strict limitation of the damage to the abductor fibres. So far as purely laryngoscopic appearances are concerned my own experience accords closely with that of New and Childrey.[24]

It is much more difficult to obtain a clear idea as to the incidence of pre- and post-operative laryngeal paralysis than would be expected. The reasons are:—

(1) Systematic pre-operative and post-operative laryngoscopic examinations have seldom been carried out in a series of cases.

(2) Such statistics as are available are based largely upon the patient's capacity to speak clearly. We know that complete unilateral paralysis, whether pre- or post-operative, is compatible with a normal speaking voice. On the other hand post-operative laryngitis and tracheitis are very likely to modify the voice without any gross paralytic changes.

Pre-Operative Paralysis.—In the case of malignant goitre, paralysis may be bilateral or unilateral, complete or incomplete, and is probably due to direct invasion of the nerve. In simple goitre, paralysis is due to stretching of the nerve over a tense,

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bulky or rapidly growing nodule or cyst, but it may also follow the attacks of low inflammation common in and around nodular goitres, or be the result of hæmorrhage into an adenoma. In many cases it is impossible to explain the exact mechanism of the paralysis (*cf.* Layton's case[9] of bilateral paralysis in Graves' disease).

Very few reliable statistics are available. In 1,000 non-malignant goitre cases at the Mayo Clinic, Matthews[1] found 262 in which partial or complete, unilateral or bilateral paralyses existed, but he fails to differentiate between functional and organic lesions and it is clear that his criteria must be at fault to produce so high a figure. Feuz[20] in a series of 250 cases from Roux's Clinic found only one instance of pre-operative paralysis. In 1,000 cases from de Quervain's Clinic, the pre-operative incidence was 3·1 per cent. (partial and complete). In a series of 422 cases of my own examined by my laryngological colleagues at the Royal Free Hospital, there was a total of five functional and eleven organic lesions (see Table I).

TABLE I.

LARYNGEAL EXAMINATION OF A SERIES OF CASES OF THYROID DISEASE AT THE ROYAL FREE HOSPITAL FROM JANUARY 1ST, 1930 TO SEPTEMBER 30TH, 1934.

Type of disease.	Number of cases examined by laryngoscope.	Normal.	Abnormal.	Pre-operative.	Post-operative.
I. Primary thyrotoxicosis	200	195	5	4	1
II. Secondary thyrotoxicosis	98	92	6	4	2
III. Generalized nodular goitre	48	44	4	4	0
IV. Localized nodular goitre	63	60	3	3	0
V. Malignant goitre ..	4	3	1	1	0
VI. Colloid goitre	7	7	0	0	0
VII. Lymphadenoid goitre ..	2	2	0	0	0
Total ..	422	403	19	16	3

Statistics of post-operative laryngeal paralyses are largely invalidated by the absence of a systematic pre-operative and post-operative laryngoscopic examination. Kocher[6] had 9 per cent. in 900 cases, von Eiselsberg[15] 20 per cent. in 330 cases and de Quervain[2] 2·9 per cent. in 1,000 cases; most of these figures being subject to the proviso I have already made.

Feuz[20] reports a series of 250 cases from Lausanne examined carefully, before and after operation, with a 7·6 per cent. incidence of organic paralyses due to the operation.

In my series of 422 Royal Free Hospital cases the post-operative laryngoscopic findings were as follows (it must be noted that unless for special indications most of the post-operative laryngoscopic examinations were made several days after the operation, so that transient paralyses may have been missed).

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TABLE II.

PERCENTAGE INCIDENCE OF LARYNGEAL PARALYSIS.

Total:	4·52%
(*a*) Pre-operative	3·79%
(*b*) Post-operative	0·73%

	Total incidence.	Pre-operative.	Post-operative.
I. Primary thyrotoxicosis ..	2·5%	2%	0·5%
II. Secondary thyrotoxicosis ..	6·12%	4·08%	2·04%
III. Generalized nodular goitre ..	8·33%	8·33%	0·0%
IV. Localized nodular goitre ..	4·79%	4·79%	0·0%
V. Malignant goitre	25%	25%	0·0%

TABLE III.

ANALYSIS OF CASES OF LARYNGEAL PARALYSIS IN DISEASES OF THE THYROID GLAND.

	Primary thyrotoxicosis 5 cases.	Secondary thyrotoxicosis 6 cases.	Generalized nodular goitre 4 cases.	Localized nodular goitre 3 cases.	Malignant 1 case.
Functional paralysis	3 pre-operative	0	2 pre-operative		
Unilateral recurrent laryngeal nerve paralysis					
(*a*) Incomplete	0			1 pre-operative	
(*b*) Complete	1 post-operative	4 (2 post- and 2 pre-operative)	2 pre-operative	1 pre-operative	
Bilateral recurrent laryngeal nerve paralysis					
(*a*) Incomplete					1 pre-operative
(*b*) Complete		1 pre-operative		1 pre-operative	
External laryngeal paralysis	1 pre-operative	1 pre-operative			

Prognosis.—Judd[3] states rather vaguely that the prognosis in immediate paralysis is good, but appears to refer to the vocal recovery only. Feuz[20] reports that approximately 50 per cent. of the cases of complete and incomplete recurrent laryngeal paralysis which occurred in the Lausanne series (250 cases) disappeared within six to twelve months. The prognosis for pre-operative paralysis seems to be less favourable than for those which are the direct result of the operation.

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In most cases the cord lies in the median position and remains there ; only rarely does it pass into the cadaveric position. Apparently changes from the cadaveric to the median position are not uncommon. Some paralyses are said to be transitory, but in the absence of repeated examination of the larynx this class of case is not easily definable and the symptoms ascribed to them may be due to local mechanical or inflammatory changes in the larynx rather than to actual nerve involvement.

Compensatory changes are, fortunately, the rule : so that functional recovery is commoner than restoration of nerve function—the contralateral cord swinging over the mid-line to lie close to its paralysed fellow.

In my series, in one pre-operative case of incomplete bilateral paralysis the larynx is now quite normal. In three others, all of them pre-operative complete unilateral lesions, there has been no change except a trivial alteration from the cadaveric towards the median position. In two partial unilateral lesions there has been restoration to normal ; in a third case there has been no change. The post-operative paralyses have persisted, though phonation is good in all three patients.

If prophylactic hæmostasis is made the primary principle of the operative technique, the nerve is, *ipso facto*, protected. There will then be no necessity for forcible plugging of the wound with gauze, or for the blind and hurried application of hæmostatic forceps in the depth of the wound. If by good fortune it is recognized during the operation that one recurrent nerve has been divided or included in a ligature, it is essential to remove the latter or to carry out primary suture of the divided nerve-ends, using for this purpose the finest silk on round-bodied needles, similar to those used in vascular suture. All attempts to practise *secondary nerve suture* for recurrent nerve lesions appear to me to have little chance of success. It is unlikely that the nerve-ends will be recognizable, or that they can be mobilized sufficiently to permit repair. Lahey[10] claims a success by this method, but his description of the result ten months later hardly bears out his claim. The same applies to the case of Horsley and Miller.[25] Colledge and Ballance[27] describe a successful bilateral anastomosis between the phrenic nerve and the divided inferior laryngeal in a woman of fifty-four. Provided the distal end of the latter can be exposed and recognized for certain, this appears to me a more promising method than secondary nerve suture.

It is almost impossible at first to distinguish between bilateral laryngeal paralysis and tracheal collapse, but I am convinced that when the former is asserted to have cleared up quickly, following a tracheotomy, the actual lesion present was, in fact, confined to the trachea. I have had a case of this sort. Sudden stridor and

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cyanosis was noticed during an operation for thyrotoxicosis in a middle-aged man. A diagnosis of bilateral paralysis was given and a low tracheotomy opening made, but within a couple of days the patient was able to dispense with the tube. His cords were found to be sluggish and inflamed, but free from paralysis. No gross evidence of tracheal collapse had been detected at the time of the operation. I have no personal experience of the use of a permanent tracheotomy tube for these bilateral cases. The only proved case of bilateral paralysis which has arisen in my own practice (not, however, in the statistical series which I have presented in this paper), was of delayed onset, and the dyspnœic attacks which drew my attention to the case did not cause much distress until upwards of a fortnight after the operation. Possibly this was an instance of paralysed cords passing from the cadaveric to the median position, as Lahey says is the rule. Crile[7] recommends the permanent use of a special type of valved tracheotomy tube (Waugh's) for these cases, and there are other forms of cannulae available which carry the stream of air back above the adducted cords, thus permitting improved respiratory freedom.

Réthi[21] has described a method for detaching the adductors of the cords from their insertions after laryngofissure and claims that his successes (15 out of 16) have been confirmed by Kahler and by Albrecht.

Lemere,[22] in bilateral cases, advocates inducing paralysis of the cricothyroids by injecting the external laryngeal nerves with alcohol. He asserts that by abolishing the tensor effect of these muscles, the cords move apart and that breathing is much improved, though at the expense of phonation.

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GEORGE RIDDOCH: Discussion still centres round the question of the central connections of the laryngeal nerves. The consensus of opinion now is that the motor fibres arise from cells in the lower part of the nucleus ambiguus. This nucleus, which innervates the palate from its upper part and the pharynx from its middle part, belongs to the ventrolateral column of nuclei, of which the fifth and seventh also form a part. These groups of cells are concerned with the striated muscles which are derived from the branchial arches, and constitute the special visceral efferent column. The fibres which are destined for the larynx leave the medulla by the accessory nerve but, through the internal ramus, soon join the vagus by which they travel to their terminations. Sensory impulses from the larynx conveyed by the superior and recurrent laryngeal nerves and vagus trunk, pass through the ganglion nodosum, in which their cells lie, to the dorsal vagus nucleus in the bulb. It is probable that the chief medullary end-station for non-sensory afferents is the tractus solitarius and its nucleus (visceral afferent column). These impulses are concerned with visceral reflexes and, by means of a descending pathway, take part in the reflex activities of respiration and coughing (Ransom).

CAUSES OF LARYNGEAL PARALYSIS

Supranuclear: In Parkinsonism the only prominent disorder of laryngeal function is a low-pitched, monotonous voice. This common clinical condition is easily recognized by the mask-like face, bent posture of the body, slow movement, and cogwheel rigidity. Like hemiplegia, it is not in itself a disease, but a syndrome, the result of lesions of the motor basal ganglia due to several causes, such as degeneration—in paralysis agitans; inflammation—in encephalitis lethargica and, rarely, in syphilis; or vascular—in cerebral arteriosclerosis.

In double hemiplegia and pseudobulbar palsy, similar dysphonia occurs. These disorders are uncommon and are usually found in the middle or late periods of life. They are distinguished by weakness, spasticity and the reflex changes characteristic of pyramidal lesions and facile emotionalism.

Nuclear: (1) Acute bulbar palsy.

(*a*) *Vascular:* Hæmorrhage with survival is rare. The usual lesions are thrombosis of the basilar, or posterior inferior cerebellar arteries from either arteriosclerosis or syphilis.

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The onset is sudden but may have been preceded by other symptoms and signs of cerebral arteriosclerosis or syphilis, such as headache, giddiness, mental impairment, or ocular palsies.

With thrombosis of the basilar artery, symptoms vary with the severity of the block and are usually asymmetrical. Dysphonia, if present, is associated with dysarthria, dysphagia and sometimes dyspnœa. There is weakness of the face and the limbs on one or both sides, with reflex changes and commonly unilateral or bilateral sensory loss, especially to pain, heat and cold on the face, body, and limbs. Cerebellar and vestibular disorders such as ataxia, nystagmus, and vertigo are usually present.

Thrombosis of the posterior inferior cerebellar artery, a branch of the vertebral, which supplies a wedge-shaped area on the lateral aspect of the medulla as well as the inferior part of the cerebellum and the dentate nucleus, is a relatively common vascular lesion in this situation, and its syndrome is easily recognized. There is sudden vertigo, commonly diplopia from skew deviation, and staggering with or without vomiting. Sometimes there is severe pain in the face and head on the side of the lesion, which may persist long after the acute phase is over. The limbs, especially the upper limb, on the side of the lesion, are inco-ordinate and hypotonic, and there is nystagmus. On the same side, the palate, pharynx and larynx may be paralysed with some dysphagia and dysphonia. Involvement of the descending sensory root and nucleus of the fifth and of the spinothalamic tract results in loss of sensibility to pain, heat and cold, and sometimes to light touch, although in less degree, on the face on the side of the lesion and on the trunk and limbs on the opposite side.

(*b*) *Inflammatory.*—Acute anterior poliomyelitis may attack the pons and bulb, giving rise to nuclear palsies, weakness of the limbs and cerebellar defects. The death-rate is high from respiratory paralysis. It occurs in young people, and the onset of the nervous symptoms is usually preceded and accompanied by fever, malaise, pains and meningeal signs. Excess of cells, chiefly lymphocytes (although polymorphonuclears are found in the acute stage), slight reduction of chlorides and slight increase in protein are found in the cerebrospinal fluid.

Ascending atonic paralysis, first described by Landry, which begins in the lower limbs and spreads to the upper limbs and trunk, may reach the brain-stem and give rise, amongst other symptoms, to weakness of the vocal cords. It is a rare event in epidemics of acute anterior poliomyelitis, but is most commonly due to severe polyneuritis from various causes such as alcohol, diphtheria and other intoxications. I have recently seen a case which followed acute streptococcal infection of the tonsils in a boy aged ten years

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and, because of respiratory paralysis, the patient, who is steadily recovering, was saved only by being kept continuously in a Drinker respirator for five weeks.

Disseminated sclerosis sometimes declares itself in young adults with symptoms pointing to lesions in the pons and occasionally in the bulb. The combination of vertigo, ataxia, unilateral deafness and external rectus and facial palsy with pyramidal signs of sudden onset is not very rare, and with it there may be such bulbar symptoms as dysphonia and dysphagia. As with all acute or subacute lesions, if the patient survives, and this is the rule in disseminated sclerosis at this stage, improvement soon begins.

Encephalitis lethargica in the acute stage occasionally attacks the bulb and gives rise to laryngeal palsies. But they are commonly associated with distinctive features of the disease from lesions in the mid-brain and hypothalamus, such as diplopia and nystagmus, disorders of sleep-rhythm and involuntary movements.

(*c*) *Traumatic.*—Severe injuries of the bulb are almost always fatal. But in injuries of less severity, weakness of the laryngeal muscles, presumably from lesions of the nucleus ambiguus, are sometimes found in association with other bulbar signs.

Chronic bulbar palsy.—Progressive bulbar palsy: This disease of unknown origin is rare under the age of fifty. It attacks the motor nuclei of the bulb (almost always in the first instance the hypoglossal) so that by the time the larynx is affected, dysarthria, especially for consonants, for which free movement of the tongue is necessary, has become evident. Fibrillation of the tongue can be seen from the first, but wasting comes later. The orbicularis oris, which is supplied through the seventh nerve from the hypoglossal nucleus, is affected at the same time. Later the palate, pharynx, larynx, lower part of the face and the respiratory muscles become progressively paralysed and there may be spastic weakness of the limbs from pyramidal lesions. Dysphonia is associated with attacks of choking, dysphagia and nasal regurgitation of fluids.

Syringobulbia: The tumour process is rarely primary in this region and usually extends from the cervical enlargement of the spinal cord. Bulbar signs, which include dysphonia, dysphagia and dysarthria from destruction of the nucleus ambiguus, often unilateral or asymmetrical, are preceded by the characteristic analgesia and thermo-anæsthesia in the face, head, neck, upper limbs and chest.

Bulbar compression.—Tumours of the cerebellum from compression, invasion, or both, may lead to laryngeal palsy, but the associated manifestations of hydrocephalus and cerebellar disorders make the diagnosis clear. Should bulbar symptoms appear early they, as a rule, indicate an infiltrating, gliomatous tumour.

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Extracerebellar tumours, especially when posteriorly placed, compress the bulb and cerebellum as well as the cranial nerves which arise from the pons and medulla. Aneurisms of the basilar or vertebral arteries, if they are large enough, do the same, but they are uncommon and their symptoms are variable and asymmetrical. With aneurisms a murmur can sometimes be heard on auscultation of the back of the head.

Weakness of the vocal cords in tabes may be due to lesions of either the nucleus ambiguus or the vagus.

Peripheral.—The vagus along with other bulbar nerves may be damaged within the skull or in the jugular foramen by inflammation, for example, secondary to otitis or infective lateral sinus thrombosis or from syphilis, or by tumour. The ninth, eleventh or twelfth cranial nerves, because of proximity, may be injured with the vagus, and various syndromes have been described.

The superior laryngeal nerve, along with the fibres for the palate and pharynx, leave the vagus at the ganglion nodosum so that lesions at or above this level are associated with sensory loss in the larynx.

The superior laryngeal branch is rarely damaged alone, but injury of the recurrent laryngeal is the most frequent cause of unilateral paralysis of the vocal cord. The commonest causes of recurrent laryngeal paralysis are, in the chest, in addition to aortic aneurism, mediastinal neoplasm and enlarged glands in Hodgkin's disease and, in the neck, enlarged glands, and cancer of the œsophagus.

Paralysis of individual muscles in the larynx is always due to local conditions, such as inflammation or growths.

Muscular.—Laryngeal palsy is frequently a symptom of myasthenia gravis. It is a disease of early adult life, and affects females more commonly than males. Although nervous lesions, except occasionally retrograde changes, are absent, the muscles which are usually earliest and most severely affected are those supplied by the brain-stem. Ocular palsies, such as ptosis, diplopia, squint, and weakness of orbicularis oculi commonly come first but are followed by paresis of the face, masseters, tongue, palate, pharynx and larynx, with corresponding symptoms. All the voluntary muscles of the body may become affected, and life may be threatened or ended from feebleness of the muscles of respiration. The weakness temporarily diminishes or disappears with rest and in consequence all voluntary muscles, including those of the larynx, should be used as little as possible.

Hysterical.—Aphonia is a well-known hysterical manifestation.

Spasm of the Glottis.—Adduction is most common but transient spasm of the abductors is sometimes a consequence of sudden fright

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or severe pain. Laryngismus stridulus usually occurs in children but is less common since rickets has become rarer.

Adductor spasm also occurs in the tabetic laryngeal crises, in hydrophobia and in tetany, and is the cause of the " cry " which often ushers in an epileptic fit.

Sir FREDERICK HOBDAY said that veterinary surgeons operated for laryngeal paralyses with a different motive from that of operators on the human subject and he had now operated on some 4,000 cases. Sir Felix Semon once asked him what became of the voice, and he had replied that if the operation was a success, his patient, although it could gallop without distress, was dumb. Sir Felix then said that the operation would be useless for the human patient, for a man might not mind his inability to run but he did wish to talk. However, when the number of operations had reached about 1,500, he had found that a number of his patients did attempt to " talk ", for they produced a muffled neighing sound, which was one of the means of knowing whether a horse had undergone an operation of this kind.

A point of agricultural importance was that Government premium stallions travelling the country must now be certified not to have had an operation on the throat. He himself did not believe that roaring was universally hereditary. Roaring usually occurred after a cold, pneumonia, strangles, or influenza, and the causes were mainly invasion of the throat glands by the streptococcus of Schultz.

In one case only of those upon which he had operated was the right side alone paralysed. In a few both sides were affected, but in a large majority the left side alone was affected.

With animals the operation was solely a matter of utility. Whether the patient was a hunter or a cart-horse, it was better to have this operation performed than for the animal to have to wear a tube.

After the operation he usually asked that the horse should be given three months' rest. The minimum should be six weeks, the object being to make sure that the adhesions had become firm. The operation was a plastic one and merely consisted in stripping the ventricle of Morgagni. By stripping the ventricle two raw surfaces were made, and the vocal cord was gradually pulled back by the adhesions. In from twelve to fourteen days the external wound was healed and the horse was then kept quiet on the pillar-rein for another week.

Günther had carried out a similar operation in Germany a hundred years ago, but he had so many deaths that he had advised his students not to do it. In those days, however, there were neither anæsthetics nor antiseptics.

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