CHAPTER VIII.

DISEASES OF THE VAGUS (PNEUMOGASTRIC NERVE), "VAGUS NEUROSES."

IMMEDIATELY behind the superficial origin of the glosso-pharyngeal on the postero-lateral aspect of the medulla oblongata, the vagus appears, with its ten to fifteen separate bundles, which soon unite to form one trunk. This is a flat band which, accompanied by a process of the dura, passes outward below the flocculus, together with the accessorius, to the anterior division of the jugular foramen, inside of which is to be found the ganglion of the root of the vagus, or, as it is also called, the jugular ganglion. After its exit from the skull the vagus receives a part of the accessorius, and forms the ganglifform plexus or the ganglion of the trunk, which, however, only transmits a part of its fibres.

About the difference in the further course of the left and right vagus we shall have a few words to say later.

That the nucleus of the vagus is only a part of the nucleus common to it, the glosso-pharyngeal, and the accessorius, has already been stated in the preceding chapter. The cells of the part belonging properly to the vagus are spindle-shaped, multipolar, 30 to 45 μ long and 12 to 15 μ broad (hence much smaller than the cells of the hypoglossus nucleus, which we shall describe later). As another important origin of the root fibres of the vagus, a compact round nerve bundle following the longitudinal axis of the medulla oblongata must be mentioned. It has been described by Meynert as the solitary fasciculus, while Krause designates it as the respiratory fasciculus, because it connects the vagus with the origin of the most important respiratory muscles (cf. Fig. 18). The so-called nucleus ambiguus (in the diagram *n. am*) is held to be still another nucleus of the vagus. This is a collection of peculiar nerve cells situated within the formatio reticularis to the mesial side of the nucleus lateralis.

Just as most of the cranial nerves, the vagus may be diseased in its centre as well as in its peripheral course. The first class of cases are usually met with as partial manifesta-

tions of other, general, diseases (tabes, hysteria). The latter are distinct affections in themselves, which may occasionally be due to peripheral causes, such as indigestion, catching cold, or reflex influences, diseases of the intestines and the uterus. Very frequently, it is true, the seat of the disease remains ante as well as post mortem obscure, and this is not to be wondered at if we remember that we know little or nothing about the pathological anatomy of the vagus. Among the cases hitherto observed, many were not fitted to throw any light on the symptoms manifested during life, as in numerous instances no abnormality at all was found in the nerve, so that we are led to assume that the disease was purely functional (i. e., a disease without appreciable anatomical basis). The pathology of the vagus, therefore, belongs to the most obscure chapters in the pathology of the cranial nerves, and the following can only be considered to be an imperfect attempt at giving a comprehensive exposition of the highly interesting diseases connected with this nerve.

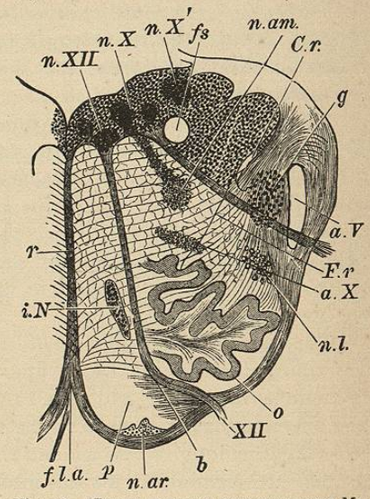

Fig. 18.—CROSS-SECTION THROUGH THE MEDULLA OBLONGATA. (After SCHWALBE.) *a.V*, ascending root of the fifth. *n.XII*, nucleus of the hypoglossus. *n.X* and *n.X'*, nucleus of the vagus. *XII*, hypoglossal nerve. *fs*, solitary funiculus (respiratory fasciculus). *p*, pyramidal tract. *o*, olive. *i.N*, pyramidal nucleus. *f.l.a.*, anterior longitudinal fissure. *n.am*, nucleus ambiguus. *n.l.*, nucleus lateralis.

Since the symptoms may sometimes be the same whether the disease is of central or peripheral origin, we shall, so as to avoid repetition, deviate from our usual method of division, and give our attention chiefly to the question how lesions of the vagus may influence (*a*) respiration, (*b*) circulation, (*c*) digestion, functions which, as is well known, are chiefly under the control of this nerve.

A. Affections of the Air-passages due to Lesions of the Vagus.

1. The larynx, above all, interests us in this connection. This organ is innervated by the vagus and the accessorius, though it is still a matter of doubt whether all the motor fibres originate from the latter or only those that innervate the muscles used in the production of voice, while the vagus presides over the respiratory movements of the vocal cords; the sensory fibres of the larynx certainly all belong to the vagus.

The branches of the vagus, which come off in the cervical portion of the nerve and innervate the laryngeal muscles, are the superior laryngeal and the inferior or recurrent laryngeal. The former leaves the vagus at the lower end of the gangliform plexus, and divides into a motor branch, which goes to the crico-thyroid muscle, and into a sensory branch, which contains the fibres for the mucous membrane of the epiglottis and the whole laryngeal mucous membrane above the vocal cords.

The recurrent laryngeal is shorter on the right side, because, without going beyond the upper aperture of the thorax, it curls around the subclavian artery, and runs back in a groove between the trachea and the œsophagus upward to the larynx, while on the left side it has to make the long course around the arch of the aorta. Its terminal branch (R. terminalis) divides into two twigs, which together supply all the muscles of the larynx, with the exception of the above-mentioned crico-thyroid, with motor nerves, and the mucous membrane of the parts below the vocal cords with sensory fibres.

Of the laryngeal muscles, the posterior crico-arytenoids draw the vocal cords apart—that is, they are the abductors or openers—while the lateral crico-arytenoids in conjunction with the lateral thyro-arytenoids draw them together, and are therefore called adductors or closers. Of these muscles, on each side the "abductor" arises at the posterior surface of the cricoid cartilage and passes upward and outward to the end of the muscular process of the arytenoid cartilage, while the other, the "adductor," arises from the upper margin of the cricoid cartilage and is inserted at the outer side of the muscular process of the arytenoid cartilage. It moves the muscular process forward, being thus the antagonist of the abductor. The crico-thyroids provide for the elongation and tension of the vocal cords; they are assisted by the internal thyro-arytenoids, which run parallel with the vocal cords.

In the laryngeal muscles paralysis and, though comparatively rarely, spasms have been observed.

The chief forms of paralysis, which we shall here consider, are (1) the paralysis of the recurrent laryngeal, in which case all the muscles supplied by this nerve are paralyzed (or weakened); (2) the so-called abductor paralysis—that is, paralysis of the posterior crico-arytenoids, the openers of the glottis; (3) paralysis of the internal thyro-arytenoids.

Without going into the much-discussed and still unsettled question as to the mechanism of these paralyses, we have attempted to give a succinct and clear summary of the clinical symptoms, including the appearances found on laryngoscopical examination (cf. table on page 116).

The existence of a cerebral centre for the laryngeal muscles is shown by the fact that in different cerebral affections—e. g., pseudo-bulbar paralysis and certain brain tumors—but only in rare instances (Rougé, Progrès méd., 1892, 36), paresis or paralysis of the vocal cords has been observed. In chorea adductor paresis has been noted. A most curiously perverted action of the vocal cords has been observed by Krause in the course of hysteria; on inspiration they were approached, while on expiration the glottis was wide and gaping.

Another form of central paralysis is the nuclear. In complete paralysis of one vocal cord a lesion in the accessorius nucleus of the corresponding side has been found; the usual cause of this, however, seems to be a peripheral affection of the trunk of the vagus, or of the recurrent laryngeal (by pressure, contusion, injuries, surgical operations, tumors, and aneurisms), yet we are not often in a position to speak with certainty as to the seat of the affection, and to say whether this is central or peripheral. The nature of the laryngeal paralyses which occur in general neuroses (hysteria, epilepsy), intoxications (lead), infectious diseases (diphtheria, dysentery, cholera), is quite obscure. The easiest to understand are those acquired through straining of the voice and diseases of the larynx itself (catarrh, perichondritis). (B. Fränkel on mogiphonia, cf. lit.)

The prognosis ought to be guided by the consideration of the nature of the primary affection, but we should also take into consideration the functions of the affected muscles, and not forget that, for instance, in abductor paralysis, danger of suffocation may arise at any moment. It is always unwise to predict the exact time of recovery; the course of such paralyses is usually very protracted.

The treatment of most of the cases has to be conducted by

a specialist, and consists in touching the vocal cords with the sound (Rossbach), and in the external or intralaryngeal use of electricity. Faradization of the different laryngeal muscles necessitates a dexterity which can only be attained after a thorough acquaintance with the laryngoscopical technique. The general treatment of any primary affection need not be discussed here.

Spasms of the laryngeal muscles, we have said before, are very rare, and are in general, with the exception of the spasm of the glottis, of not much practical importance. Most frequently the spasm affects the adductors, and the condition then resembles very much that of abductor paralysis, with this exception, that the spasm is generally quite transitory, while the paralysis is often of long duration. The aphonia spastica described by Schnitzler, a disturbance of co-ordination of the muscles of the vocal cords, which, on an attempt at phonation, contract spasmodically, is found occasionally in chorea and hysteria.

The spasm of the adductors, which occurs especially in early childhood, is called spasm of the glottis (laryngismus stridulus, laryngospasmus, asthma thymicum sive Millari). Its paroxysms usually occur unexpectedly without external cause. They consist in the main in a total arrest of respiration lasting from several seconds to a minute and a half, and are ushered in by a deep inspiration which is accompanied by signs of suffocation. Only rarely does the child die during the attack; usually a few deep, very audible respirations indicate the cessation of the spasm, and the child seems completely well after a comparatively short while. No definite statement can be made with regard to the number and intensity of the individual attacks, because innumerable variations can occur. The anatomical seat of the disease is entirely unknown; yet the fact that not rarely eclampsia or epilepsy complicates the affection rather speaks for the possibility of a temporary irritation of the cortical centre for the laryngeal muscles. The remarkably frequent occurrence of it in conjunction with rachitis has led to the idea (Elsässer) that we are dealing with a rachitic softening of the posterior part of the skull, which has rendered possible pressure upon the brain. Nothing definite is known about the cause. In the treatment early hardening of the child and rational nutrition play an important *rôle*. Robust, well-nourished children who can stand changes in temperature without at once catching cold, etc., are hardly ever affected with laryngismus stridulus; only delicate children with a convulsive tendency, who have been fed on farinaceous foods and other inappropriate substitutes for the mother's milk, fall a prey to the disease. There is no medicinal treatment for the affection. During the attacks we have to avoid the danger of suffocation by carefully watching the epiglottis, sprinkling the body with ice-water, brushing and tickling the soles of the feet. After the attack we may give nervines (belladonna, bromide) and, perhaps to avoid a too frequent repetition, narcotics (morphine, 1 to 3 milligrammes—gr. $\frac{1}{60}$–$\frac{1}{20}$ subcutaneously). The treatment of the rachitis should never be omitted.

Sensory disturbances of the larynx manifest themselves either in anæsthesias, or, what is less common, in hyperæsthesias of the mucous membrane, and are especially found in the distribution of the superior laryngeal. They are not rarely combined with motor changes, paralysis or paresis of the pharyngeal muscles (cf. chapter xi), but often they appear alone. The most common form is the anæsthesia attending diphtheritic paralysis; it is characterized by the absence of the reflex gagging and cough which normally follow touching the laryngeal mucous membrane with the sound, the finger, or the laryngoscope. In such cases it may happen that the food on deglutition enters the larynx, and, through faulty closure of the glottis, can not be removed by coughing, and thus gives rise to dangerous attacks of choking, and even to aspiration pneumonia. The latter does not seem to occur in cases of purely hysterical anæsthesia.

The hyperæsthesia is found in ulcerative processes, or in bad, acute catarrhs. Although it seems to play a prominent *rôle* in hysterical patients, it is in reality not present, but is erroneously said to exist by patients who are forever worrying themselves and finding new ailments, or is produced by autosuggestion.

The anæsthesia calls for electrical treatment, galvanization of the larynx and the palatal muscles, the faradic brush to the throat, etc. To meet the hyperæsthesia, narcotic remedies may be of service, but in hysterical patients often no other treatment but a good sensible lecture is needed.

LITERATURE.

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Fränkel, Bernh. Ueber die Beschäftigungsschwäche der Stimme, Mogiphonie. Deutsche med. Wochenschr., 1887.

TABLE OF THE MOST COMMON PARALYSES OF THE LARYNGEAL MUSCLES.

	Kind of paralysis.	Occurrence.	Symptoms.	Ophthalmoscopic picture.
Inferior or Recurrent Laryngeal.	Complete recurrent laryngeal palsy.	In compression paralyses of the vagus or the recurrent laryngeal (carcinoma œsophagi), often unilateral (left), as initial symptom of aortic aneurism. In tabes.	Voice not clear. Patient is easily tired on talking. Coughing impossible.	Vocal cords slightly abducted, the so-called "cadaveric position" (Fig. 19). In forcible phonation the healthy cord reaches beyond the middle line. Overriding of the arytenoid cartilages (Figs. 20, 21).
	Abductor paralysis (paralysis of the posterior crico-arytenoids).	In diseases of the nerve itself, the causes of which are often unknown.	If bilateral: extreme inspiratory dyspnœa; if unilateral: inspiration hampered, long-drawn, noisy. Dyspnœa on the least exertion. Speech but little affected.	Glottis appears as a narrow slit, becoming still narrower on inspiration (Fig. 22). Inability to abduct the paralyzed vocal cord (Fig. 23).
	Paralysis of the internal thyro-arytenoids.	In catarrhs of the mucous membrane of the larynx. After overexertion of the voice. In hysteria.	Voice hoarse; speaking an effort.	Glottis does not close completely on phonation (Fig. 24). If at the same time the arytenoids are paralyzed, the glottis presents an hour-glass outline (Fig. 25). Neither anterior nor posterior portion is closed, but the vocal processes are in their normal position.
	Adductor paralysis (paralysis of the lateral crico-arytenoids).	Rarely isolated. In hysteria.	Absolute absence of voice. Power of coughing retained. "Phonic paralysis" (Türck).	Nothing characteristic.
Superior Laryngeal.	Paralysis of the crico-thyroids.	After diphtheria.	Voice rough; high tones impossible.	Excavation of the vocal cords. Cords do not vibrate visibly.

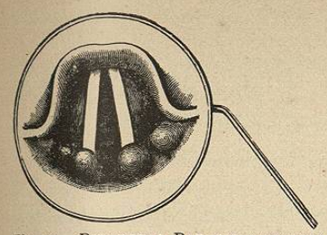

Fig. 19.—Bilateral Paralysis of the Recurrent Laryngeal. "Cadaveric position" of the vocal cords.

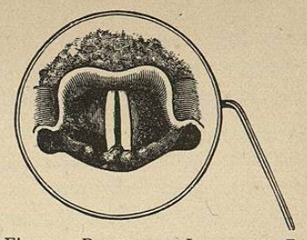

Fig. 20.—Recurrent Laryngeal Paralysis. Overriding of the arytenoid cartilages.

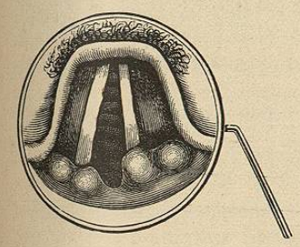

Fig. 21.—Paralysis of the Recurrent Laryngeal on the Left Side (in inspiration).

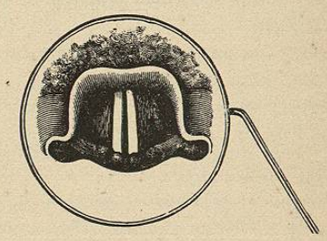

Fig. 22.—Paralysis of both Posterior Crico-arytenoids (in inspiration).

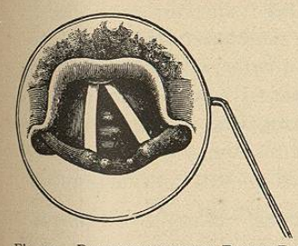

Fig. 23.—Paralysis of the Right Post. Crico-arytenoid (in inspiration).

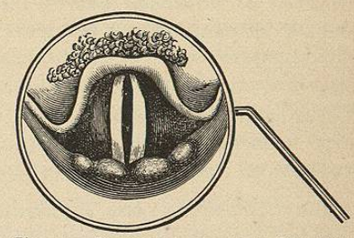

Fig. 24.—Paralysis of both Internal Thyro-arytenoids (acute laryngitis).

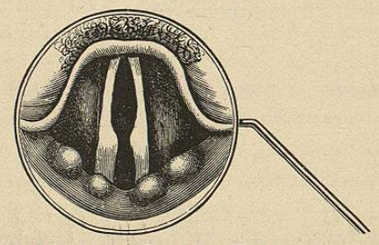

Fig. 25.—Paralysis of both Internal Thyro-arytenoids, associated with paresis of the arytenoid muscle.

Figs. 19–25.—Partly after Strümpell, partly after Eichhorst.