CHAPTER X.

DISEASES OF THE HYPOGLOSSAL NERVE.

The ten to fifteen bundles of fibres of which the hypoglossal nerve consists, as it emerges from the medulla oblongata in the groove between the anterior pyramid and the olivary body, unite to form two larger bundles, which leave the dural space separately, and, after their entrance into the hypoglossal canal, the anterior condyloid foramen, become a single stem, which leaves the cranial cavity by this canal. Outside the base of the skull it passes along to the mesial side of the vagus, at first obliquely downward and forward, then obliquely upward, runs on the outer surface of the hyoglossus muscle, and soon reaches the region where the genioglossus muscle radiates into the tongue. There are various communications between the hypoglossus, the vagus, the anterior branches of the upper cervical nerves, and the lingual branch of the trigeminus.

The cortical area of the hypoglossal nerve is found, according to Exner, in the lower portion of the anterior central convolution and the adjoining portion of the inferior frontal convolution, as shown in Fig. 28. Its nucleus is situated in the floor of the fourth ventricle, where its very large nerve cells, which measure up to 60 μ in diameter, closely resemble the large multipolar cells of the anterior horn in the cord. After the closure of the central canal it is situated to the ventral side of the latter.

The root fibres of the hypoglossus certainly arise in part from the nucleus of the same side. To what extent the nucleus of the opposite side, as well as the group of nerve cells situated in its neighborhood and the above-mentioned nucleus ambiguus, can be considered sources of origin for them, and, moreover, whether direct fibres of the hypoglossus have their origin in the cerebrum, is still undecided.

While in certain of the cranial nerves—for instance, in the facial—peripheral affections occur at least as frequently as central, in the case of the hypoglossal this is not true. Often as its nuclei take part in the most diverse diseases, especially of the cord and medulla oblongata, it is rare that a peripheral

affection comes under observation. That, in a given case, the disturbance is peripheral and not central, more especially not bulbar, we may conclude from the absence of other symptoms of bulbar disease, and from the possible presence of complete

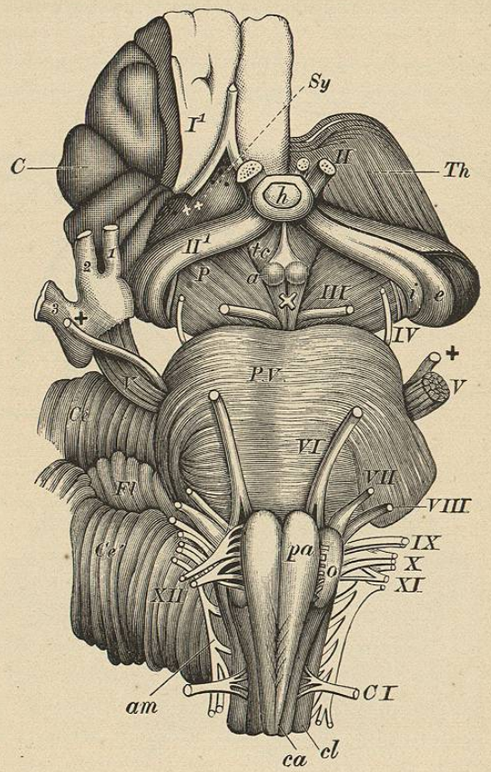

Fig. 27.—SUPERFICIAL ORIGIN OF THE CRANIAL NERVES. *I–XII*, the twelve cranial nerves. *CI*, anterior root of the first cervical nerve. *ca*, anterior column of the spinal cord. *cl*, lateral column. *pa*, anterior pyramids. *o*, olivary body. *P. V.*, pons Varolii. *i*, internal geniculate body. *e*, lateral geniculate body. *tc*, tuber ciner. *h*, pituitary body. *P*, cerebral peduncle. *Sy*, region of the fissure of Sylvius. *a*, corpora albicantia. *C*, island of Reil. *Th*, optic thalamus.

reaction of degeneration, as Erb (cf. lit.) has done in his recently described case. The symptoms otherwise are the same as in the central disease.

Central paralysis of the hypoglossus may be, in the first place, of cortical origin. According to Exner, as has been stated, the cortical area for the tongue is situated close to the

point where the middle and inferior frontal convolutions join the anterior central convolution, and it is very probable that injury to this region causes a motor disturbance in the tongue. In a case of tubercular meningitis, Matthes (Münchener med. Wochenschrift, 1892, 49) has observed a hypoglossal paralysis,

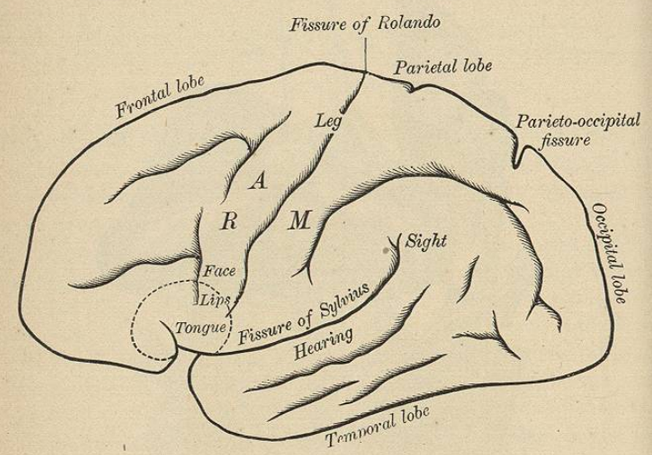

Fig. 28.—Cortical Centres of the Left Hemisphere. (After Gowers.)

which he attributes to a localized tubercular meningitis at the convexity over the centre for the hypoglossus. On the whole, central palsies of this nerve are rare.

The bulbar lesion of the nerve, or rather of its nucleus, is somewhat better understood; it has undoubtedly been observed, if not frequently, at least repeatedly, that this lesion can occur unilaterally. There is then an atrophy of the nucleus, in which the nerve cells and the medullated fibres become decreased in number or disappear entirely, while the roots appear as fine threads. In such cases (see especially Fig. 29) the tongue is protruded, not straight, but deviates toward one side, and be it remembered toward the affected side (m. genioglossus and geniohyoideus); it shows fibrillary twitchings, and an atrophy of the diseased side—hemiatrophia linguæ—which in such a case looks flabby and shrunken in comparison with the full and firm healthy half; it is wrinkled, contracted, and much smaller than the latter (cf. Figs. 29 and 30, showing my two cases). The electrical examination shows either normal reaction or reaction of degeneration; that the

latter may also occur in central lesions has been demonstrated by one of my cases, which, however, did not come to autopsy. Speech, mastication, and deglutition often suffer considerably; on the other hand, the healthy half of the tongue may develop so satisfactory and vicarious an activity that little disturbance is observable.

Unilateral paralysis of the hypoglossal nerve, due to peripheral causes (Birkett, Neurol. Centralblatt, 1891, 24) has been ob-

Fig. 29.—Hemiatrophia Linguæ (personal observation).

served as a result of traumatism; further, also, in diseases of the vertebral artery, as the result of new growths in the medulla oblongata and in cases of embolic softening in the region of the nucleus (Hirt). Whether it can be also of saturnine origin seems to me to be doubtful, in spite of the report of Remak. In a recently published article by Koch and Marie (cf. lit.) may be found all the cases observed up to the present time collected and minutely analyzed. A case of congenital hypoglossal paralysis has been observed by Francotte (Annal.

de la soc. méd.-chir. de Liège, 1889), which is undoubtedly an instance of infantile nuclear degeneration (Möbius).

In bilateral paralysis of the hypoglossal the tongue, atrophic, wrinkled, and shrunken, lies almost motionless on the floor of the mouth; the patient can not protrude it, and has entirely lost control over it. Speaking and chewing are rendered difficult, even quite impossible. This sad picture is seen not infrequently in Duchenne's progressive bulbar paralysis, occasionally

Fig. 30.—HEMIATROPHIA LINGUÆ (personal observation).

in progressive muscular atrophy, very rarely in tabes. The hemiatrophy of the tongue, too, occurs much less frequently in the course of tabes than, to judge from the communications—for instance, those of Ballet (cf. lit.)—would seem to be the case.

The peripheral form of the affection may yield to electrical treatment (faradization and galvanization); the central, so far as we know at present, is not amenable to any treatment.

Hypoglossal spasm occurs sometimes unilaterally, somewhat more frequently bilaterally. It is an exceedingly rare

affection, in regard to which there have been but few good publications. There is a paroxysmal, involuntary spasm of the tongue, by which it is protruded and retracted, rolled violently around in the mouth, and so roughly pressed against the teeth that it may be quite severely injured. In some instances there occur short rhythmical twitchings in the whole tongue which disappear at times. Berger observed an aura before such an attack, which consisted in a sensation of tension and swelling of the tongue. In Dochmann's case the attacks occurred especially at night, and were so violent that the patient was awakened from her sleep by the sudden spasmodic protrusion of the tongue. In one of my own cases the muscles of mastication took part in the affection in such a way that before the actual hypoglossal spasm occurred, the lower jaw was for half or a whole minute spasmodically jerked to and fro, up and down. After these movements had ceased the mouth remained half open, and the turning and rolling movements of the tongue commenced and lasted for about one minute. These attacks recurred ten to twenty times a day; they came on for the first time three days after an epileptic fit, and have lasted unaltered ever since (for three years). The patient is otherwise perfectly healthy, and has a good family history. The pathogenesis of the disease, its anatomical seat (irritation of the hypoglossus centre? cortical or bulbar?), is obscure. As an accompanying symptom of chorea and hysteria it is by far more common than as an independent affection. Possibly the so-called auctioneer's spasm (Zenner, Berliner klin. Wochenschrift, 1887, 17), which is caused by overexertion (speaking and shouting), should be classed as a form of hypoglossal spasm. The treatment is the same as in paralysis of the tongue.

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CHAPTER XI.

SIMULTANEOUS AFFECTION OF SEVERAL CRANIAL NERVES—MULTIPLE PARALYSIS OF THE CRANIAL NERVES.

AFTER having thus considered the lesions of the individual cranial nerves, it remains for us to inquire under what conditions several of them may be simultaneously affected, and into the symptoms thus produced. According to the observations collected up to the present time, an affection of this kind may have its seat in the peripheral or in the central course of the nerves, as well as in the cortical or nuclear centres. Only certain of the affections of this latter kind are to be regarded as independent diseases, while the peripheral lesions are always only partial manifestations of other conditions. In rare cases a simultaneous peripheral lesion of several cranial nerves may occur in consequence of traumatism, operative interference, etc. A case in point, in a patient operated upon by Israel, has been published by Remak (Berl. klin. Wochenschr., 7, 1888). A carcinoma of the neck was extirpated, and by the operation the accessorius, the hypoglossus, and the sympatheticus were injured, or rather resected. The symptoms caused by the accident were accurately described by Remak. Other instructive cases, due to traumatism, have been described by Möbius (cf. lit.).

Among the general diseases in which multiple cranial nerve lesions may occur are chiefly tuberculosis and syphilis.

Tubercular meningitis attacks, by preference, the membranes at the base, and implicates most of the cranial nerves emerging in that region, as we have seen in our account of the diseases of the meninges. Lately Kahler (cf. lit.) has again directed attention to the fact that, in consequence of syphilis, a peripheral neuritis of the cranial nerves sometimes develops, and that we may, besides general cerebral symptoms, have a progressive slow paralysis, which attacks one cranial nerve