Sauer. Fall von traumat. Hypoglossus- u. Accessoriuslähmung. Inaug. Diss., Göttingen, 1886. (Unilateral Luxation between Atlas and Epistropheus.)
Peel. Berl. klin. Wochenschr., No. 29, 1887. (Hemiatrophy of Tongue, with Left-sided Recurrens Paralysis.)
Koch et Marie. Hémiatrophie de la langue. Revue de méd., viii, 1, 1888.
Morison. Brit. Med. Journ., July 14, 1888. (Unilateral Paralysis of the Hypoglossal in Consequence of Traumatism.)
Limbeck. Prager med. Wochenschr., 1889, 16.
Pasquier et Marie. Séméiologie de la langue. Progrès méd., 1891, 12.
Birkett. Neurol. Centralbl., 1891, 24.
Lange, F. Ueber Zungenbewegungen. Arch. f. klin. Chir., 1893, xlvi, 3, p. 634.

2. *Hypoglossal Spasm.*

Berger. Ueber idiopathischen Zungenkrampf. Neurol. Centralbl., i, 3, 1882.
Dochmann. Petersb. med. Wochenschr., 1, 1883.
Wendt. Unilateral Spasm of the Tongue. Amer. Journ. Med. Sc., clxxvii, p. 173, Jan., 1885.
Erlenmeyer. Centralbl. f. Nervenheilk., ix, No. 5, 1886. (Case of Idiopathic Spasm of the Tongue.)
Bernhardt. Ueber idiopathischen Zungenkrampf. Ibid., No. 11, 1886.
Lange, F. Ein Fall von beiderseitigem idiopathischem Hypoglossuskrampf. Ein Beitrag zur Lehre vom Ritter-Rollett'schen Phänomen. Arch. f. klin. Chir., 1893, xlvi, Heft 4.

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

after the other in irregular succession (cf. also Rothmann, Deutsche Med.-Ztg., 1893, 46).

After diphtheria peculiar forms of paralysis are observed, which chiefly take in the muscles of the soft palate and the pharynx. Since these muscles are innervated by certain of the cranial nerves, and the disease is unquestionably—e. g., when the paralysis is unilateral—often of peripheral origin (central diseases can not in all other cases be excluded), we shall devote a few lines to the consideration of their nerve supply.

The innervation of the palatal and pharyngeal muscles is by no means one of the clearest chapters in neurology. We do not know exactly which of the cranial nerves are concerned, nor their mode of distribution. Of the palatal muscles the levator palati is the most important. This receives motor fibres through the large superficial petrosal (of the trigeminus) from the spheno-palatine ganglion, which come from the facial and which also innervate the azygos uvulæ. Whether or not, however, the vago-accessorius and the glosso-pharyngeus are also concerned in the innervation of these muscles, as Gowers, for instance, seems to think, basing his arguments upon clinical observations, is not as yet decided. With regard to the pharyngeal muscles, it is generally assumed that the stylo-pharyngeus and the middle constrictor are supplied by the glosso-pharyngeal nerve, and that the palato-pharyngeus, the superior and inferior constrictors, are innervated by the vagus. The participation of the accessorius is doubtful (Schwalbe). We see then that the nerves concerned in a paralysis of the pharynx are the facial, the glosso-pharyngeal, the vagus, possibly also the fifth and the accessorius.

Pharyngeal paralysis may be either unilateral or bilateral. The unilateral form can only be diagnosticated if the patient is made to move the soft palate, for instance, in saying "Ah!" While during rest it appears to be perfectly symmetrical, the base of the uvula deviates somewhat on motion towards the affected side, so that on that side a little way from the median line there is a slight depression not present on the well side; sometimes also the soft palate is a little lower on the paralyzed side even during rest. In the bilateral complete paralysis of the soft palate, the latter hangs down flaccidly and the uvula appears elongated; on deep respiration and on phonation it remains motionless, and the reflex movements evoked by tickling the mucous membrane are lost. Speech becomes markedly altered, the voice acquires a nasal tone, due, of course, to the cavity of the nose not being shut off during phonation; hence also the pronunciation of the explosive consonants "P" and "B" becomes impossible, owing to the imperfect compression of the air; they sound like "M." Closure of the anterior nares removes, as Duchenne has shown, this disability. From the same cause also fluids are regurgitated through the nose on attempts at swallowing, and deglutition in general becomes difficult.

Recent examinations of the nerves (Arnheim, Arch. f. Kinderkrankheiten, 1892, xiii; and Hochhaus, Virchow's Archiv, 1892, cxxiv, Heft 2) have demonstrated that lesions are present in various peripheral nerves, not only those going to the muscles of the palate and the fauces. Hansemann also has described (Virchow's Arch., 1889, cxv, Heft 3) the condition of the cranial nerves in diphtheria. Absence of the knee-jerks has been repeatedly found associated with paresis of the palate in diphtheria (Berl. klin. Wochenschr., March 30, 1885, p. 204).

The prognosis in post-diphtheritic paralysis is not unfavorable if the velum palati alone is paralyzed. If, on the other hand, the muscles of the œsophagus also take part, the outlook becomes graver on account of the inability of the patient to take nourishment, and all the more so if feeding by the stomach-tube is not constantly and carefully practiced. If this is not done, aspiration pneumonia or inanition may bring about a fatal issue.

The electrical treatment ought to be begun as early as possible. It consists in the direct faradization or galvanization of the velum and the frequent excitation of reflex movements of deglutition by stimulation of the throat. The uvula, the pillars of the pharynx, etc., are directly touched and repeatedly stimulated by means of the curved button electrode (cf. Fig. 31). The movements of deglutition are obtained if the anode is placed on the neck and the cathode (button electrode) is quickly drawn

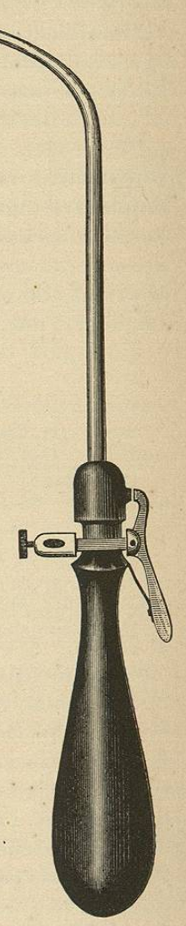

Fig. 31. — Pharyngeal and Laryngeal Electrode with Arrangement for making and breaking the Current. (After Erb.)

over one of the lateral surfaces of the larynx, six to ten cells sufficing for the purpose. These gymnastics of the pharyngeal muscles constitute an excellent remedy which can not be replaced by any other. It often leads rapidly to recovery.

Central diseases of several cranial nerves at the same time may also occur, and that, too, not only in their intracerebral course—which for but few of them is known, and for those only imperfectly—but also in the centres themselves. As a matter of fact, our knowledge about the centres situated in the cortex is also very incomplete, since we must again confess our comparative ignorance of the anatomy. Still, we shall not go too far if we assume that extensive cortical lesions may implicate several centres together, and there is no doubt but that they may be affected after or rather during an apoplectic attack by "indirect action."

LITERATURE.

Reinhard. Deutsche med. Wochenschr., 1885, No. 19. (Subcutaneous Injections in the Region of the Neck of 0.001 (gr. $\frac{1}{60}$) of Strychnia daily.)
Rothmann. Ibid., 1885, No. 52. (Paralysis of the Muscles of Respiration after Diphtheria.)
Hansemann. Virchow's Archiv, 1889, cxv, Heft 3.
Hallager. The Paralyses after Diphtheria. Hosp. Tid., 1890, 4.
Garcia y Mansilla. Nature and Treatment of the Post-diphtheritic Paralyses. Rivista clin. de los hospitales, 1891, 32.
Suckling. Brit. Med. Journ., May 28, 1892. (Three Cases of Paralysis of the Diaphragm after Diphtheria.)

Of eminently practical importance are the nuclear affections of the cranial nerves. Referring the reader to the preceding chapters for the anatomical position of the individual nuclei, we will only remind him of the fact that these nuclei are situated in the gray matter, partly of the mid- and 'tween- brain, partly in the medulla oblongata. The portion situated above the latter extends from the posterior wall of the infundibulum in the third ventricle to the level of the nucleus of the abducens, and embraces the nuclei of the eye muscles (Wernicke). The other nuclei belong to the lower portion.

Clinical observations now teach us that either of these portions may be affected by itself, and we may with Wernicke call the disease of the upper, polioencephalitis superior; that of the lower, polioencephalitis inferior. According to the course, we distinguish in either case an acute and a chronic form, so that

there are altogether four clinical pictures of these nuclear affections.

Polioencephalitis superior acuta has only been observed in very few instances. The best observations we owe to Wernicke. According to him, this is essentially an acute inflammatory disease of the nuclei of the ocular muscles, and proves fatal in from ten days to a fortnight, the focal symptoms consisting in an associated paralysis of the eye muscles, the general symptoms being grave disturbances of consciousness. The walk presents a peculiar combination of spasm and ataxia. Anatomically, foci of acute softening are found in the region of the nuclei, which are either due to obstruction of the blood-vessels or to inflammatory infiltration of the tissues. Ætiologically, the abuse of alcohol may be mentioned.

With reference to the diagnosis, the presence of a tumor in the region of the corpora quadrigemina should be considered (B. Sachs, New York, Diseases of the Mid-brain Region, Am. Jour. of the Med. Sci., March, 1891).

Polioencephalitis superior chronica was described in 1868 by von Graefe, and called by him ophthalmoplegia progressiva. The first published case presented, according to von Graefe (Berl. klin. Wochenschr., 11, 1868), a peculiar clinical picture:

"Gradually all the muscles concerned in the movements of the eye become paralyzed, so that there results first a diminution in the range of sight, and finally complete immobility of the eyeballs. The levator palpebræ superioris is wont to be implicated, although the consequent ptosis is rarely as marked as that occurring in complete oculo-motor paralysis. It is remarkable that, on examination for reaction to light and accommodation, the sphincter pupillæ as well as the ciliary muscle present no changes. This condition, which we very rarely find in other extensive oculo-motor paralyses, seems here constant and characteristic of this disease. Another feature which distinguishes this form from other associated paralyses in the distribution of the third, fourth, and sixth nerves is the progress of the disease *pari passu* in the antagonizing muscles. Thus we never find a marked strabismus divergens owing to a dominating oculo-motor paralysis, because here the external rectus loses its functions sufficiently to neutralize the tendency to deviation, and the sight of the patient is therefore, in spite of the associated paralysis of the eye muscles, affected much less than

in simple oculo-motor or abducens paralysis. . . . Still, a certain degree of asymmetry in the affection of the different muscles of one eye, as well as in the development of the whole disease in the two eyes, may at times be found." (Cf. Wernicke, *loc. cit.*, vol. iii, p. 463.)

With the exception of this associated ocular palsy, which, developing progressively, may remain stationary without being completely symmetrical, the patient enjoys good health and complains neither of headache nor of symptoms of increased intracranial pressure. In isolated instances bulbar paralysis has been known to be later superadded, and in others the disease was found associated with multiple sclerosis or with general paralysis (Ballet, Progrès méd., 1893, 23). Anatomically, the affection depends either upon a primary disease of the nerve nuclei or upon a diffuse sclerotic process in which the nuclei take part. In exceptional cases, which in their nature are as yet entirely obscure, no organic changes whatever have been found, although the clinical picture corresponded exactly to that described by von Graefe. (Eisenlohr and Oppenheim.)

Not less interesting, and at the same time of far greater practical importance because relatively far more frequently met with, is the fourth and last of the affections under consideration—a disease the first accurate description of which we owe to Duchenne, of Boulogne, and which after him has been carefully and successfully studied by German investigators (Wachsmuth, Kussmaul, Leyden)—the chronic progressive bulbar paralysis.

Progressive Bulbar Paralysis.

Paralysis of the Tongue, the Soft Palate, and the Lips (*Duchenne*, 1860), *Glosso-labial Laryngeal Paralysis* (*Trousseau*), *Chronic Progressive Bulbar Paralysis* (*Wachsmuth*, 1864), *Atrophic Bulbar Paralysis* (*Leyden*), *Bulbar Nuclear Paralysis* (*Kussmaul*), *Polioencephalitis Inferior Chronica* (*Wernicke*).

Duchenne's Disease.

Symptoms and Course.—In the majority of instances the onset of progressive bulbar paralysis is very gradual, and only rarely do we meet with cases in which it is ushered in by an apoplectiform attack. After having complained for weeks, perhaps months, of drawing, tearing pains in the neck and the back, the patient discovers of his own accord or from the remarks of his relatives that the enunciation of certain words,

especially those containing *l*, *r*, and long *e*, has become very difficult. If he happen to use a word containing all these letters (for instance, reel), he becomes painfully conscious of his indistinct enunciation. In vain he attempts to repeat the troublesome words over and over again in order to correct his mistake. He only becomes more convinced that the movements of his tongue have become clumsy, and that he has lost his former ease and fluency of speech; and, in truth, it is the oncoming paresis of the lingual muscles which is the main cause of the disturbance.

The tongue, which can not be raised to the normal extent, can no longer be approached sufficiently to the hard palate, and thus the long *e*, for the pronunciation of which the movement is necessary, can only be pronounced with difficulty. In the same way all the finer muscular movements required for the formation of the linguals are imperfect, and consequently the enunciation of these sounds is bad. The disease progresses and the articulation becomes worse and worse, the less perfectly the lingual muscles are innervated, and other letters, *s*, *t*, *g*, finally also *d* and *n*, begin to suffer, so that conversation with the patient becomes very uncomfortable, as certain words are almost unintelligible and others at least difficult to understand.

The lips also begin to do their duty badly, so that the enunciation of the so-called labials—*o*, *u*, *a*, *b*, *p*—gradually becomes indistinct. The presence of strangers with whom he has to converse excites the patient, and, avoiding all society, he prefers the quiet monotony of the family circle, where nobody seems to pay much attention to the change in his speech ("alalia and anarthria"). Moreover, a change in the features of his face, at first slight and only noticed by the patient himself, but later more perceptible and evident also to his friends, gradually manifests itself, which serves as an additional reason for seclusion (Fig. 32). When laughing, it appears to him as if a certain tension in his lips prevented the usual play of the mouth. In the attempt to whistle, the lips can not be puckered as well as formerly; the muscles of the cheek have become more rigid and inactive, and as the disease progresses the whole lower half of the face assumes a characteristic appearance—a peculiar lachrymose and astonished expression—which, as is easily seen, is due to the drooping of the lower lip and to the deepening of the naso-labial fold. The upper half of the