ever, by the third branch of the fifth. In other words, at least three cranial nerves are concerned in the motion and fixation of the uvula, and besides, even under normal conditions, the uvula is occasionally found to deviate to one or the other side. The only thing of which we can, perhaps, be sure is that if during phonation paresis of the velum palati and deviation to the sound side becomes apparent, the large superficial petrosal is most likely affected (paralysis of the levator palati and azygos uvulæ). Of greater importance for the diagnosis of central facial paralysis is the persistence of the reflexes, which in peripheral paralysis are often diminished or sometimes completely lost.

Furthermore, the disturbances in hearing, the alterations in taste and in the salivary secretion, so frequently observed in the latter, are almost always absent in central affections.

The existence of a bilateral facial paralysis—diplegia facialis—points as a rule to a central lesion, and more especially to a bulbar affection. It certainly is one of the greatest rarities to have a simultaneous paralysis of both as the result of a peripheral lesion.

Prognosis.—The prognosis depends upon the anatomical basis of the disease. Lesions of the cortex and the pons often bring about facial paralyses that are incurable, while those observed in conjunction with capsular hemiplegias, especially in the early stages of the latter, frequently present a decided improvement after a time. As was stated above, it is impossible in the cases which depend upon a neuropathic predisposition to make any statement either with reference to duration or with reference to a possible recurrence of the trouble.

Treatment.—The question of treatment arises only when the primary lesion is amenable to therapeutic measures. Since this, however, is only very rarely the case, it is best, at least in the central facial paralysis, to restrict ourselves to the expectant treatment. The measures that will be recommended as indicated in the peripheral form are here of very little avail.

B. PERIPHERAL FACIAL PARALYSIS.

In its peripheral course the facial may be divided into two portions—an intracranial and an extracranial. The former is less frequently affected than the latter, which is more exposed to atmospheric influences, especially cold. For practical reasons we prefer to consider the diseases of these two segments separately.

1. *The Intracranial Lesion.*

This form is distinguished by the fact that besides the constant existence of paralysis of all the facial branches, we have

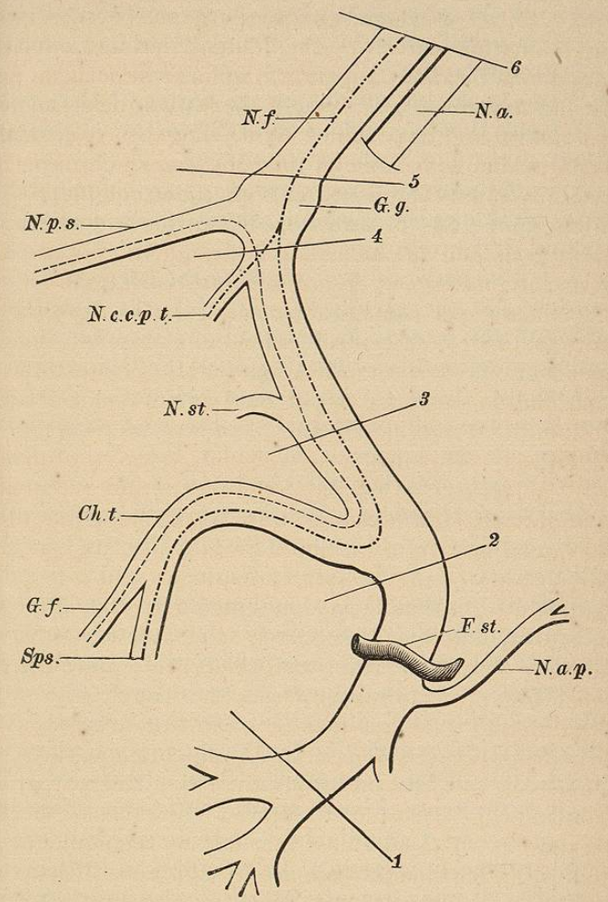

Fig. 15.—Erb's Diagram for Facial Paralysis. Representing the course of the facial trunk from the base of the skull to the pes anserinus. *N. a.*, auditory nerve. *N. f.*, facial nerve. *N. p. s.*, large superficial petrosal nerve. *G. g.*, geniculate ganglion. *N. c. c. p. t.*, communicating branch to tympanic plexus. *N. st.*, stapedius nerve. *Ch. t.*, chorda tympani. *G. f.*, gustatory fibres. *Sps.*, secretory nerve to salivary glands. *F. st.*, stylo-mastoid foramen. *N. a. p.*, posterior auricular nerve.

often certain concomitant symptoms, which can only be fully understood if we picture to ourselves the exact course of the

nerve. This can be done with the help of the diagram (taken from Erb) here represented, which permits an accurate localization of any given intracranial lesion.

(*a*) If the lesion be between the exit of the facial stem (from the pons) and the geniculate ganglion, we shall find a paralysis of the velum palati, abnormal acuteness of hearing, and diminished salivary secretion.

(*b*) If the facial be affected in the region of the geniculate ganglion itself, then we find in addition to the just-mentioned symptoms alterations in the sense of taste.

(*c*) A lesion between the geniculate ganglion and the stapedius nerve produces the symptoms described in *a* and *b*, but no abnormality of the velum palati.

(*d*) A lesion between the origin of the nerve to the stapedius muscle and the giving off of the chorda tympani gives alterations in the sense of taste and diminishes salivary secretion, but no abnormality of hearing or of the velum palati.

(*e*) If, finally, the nerve is diseased below the giving off of the chorda, in the Fallopian canal, we only find paralysis in the distribution of the posterior auricular branch without any trouble with taste, hearing, the condition of the velum palati, or the secretion of saliva. We should state again, however, that in all cases from *a* to *e* all the facial branches take part in the paralysis.

Valuable as this diagram is, undoubtedly, regarded from a theoretical stand-point, yet in practice we but rarely meet with opportunities for observing cases which exactly correspond to it; nevertheless, in every instance we should not fail to attempt to locate the lesion with as much accuracy as possible.

A physiological explanation for the appearance of the above-mentioned concomitant symptoms is not always easy. That alterations in the sense of taste are due to lesions of the chorda tympani can not be doubted, and if they are present the lesion is situated between the geniculate ganglion and the giving off of the chorda; if they are absent the lesion must be sought below this region. The disturbance in the sense of taste is limited to the anterior two thirds of the tongue, and exists, of course, only on the paralyzed side. Sensory changes in the tongue are not necessarily present. Less clear is the cause of the diminished salivary secretion. Its occurrence is said to point to a lesion above the geniculate ganglion (Wachsmuth). Mendel has observed increased salivary secretion in an instance

in which it was also difficult to find an adequate physiological explanation (Neurol. Centralblatt, 1890, 16).

Among the most common and best known symptoms are the disturbances in hearing, which consist either in an abnormal acuteness of hearing (hyperacusis, oxyacoia) or in a decrease in the power of hearing. In the first case, where we have a kind of hyperæsthesia for all musical tones, the alteration is supposed to be due to a paralysis of the stapedius muscle (which is supplied by the facial) and a consequent overaction of the tensor tympani (Lucae, Hitzig, Roux). The latter—the hardness of hearing—can be due to several causes. We may either have a disease of the middle ear and the adjoining portion of the temporal bone, which has affected the facial nerve by contiguity, or a simultaneous affection of the auditory nerve, which, in the internal auditory meatus, has been exposed to the same deleterious influence, and become affected by the same disease as the facial. Quite lately again the frequency of this combination of facial paralysis with a slight paralysis of the auditory nerve has been pointed out by O. Rosenbach (cf. lit.).

2. *The Extracranial Lesion.*

The peripheral paralysis of the facial after its exit from the skull is, as we have already said, the most common. Of this class the so-called rheumatic form, which is attributed to the influence of cold (*a frigore*), and the traumatic, often observed after operations, gunshot injuries, etc., or which may be caused by the pressure of impacted cerumen in the ear and mastoid cells (Dalbey, New York Med. Journal, liv. 3, 1891), are the two chief representatives. When any one, heated as he is, passes from a warm room into a cold wintry night, or is exposed to draughts in the railroad cars, and finds himself a few hours later taken with a paralysis of one side of the face, this is the so-called rheumatic form which has attacked the stem of the nerve after its exit from the Fallopian canal. But the influence of cold in such instances must be regarded only as the precipitating cause in individuals with a neuropathic predisposition (Neumann, Arch. d. Neurologie, July, 1887, xiv, 40).

In these cases all three facial branches are affected, and the appearance of the patient is changed in a very material and striking manner. Even the layman notices that the patient now wrinkles only one half of his forehead, and that the folds and furrows generally present are obliterated on one side; that

he can shut one eye only while the other remains wide open and can not be closed despite the strongest efforts. If the attempt is made, the eyelids remain gaping, the eyeball is rolled inward and upward, and the pupil disappears behind the upper lid, a position which is also maintained during sleep (lagophthalmos). The inability to shut the lids prevents the tears from running into the tear ducts and interferes with the process by which foreign bodies, particles of dust and the like, are removed from the eye. It happens, then, that the tears are always running down the cheeks, and that a conjunctivitis, even an ulceration of the cornea, may be developed through the mechanical irritation caused by such foreign bodies. The appearance of the lower part of the face has already been described. In mild cases the tongue does not deviate at all; in grave cases it is turned toward the well side (Hitzig, cf. lit.).

It is interesting to note that in the first stage of rheumatic facial paralysis the patient often complains of pains the intensity of which seems to be proportional to the degree of the paralysis. These are usually localized in front or behind the ear and radiate toward the forehead, the temple, and the cheek; sometimes they last but a few days, in other cases they persist for weeks. They must be referred to an affection of sensory branches belonging to the trigeminus.

The hyperidrosis associated with facial paralysis, as observed by Windscheid (Münchener med. Wochenschr., xxxvii, 50, 1890), as well as the frequently noted puffiness and the porcelain-like induration of the affected side associated with vascular dilatation and elevation of temperature (von Frankl Hochwart, Deutsch. med. Ztg., 1891, 35), show that vaso-motor fibres are also implicated in facial paralysis. I have observed the appearance of œdematous swelling especially in the recurrent forms. The implication of trophic fibres is shown by the not rare occurrence of herpes zoster, which has recently been described by Letulle, Strübing, Voigt, and Perrin (cf. lit.). Whether this is due to an inflammation of the peripheral endings of the fifth, which is transmitted to the facial (Strübing), or whether the stem of the facial contains in parts fibres, an inflammatory irritation of which may produce herpes zoster (Eulenburg), is not clear.

I have only in rare instances seen this complication, and have found that whenever it was present the cases pursued an unusually protracted course.

Duration and Course.—The duration and course of rheumatic facial paralysis are extremely variable, and it is of great importance for the physician to be able to give at the beginning an approximately accurate opinion as to the length of time necessary for recovery. This we can, however, only do if we investigate the electrical condition of the paralyzed muscles, and hence it follows that it should be our invariable rule to make an electrical examination before venturing upon any expression of opinion. The following are the chief points to guide us:

1. If we find no changes either in faradic or in galvanic excitability the prognosis is favorable; recovery in from seven to twenty days (light form).

2. If we find the faradic and galvanic excitability of the nerve diminished, but not lost, the galvanic excitability of the muscles, however, increased, and the usual formula of contractions changed (A. C. C. > C. C. C.), then the prognosis is relatively favorable; recovery in from four to six weeks (intermediate form of Erb).

3. If the reaction of degeneration be found—i. e., if the faradic and galvanic excitability of the nerve and the faradic excitability of the muscles be lost, while there is an increase in the galvanic excitability of the muscles associated with qualitative changes and changes in the mechanical excitability—then the prognosis is relatively unfavorable, and for recovery two, four, six, eight, even twelve months, may be required (grave form). These are those bad cases in which secondary contractures and spasmodic twitchings of the muscles also appear, which, according to Hitzig's opinion, are to be referred to an obscure abnormal irritation in the medulla oblongata. It is well to know that, as convalescence begins, voluntary motion may return long before the electrical excitability, so that often the patient is able to perform some slight voluntary movements before faradic stimulation provokes the least contraction.

The pathological changes have been studied by Minkowski (Berliner klin. Wochenschrift, 1891, 27), and quite recently by Darkschewitsch and Tichonow (Neurol. Centralblatt, 1893, 10). The latter found a parenchymatous neuritis in the peripheral portion of the nerve, and in the central portion the signs of secondary degeneration, with many perfectly atrophied fibres; in the nuclei also the signs of a well-marked atrophy were present.

Diagnosis.—With regard to the diagnosis there is even for the beginner no more easily recognizable disease. Still, there are cases where it is difficult, not to say whether there is any paralysis, but, strange as it may sound, which is the affected side. One is particularly liable to mistakes in old people, in whom the wrinkled, inelastic skin has produced a stereotyped expression, which, even when the facial muscles contract, is but little changed. Suppose now the muscles to have lost their innervation, the paralyzed side takes on the soft features of an earlier period of life, and this may go so far that the patient believes his rigid, wrinkled side to be the paralyzed, and the affected side the healthy one (Gowers). We also must remember that the non-paralyzed zygomatici pull the face sharply toward the well side, a condition which easily produces in the layman the impression of something abnormal, so that he takes the side thus distorted for the diseased one. In general, however, we may say that the diagnosis of a peripheral facial paralysis is one of the easiest imaginable in neuropathology.

Treatment.—In the treatment we may in recent cases recommend for trial steam-baths and counter-irritation to the skin; but never, unless there is a special indication, should internal remedies be advised, because in a non-complicated rheumatic facial paralysis they are absolutely superfluous. In more protracted cases the methodical use of electricity is strongly indicated, for even though it is undoubtedly true that the disease, if the prognosis is at all favorable, gets well of its own accord, and really requires no treatment at all, there can, on the other hand, be no doubt but that the electrical treatment hastens the cure in a marked degree; therefore, electricity should be used under all circumstances. Just which method should be employed can not be definitely laid down, but we should keep in mind that not only the galvanic current is beneficial, but that the faradic brush applied to the stem and the individual branches of the facial gives good results, and the patient should, therefore, be persuaded to submit to this somewhat disagreeable procedure. The places from which the most important facial muscles can best be stimulated are seen in Fig. 16. At these points the motor-nerve branches to the muscles concerned lie very near the surface. They are called "motor points" (Ziemssen). In galvanization every specialist has his pet method of application and his own ideas about the strength and direction of the current. The one prefers to apply the

electrode over the mastoid process, placing either the anode or the cathode on the affected side of the face; another treats at the same time the sympathetic in the neck; a third, again, applies the anode over the affected nerve and the cathode to an indifferent point, and so forth. Whichever method we may

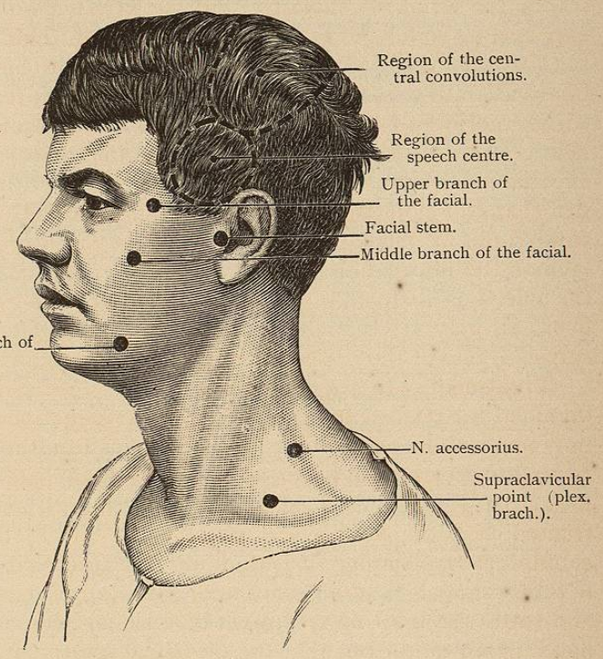

Fig. 16.—Some of the so-called "Motor Points" on the Face and Neck.

prefer, the main thing, after all, is to produce by repeated opening and closing of the current contractions of the muscles by which the tonus of the latter will soon be improved. I should like to mention, too, that I have seen the application of the galvanic brush and the use of the combined current (de Wattewille) repeatedly attended with satisfactory results (Hirt, Lehrbuch, etc., *loc. cit.*, p. 102 *et seq.*).

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CHAPTER VI.

DISEASES OF THE AUDITORY NERVE.

The auditory nerve emerges at the base of the brain, alongside of the facial, and takes with this latter a forward and outward course. After having entered the internal auditory meatus, it divides before reaching the cribriform plate, which separates the internal meatus from the internal ear, into two main branches, an anterior inferior and a posterior superior. These nerves pass as small filaments through the openings in the plate, to be distributed respectively to the cochlea and vestibule, and are hence called ramus cochlearis and ramus vestibularis.

The cortical centre of the nerve is probably to be sought for in the temporal lobe; the fibres are said to run through the last third of the posterior division of the internal capsule, through the middle geniculate body, through the brachia conjunctiva posteriora, the posterior corpora quadrigemina, and the inferior fillet (v. Monakow, Baginsky).

About the situation of the nuclei of the auditory nerve there seems still to exist a difference of opinion among the anatomists. Usually two nuclei are distinguished, an inner or principal nucleus and an outer one situated laterally from the first. In their structure these present material differences. While the former—the inner nucleus—only contains scattered, small, slender, ganglionic cells (15 to 20 μ long), the latter contains cells of considerable size (60 to 100 μ long and 15 to 21 μ broad). The situation of the two nuclei may be understood from the accompanying diagram.

Of the two roots, the superficial terminates in the internal auditory nucleus, while the deeper one passes between the restiform body and the ascending root of the fifth, and turns toward the outer one. This, also, the diagram, which is taken from Wernicke, and which demonstrates the views of Meynert, illustrates.

Although the diseases of the auditory nerve are not, as a rule, treated of in neurological text-books, they are found sometimes so closely connected with other nervous diseases, and