### 3. *Trigeminal Cough.*

Finally, we may call attention to a reflex neurosis, which was first described by Schadewald, and then studied by Wille. This is a paroxysmal cough which, occurring in individuals whose respiratory organs are perfectly sound, is entirely due to an irritation of the trigeminal fibres distributed to the nose, pharynx, and the external auditory meatus. These two writers distinguish accordingly a nasal, a pharyngeal, and an auricular trigeminal cough, and declare the first (nasal) to be the most frequent variety. According to them also, this neurosis is by no means rare, and the possibility of its existence ought always to be thought of where we have to treat cases of an obstinate paroxysmal cough, which is liable to be produced by the action of pungent odors and by changes of temperature, and which is accompanied by hypersecretion of the nasal mucous membrane. The treatment consists in the use of the nasal douche, the application of weak induction currents directly to the nasal cavity, and the administration of potassium iodide. Further observations are still needed to decide whether we actually have to deal in these cases with a neurosis of the trigeminus, or whether the vagus has not something to do with the affection, or whether, finally, as Hack suggested, the erectile tissue of the nose is responsible for it.

Quite lately it has been claimed that peripheral irritation of the trigeminus (by inhalation of pungent vapors, new growths, etc.) may reflexly give rise to sensations of dizziness ("nasal vertigo," Joal). Until more confirmatory evidence is brought forward, it would be well to suspend judgment on this question.

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## CHAPTER V.

### DISEASES OF THE FACIAL NERVE.

The facial nerve emerges at the base of the brain from the medulla oblongata by the side of the abducens and behind the trigeminus on the posterior margin of the middle peduncle of the cerebellum. The auditory nerve is situated close behind it, and between the two a separate bundle of fibres is placed—namely, a second root of the facial, the so-called nervus intermedius or portio intermedia Wrisbergii. With the auditory nerve the facial then passes forward and outward into the internal auditory meatus, at the bottom of which it enters through a small opening the Fallopian canal (cf. Fig. 15). In the hiatus of this canal it makes an almost rectangular turn (genu nervi facialis), passes backward and then downward, and leaves the skull through the stylo-mastoid foramen to divide inside of the parotid gland into the terminal branches, the temporo-facial and the cervico-facial, which form together the plexus anserinus major. At the so-called genu the nerve forms a ganglifform swelling—the ganglion geniculi—from which the larger superficial petrosal nerve is given off (cf. diagram, Fig. 15). These are the fibres which communicate with the trigeminus, and have the function of gustatory fibres for the anterior two thirds of the tongue (cf. page 74).

The nucleus of the facial, a group of large multipolar ganglionic cells, lies four millimetres and a half beneath the floor of the fourth ventricle, in the region of the formatio reticularis, dorsal to the upper olive (cf. Fig. 13). From this illustration it is also apparent that the ascending root of the trigeminus has the emerging portion of the facial root to its mesial side, while the anterior root of the auditory lies external to it. The axis cylinder processes of the ganglionic cells of the nucleus are united in a larger fasciculus, forming the first part of the root (Ursprungsschenkel of Krause), which at the floor of the fourth ventricle becomes a compact bundle, the intermediate portion (VII, *a*). At the anterior end of the eminentia teres this is bent at right angles (genu cerebrale), and becomes the emerging portion (Austrittsschenkel) of the facial (VII), which

reaches its point of exit, before mentioned, through the transverse fibres of the pons.

Quite lately experiments on animals by Mendel have shown that in rabbits and guinea-pigs the facial branches to the eyes take their origin in the oculo-motor nucleus. Whether this is the case or not in man our present pathological observations do not allow us to decide with certainty.

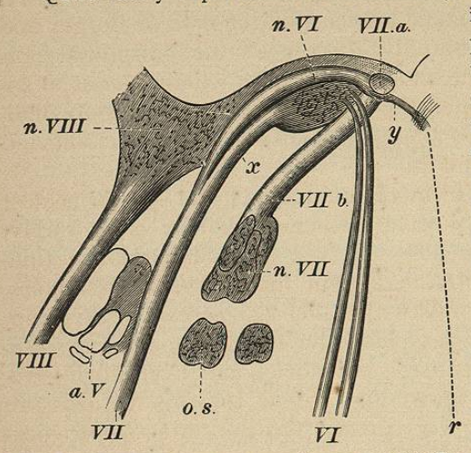


Fig. 13.—Diagram showing the Course of the Facial Fibres in the Pons. (After Schwalbe.) *n. VII*, facial nucleus. *VII b*, root-bundle of the facial nucleus. *VII a*, intermediate portion (cross-section). *VII*, emerging portion of the facial. *n. VI*, abducens nucleus. *n. VIII*, nucleus, and *VIII*, root of the auditory nerve. *y*, fibres coming from the raphe. *x*, fibres coming from the abducens nucleus. *o. s.*, upper olive. *a. V*, ascending root of the trigeminus.

Just as in the case of the trigeminus, so in the facial, we must distinguish between central (cortical and bulbar) and peripheral (intra- and extra-cranial) lesions, which, owing to the purely motor functions of the facial, may give us, clinically, spasm or paralysis. We shall discuss each class separately.

### 1. Facial Spasm—"Mimic Facial Spasm"—"Tic Convulsif."

Lesions which give rise to facial spasm may be central or peripheral in their situation. In the first case either the cortex or the nucleus (or the root) of the nerve in the medulla oblongata is concerned. According to our present ideas the cortical area for the facial is located in the lower half of the anterior and the lower third of the posterior central convolutions, and it is also supposed that the posterior halves of the two lower frontal and the anterior part of the supramarginal convolutions have some, although a less important, connection with it (Exner).

It is not known whether stimulation of these centres can produce a facial spasm, or, in other words, whether there exists a real cortical facial spasm, although the experiences of Cadiot, Gilbert, and Roger (Revue de méd., May 10, 1890, No. 5) seem to leave but little doubt upon this point. It seems, however, well established that the disease can be produced by reflex stimulation of the facial nucleus (cf. the case of Berger and its treatment). Undoubtedly, disease of the peripheral portions of the nerve is the most common, in which, just as in trigeminal affections, either the whole facial area or only individual branches may be affected. We distinguish a clonic and a tonic variety of spasm.



A patient suffering from clonic diffuse facial spasm has lost control over his facial muscles, either on one or, more rarely, on both sides. The muscles affected are in irregular motion, so that against his will the patient makes the oddest faces, wrinkles his forehead, raises the alæ nasi, screws his eyes up, etc. When the attack has passed he has a temporary respite, yet often enough the pause is very brief, and even during remissions spasms flash across his face, so that his features are never for any time entirely at rest. On the slightest provocation, by speaking, often also by eating, quite violent paroxysms are excited, so that the patient would fain cover up his distorted face.

If the spasm is tonic, the affected side of the face is singularly rigid and takes no part in the facial movements, but is distorted. The muscles are distinctly hard to the touch, the corner of the mouth is pulled toward the diseased side, the mouth firmly closed, the eyebrow drawn up—signs sufficiently marked to distinguish it from facial paralysis, in which also the affected side does not take part in the movements of expression. Vaso-motor and trophic changes are, as a rule, absent.

In cases where the spasm is confined to some branches of the facial only, we find that the muscles around the eyes are almost always the ones affected. The eyelid is attacked by a clonic or tonic spasm, and conditions are developed which go under the names of spasmus nictitans and blepharospasm.

The *spasmus nictitans* consists of spasmodic blinking, in which not only the eyes are rapidly closed and opened, but also the neighboring muscles (frontalis, zygomatici) participate in the spasmodic movements. In a mild form this spasm is seen in many people where it is only to be regarded as a bad habit.

*Blepharospasm* consists of a paroxysmal spastic contraction of the orbicularis palpebrarum, lasting a few seconds or minutes, which completely closes the lids. In rare cases the attacks

follow each other so quickly and are so prolonged that the patient has to be treated as a blind man; even a transient amaurosis has actually been observed (Silex, Klin. Monatsbl. f. Augenheilk., März, 1888). The attacks appear unexpectedly and quick as lightning. They are often precipitated by voluntary firm closure of the eyes, eye-strain, or by the action of light, and the patient is utterly unable to raise the lid until the attack has passed. The physician, however, will succeed at times in cutting short the paroxysm if he be able to discover any of the so-called pressure points, which, according to von Graefe, who first discovered them, are often present. More or less firm pressure exerted at these points is capable of producing an interruption of the spasm and a cessation of the attack. Unfortunately, however, such points are often entirely absent, and when they exist their position is so uncertain and changeable that they may only be accidentally discovered. One of the few which is present with some constancy corresponds to the supra-orbital foramen. We should, however, look for them over the whole distribution of the trigeminus, over the spinous and transverse processes of the cervical vertebræ, and even in the region of the brachial plexus. It is our duty to make a frequent and untiring search for them, as we may thus be able to afford our patients very great relief.

**Course.**—The course of the disease, be it in the form of a total or a partial spasm, is usually very tedious, and a prognosis for recovery must be very guarded. The outlook is especially unfavorable when the affection is complicated with other motor disturbances, as I have observed, for instance, in two cases where the facial spasm was associated with writer's cramp. Of late a number of cases have been observed in which various motor disturbances were associated with tic convulsif; these conditions have been described as a new disease under the name of la maladie des tics convulsifs. We shall have occasion to speak of them in our chapter on hysteria.

**Ætiology.**—We know little about the ætiology of blepharospasm. That it may be of reflex origin can not be doubted. The most varied diseases of the eyes, affections of the nasal mucous membrane, or of the trigeminus, especially tic douloureux, carious teeth, intestinal worms, or uterine troubles, may lie at the bottom of it, and the origin of the disease is cleared up only if, after removal of some primary cause, the spasm



suddenly ceases. An examination of the nose should never be neglected. It has repeatedly been noted that the tic disappeared after swellings or tumors of the mucous membrane of the nose had been removed (B. Fränkel, Peltesohn).

I saw a case of blepharospasm, which had persisted for years and was considered hopeless, cured after a coexisting flexion of the uterus had been materially improved. Diseases of the blood-vessels can, furthermore, produce the spasm, as we see from the case of Buss (cf. lit.), where an atheromatous artery, and from that of F. Schultze, where an aneurism of the left vertebral artery by pressure upon the facial nerve brought on the spasm. Finally, hysterical conditions can lead to it, as is shown by the latest communications of Charcot on the so-called hemispasmus glosso-labialis, which has been described by Marie (Progrès méd., June 6, 1887).

**Treatment.**—All these points we must keep in mind in deciding upon a line of treatment, and not imagine that we can cure a facial spasm, whether it be total or partial, clonic or tonic, with indiscriminate galvanization, for without system we shall only meet with success in rare cases, and then only by good luck. The most promising plan of treatment is the application of the anode to pressure points if such be present, while the cathode is placed on some indifferent region, the back of the neck or the sternum. Weak currents applied for one or two minutes, with careful avoidance of make and break, give the best results. The application of the anode to the back of the head, keeping it at the same point, also sometimes meets with success (Berger), but too often leaves us in the lurch; and this will hardly surprise us if we remember that even when the anode is placed on the back of the neck the abnormally stimulated reflex centre in the medulla is by no means always reached by those curves of the current which really do penetrate deeply.

Cures, such as that reported by Berger, undoubtedly depend upon a happy coincidence of circumstances. The medulla oblongata, above all, where in such an astonishingly small space a number of the most important nuclei lie close together, seems to be the most unfavorable place for local electrization (Duchenne), by which we aim at affecting individual nerves or nerve roots. We may reach all or none, no matter whether we use small or large electrodes. Still, even this method ought to be tried, since we have no positive remedy. Should the gal-

vanic treatment fail, the internal treatment is still more vague, and it is well to inform the patient of the uncertainty of this procedure. Of course, the usual nervines and antispasmodics are to be given. Hammond has seen especially favorable results from the use of coniin and atropin (Med. Record, No. 41, September, 1892). As a last resort, neurectomy of the supra-orbital or stretching of the facial nerve (Bernhardt, cf. lit.) has to be considered, yet even from this we can expect no lasting success.

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## 2. Facial Paralysis—Mimic Facial Paralysis—Hemiplegia Facialis—Prosopoplegia.

Facial paralysis is an affection the relative frequency of which makes it of the greatest practical importance. In this more than in any other nervous disease any layman can easily judge just how much the art of the physician has accomplished in a certain time in a given case, and on this account it especially behooves us at our first examination to be very careful in making a positive statement as to the prospects of recovery or the probable duration of the disease. Both these points depend chiefly on the seat of the lesion, which, as in tic convulsif, may be central or peripheral.

### A. CENTRAL FACIAL PARALYSIS.

**Symptoms and Diagnosis.**—Central facial paralysis may be produced either by a cortical lesion (cortical paralysis) or by a lesion of the facial fibres in the brain between the cortex and the pons (intracerebral paralysis *par excellence*); or, finally, it may depend upon a disease of the nuclei and nerve roots in the pons. Cortical facial palsies may be caused by tumors, abscesses, or chronic inflammations in the region of the motor centres. Those of intracerebral origin may be produced by syphilitic arterial disease or by rupture of a vessel in the region of the internal capsule and the crus cerebri. The third



form, that originating in the pons, is found in Duchenne's paralysis and, more rarely, in tabes. There exists a form of facial paralysis the pathology and the seat of which is as yet very obscure, and we can only say that probably a "nervous predisposition" is necessary for its development. It may occur in several members of the same family, may be congenital, and may be associated with paralysis in the region of the sixth nerve and of the trigeminus. It sets in without any appreciable cause, is wont to recur, and may last for an indefinite period of time; probably its anatomical seat is in the nucleus ("infantile degeneration of the nucleus," Möbius), but, as was said above, this is by no means proved. It is quite possible that some cases may have a peripheral origin, as is the case in the recurrent oculo-motor paralysis (cf. page 50).

The clinical picture differs but little in these three forms, and only at times do the accompanying symptoms make a differential diagnosis possible. Thus, for instance, the intracerebral paralysis often appears with an apoplectic attack, and is accompanied by hemiplegia and speech disturbances, while if facial paralysis is found in connection with spinal disease it is always of nuclear origin.

All three forms of central paralysis have usually, however, two features in common which can almost be regarded as pathognomonic and which distinguish them from the peripheral paralysis, namely: 1. The presence of a normal electrical excitability in the nerves and muscles to both currents. 2. The escape of the upper facial branch. While in peripheral paralysis all three divisions are equally affected, we find in the central form the upper branch usually intact, and the patient can wrinkle his forehead and close both eyes.

We say usually, not always, because there are undoubtedly exceptions, where we meet with a central paralysis in which the upper branch has not been spared. It is quite probable that the naso-labial and the orbiculo-frontal fibres of the facial have a separate cortical origin, and we can well imagine that if the cause of the paralysis—e. g., a small focus in the cerebrum—is situated above the union of those two branches, one remains intact (in the large majority of cases the upper), whereas if it is below their point of union both branches are affected.

A further guide to localization is the condition of the movements of expression (Bechterew). If these are lost while the voluntary innervation of the facial muscles is intact, we have to

assume a focus in the optic thalamus, the centre for facial expressions, or close to it (Bechterew), while a facial paralysis with retained power of facial expression allows us to exclude a lesion in the thalamus and its coronal connection with the hemispheres. In the case of Rosenbach (Neurol. Centralblatt, 11, 1886) there was an isolated paralysis of mimic expression in the left facial and right-sided bilateral hemianopia, and the lesion was taken to be in the right thalamus.

In differentiating between a cortical and a bulbar facial paralysis the following points must be taken into consideration: That the lesion is cortical is probable if the facial alone without the corresponding half of the body is paralyzed (monoplegia facialis), and if the affection is confined to the lower branches of the nerve, while the normal reaction to the electrical current remains undisturbed. It is easy to understand that the hypoglossus often takes part in the lesion if we remember in how close proximity the centres of the two nerves are situated in the cortex, and in a given case an examination of the mobility of the tongue will show whether we actually have to deal with a so-called monoplegia facio-lingualis. Sometimes a disturbance of speech points at once to this combination. In every case in which we assume a cortical lesion, the sensation in the distribution of the facial and the hypoglossus ought to be tested, because it is just in these cases that we find not infrequently sensory changes—e. g., analgesias and anæsthesias.

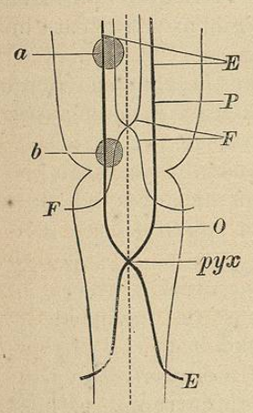


Fig. 14.—Diagram showing the Decussation of the Fibres going to the Extremities, and of those going to the Face, in the Pons and Medulla Oblongata. *F*, facial fibres. *E*, fibres going to the extremities. *P*, pons. *O*, medulla oblongata. *pyx*, decussation of the pyramidal tracts. *a*, a focus in the upper, *b*, a focus in the lower, part of the pons (the latter is situated below the decussation of the facial fibres).

We shall be led in a facial paralysis to think of an affection of the pons when not only the nerve, but with it one whole half of the body is paralyzed; and there are two types of pontine facial paralysis according as the lesion is situated in the upper or lower part of the pons. In the first case (focus *a* in Fig. 14) the facial and the same, in the second (focus *b*) the



facial and the opposite half of the body are affected (hemiplegia alternans, Gubler, 1859), because the facial fibres cross in the pons and we may have a lesion above or below this crossing, and in both cases this will be situated, of course, above the crossing of the fibres going to the extremities.

The facial paralysis caused by the lesion in the upper part of the pons, and that found in connection with hemiplegia after a lesion in the internal capsule, are in so far alike as they are both accompanied by paralysis of the extremities on the same side. But there is one point of difference which will influence our diagnosis, namely, that after pontine lesions the facial paralysis, very much as in the peripheral form, takes in all three branches of the nerve, while in a lesion of the capsule or the basal ganglia only the lower branches of the nerve are affected; but in contradistinction to what happens in the peripheral paralysis the electrical condition may, at least in some cases, remain normal.

The most striking symptom of central facial paralysis is the relaxed and expressionless appearance of the affected side. The naso-labial fold is more or less distinctly flattened, the corner of the mouth is slightly open and hangs down, the mouth seems to be drawn to the well side, the patient is unable to raise his upper lip or to whistle. On inflating the cheeks the air escapes; drinking and speaking are difficult, the latter especially, because the labial sounds are defectively formed. During eating the food gets in between the cheek and the teeth on the affected side, and the patient has to bring it to the right place again with the fingers. In biting, the mucous membrane of the cheek is often caught between the teeth. The upper part of the face is in by far the greater number of cases normal; the forehead can be wrinkled well in its whole extent, and the patient can frown and close either eye perfectly.

The condition of the velum palati and the uvula varies, and is, therefore, of no value, either diagnostically or prognostically. The uvula may deviate to the sound or to the affected side, or may occupy its normal position. With our still imperfect knowledge of the innervation of the muscles concerned, any attempt to explain the different positions of the uvula must needs be hard, but we shall be less surprised at our difficulty when we consider that the levator palati is supplied not only by the facial through the large superficial petrosal, but very probably also by the vagus accessory, the tensor palati, how-