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## The Functional System of the VII cranial nerve

The VII cranial nerve, also called facial nerve, is one of the 12 cranial nerves. It originates at the level of the bulbo-pontine sulcus and crosses the temporal bone through the Fallopian canal, it runs from the internal acoustic meatus up to the stylomastoid hole, leaving then the skull.

The injuries of the VII c.n. can be: central (or upper motor neurone) and peripheral (or lower motor neurone). When the lesion is central, the musculature of the upper quadrant on the contralesional face will be spared because it is doubly represented in the cortex. In fact, the weakness of the mouth while smiling (activity of the lower quadrant), is one of the elements to recognize the stroke. In upper motor lesions the wrinkle of the forehead and the closure of the eyes should be preserved.

On the other hand, peripheral lesions involve, in a greater or less degree, the whole ipsilateral side of the face.

Along the course of the fibers of VII c.n. we can distinguish three different levels of vulnerability and correlate them with the most frequent pathogenic causes:

 Endocranial tract, where we find central lesions such as meningitis and vascular disorders (e.g. stroke) in addition to other peripheral lesions, downstream of the motor nucleus, such as benign tumors known as neurinomas of VIII c.n. which, besides compressing the VII c.n., invade the ponto cerebellar angle, determining important neurological deficits;

- Intratemporal tract, are the most frequent injuries, caused by ear infections, fractures of the petrous bones, carcinomas of the middle ear, Ramsay Hunt syndrome type II or Bell's palsy;
- Extracranial stroke, injuries caused by traumas or facial injuries and diseases of the parotid like neoplasia or mumps.

The level of the lesion allows the clinician to orientate himself on a more or less positive prognosis of nerve recovery. In the case of peripheral lesions, upstream lesions take longer to recovery. Non-motor symptoms, which do not involve muscle activity, are pathognomonic from higher lesions (see **Fig. 2**).

The figure 1 shows the VII cranial nerve and its ramifications which perform motor and non-motor functions. We can summarize them in 4 categories:

- Somato-motor function: innervates the mimic muscles of the face (see Fig.1), modulates the intensity of acute sounds ensuring adequate tympanic tension (stapedius muscle) and it has a minor role in lingual motility (posteriore digastric muscle);
- Visceral-motor function: innervates the glands to lubricate the eyes, the mouth and the nose (Fig. 1);
- Visceral-sensory function: it conveys the tactile and gustatory sensitivity in the 2/3 anterior part of the tongue (Fig.1);
- Somato-sensory function: collects the tactile sensitivity (esteroceptive) of the skin of the auricular cone, of the external acoustic meatus and the eardrum (Fig.1).

A typical symptom reported by patients, which does not involve the muscles but the glands, is the presence of ocular dryness. This typical deficiency, which alters the visceral-motor functioning of the VII c. n., results from the damage to the large superficial petrosal nerve, branching of the facial nerve at the intratemporal level.

Peripheral facial nerve paresis, compromising the upper quadrant of the face, are reported to the ophthalmologist. The prevention of corneal lesions is a therapeutic priority since the paresis of the orbicularis muscle of the eye (paralytic lagophthalmos) exposes the eye to external aggressions and limits the containment of the tear film, facilitating its evaporation.

Using any taping, for example, to reduce the laxity of the eyelid rim must be agreed with the ophthalmologist because it improves the containment of tear (natural and / or artificial) and the distant vision. Instead, the taping for complete eyelid closure at night is almost always recommended.

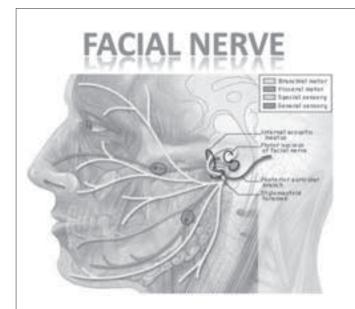
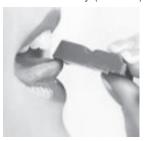


Fig. 1 Facial nerve functions: motor and non-motor activities.



Visceral-sensory (Afferent)







Somatic-motor (Efferent



Somatic-sensory (Afferent)

The patients who have affected the lower quadrant of the face are more likely referred to rehabilitation therapists, mostly, to speech therapists.

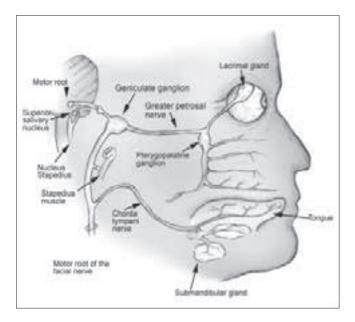


Fig. 2: Facial nerve and his branches.

In addition to not motor symptoms, we find distortion of taste (also called dysgeusia: alteration of the somatosentitive function of the VII c. n.) and dryness in the mouth because of the less production of saliva by the submaxillary glad (also called xerostomia: alteration of visceral-motor function of the VII c. n.). The patient often reports "metallic taste in the mouth" and "kneaded mouth" respectively.

The non-motor symptoms mentioned above interfere with the first step of digestion that happens into the oral cavity. Furthermore, the weakness of the facial muscles (i.e. orbicularis oris and buccinator) makes much more difficult to content the food to chew it before being swallowed.

Furthermore, removing food debris from the lower vestibule while eating is difficult for the patient.

The oral muscles cooperate with the movements of the tongue, innervated by the hypoglossal cranial nerve (XII c.n.), and with the mastication muscles, innervated by the trigeminal cranial nerve (V c.n.).

This is an example of what mean the authors, Lotter M. and Quinci A., with Super functional system of the head and face (2012). This system talks about the cooperation between the different cranial nerves for performing daily activities. In the example reported, the V, VII, XII cranial nerves work synergistically to carry out the macro function of "chewing & swallowing".

In addition to the difficulties mentioned above, poor articulation of phonemes such f, b, p are much common as well.

After facial palsy, the patient can find difficult not only while eating but also while introducing the food, overall if the patient tries to bite food instead of cutting it in small pieces.

At the same time, the patient can be imitated while performing oral hygiene, for instance, uncovering superior teeth to brush them or rinsing and expelling the mouthwash.

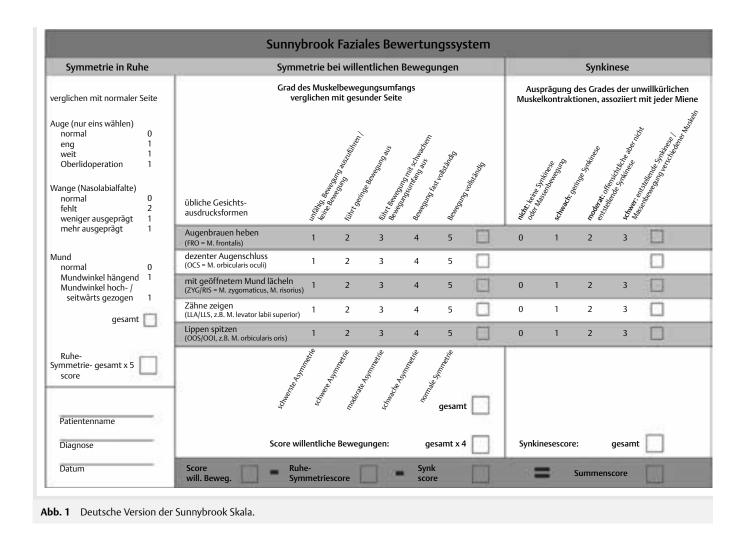


Fig. 3: German version of SFG-S (Neumann T. et al., 2016)

The motor profile of the patient must always be related to the altered functions which are performed by several structures coordinated with each other. Talking only about single muscles could be reductive.

Part of the disorders described above are reported by the patient during the preliminary interview. The moment of the interview goes beyond the anamnesis. It is a moment of active listening in which the therapist recognizes the potentially compromised functional systems, deducing as well the level of injury.

Furthermore, it represents a moment of direct observation of the patient's non-verbal behavior. It lets the examiner to observe what the patient does not say in words but says by gestures about his own experience of illness, e.g. the communicative effectiveness of its expressive mimicry and possible compensation and simulation strategies such as covering the face with the hand.

After the preliminary interview the evaluation should include at least one standardized scale. The Sunnybrook Facial Grading System (SFGS) is one of the most employed scales to assess the facial function by clinicians (Fig.3). It was introduced in 1996 by Ross B.G. and in the later 2016 was translated in German by Neumann T. In 2010, Neely J.G. have introduced the explanatory criteria for reducing potential ambiguities while scoring.

The SFG-S gives the clinician information about the regrowth of the damaged fiber. It is quantify from 0, for complete facial paralysis, to 100, for normal facial function.

This instrument analytically evaluates the muscles from the both quadrants, the upper and the lower quadrant. It is more suitable for peripheral lesions instead of central lesions.

This assessment considers 3 different sections: 1) the resting symmetry by comparison of the affected side with

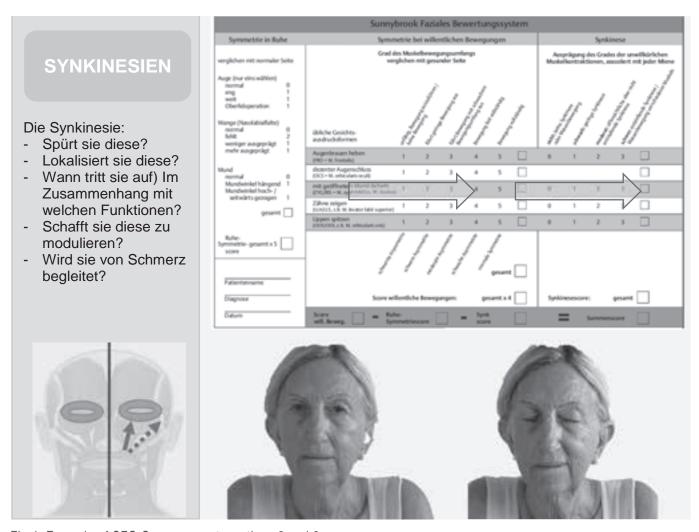


Fig.4: Example of SFG-S assessment: sections 2 and 3.

the normal side at rest, 2) the dynamic assessment that includes five standard expressions (wrinkling the forehead, closing eyes gently, snarling, smiling with the mouth open and puckering the lips) and 3) a section for assessing the degree of synkinesis.

It is well practice to fill in the 3rd section when the patient has produced each one of the items included in section 2. It is less time consuming and less tiring for the patient increasing his/her test compliance.

During the test the patience is seated, looking directly at the horizon without wearing artificial elements, like glasses or make up, that can cover the face.

It is advisable to ask the patient for a photo-card prior to the injury onset. If possible, ask him/her photographs and videos showing dynamic expressive patterns as well. This material will allow the therapist to identify any physiological asymmetries that the patient already had before the injury.

When completing section 3, ask the patient if he/she can identify and eventually located any unwanted activity in the face. Synkinesis are unwanted intrafacial movements that can be observed in the affected face during facial expression asked in section 2.

Like the **figure 4** shows, the patient when closing gently the eyes experiences on the affected side a syngenetic movement such as a smile (intensity=2/3) during the volitional movement of the eyes (intensity= 5/5). Because of this synkinetic movements, patients experience hypertonic contractures when resting and tightness during volitional or reflexive eye closure and/or volitional mouth movements.

The patient is not completely aware of this phenomenon, bringing up the patient's awareness of those beside movement is part of the therapist's goal. The therapist puts the patient in the condition of increasing the attention on those involuntary movements and on the relationship between them and the volitional movements.

The hyperkinesis is usually presented on the non-affected side; the patient tends to exaggerate the mimicry making worse the synkinesis pattern on the affected side and the facial symmetry.

The abnormal regeneration of facial nerve fibers to not related facial muscles is one of the most distressing and adverse consequences of facial palsy. Facial synkinesis comes out more often when the damage is moderate-severe losing the endoneurial tube of the nerve wire. In this case the nerve "gets lost" losing the right way out to his muscle targets. Synkinesis could appear from 3 months to 2 years after the onset, covering all the time span of the nerve recovery. They are hardly reversible.

The neurophysiological recovery of the nerve fiber can be distinguished in 3 phases:

- 1. Complete denervation (wallarian degeneration)
- 2. Initial nerve regeneration with initial motor activity and possible synkinesis
- 3. Nervous network recovery

During the denervation phase the communication between nerve-muscle is interrupted. For this reason, even if the facial muscles are healthy, do not receive the electrical impulse from the nerve blocking their expressions.

This is why the therapist should not encourage the patient to strain the facial muscles and to exaggerate facial expressions trying to train the affected side; the result will be a lack of symmetry and a delay in the recovery of the nerve.

A physical therapist specialist will tailor the most adequate rehabilitation program.

Own awareness of facial movement is poor because the absence of spindles or receptors in the tiny muscles of the face unlike the skeletal muscles. From the patient's testimony emerges a lack of perception of paralysis. Oftentimes, the patient takes awareness of the paralysis onset from external visual feedback (e.g. seeing the drooping corner of the mouth while looking at the mirror) or by the impossibility to carry out an action (e.g. drooling while drinking from a glass).

According to a neurocognitive approach, the body is as a receptorial surface capable, through its fragmentation, of transferring to the Central Nervous System the necessary information about the world (Perfetti, 1985).

The collection of sensory information is guaranteed from the adaptation of the receptorial surface to the object. It is possible by the the V c.n. and the VII c.n. cooperation. For example, the patient is no longer able to recognize the size of the gulp introduced into the mouth or when shaving, he is not longer feeling the same sensation, cause the skin could not adapt to the razor.

The assessment of the perception of the face investigates:

- How the patient constructs the midline: e.g. if he can perceive the filter column, axial element of the face between the nose and the upper lip that reflects the degree of symmetry or muscular balance between the right and left side of the face:
- Contact/touch extension and location: This test can be carried out by using different length rules in correspondence with the morphological lines of the face (e.g. along the nasal labial fold, Fig.5);
- Tactile sensation by confronting both sides, right and left, particularly in the area:
- supra-ocular: this area is innervated by the ophthalmic nerve, upper branch of V n.c.
- inter-ocular-oral: this area is innervated by the maxillary nerve, intermediate branch of V n.c.
- infra-oral: this area is innervated by the mandibular nerve, inferior branch of the V n.c. (Fig. 6)
- Recognition of spatial positions: the therapist guided passively the skin & muscles in different positions and directions. Using symmetrical movements is recommended.



Fig.5:
Evaluation of the extension and location of the contact in the nasal labial fold



Fig.6: Evaluation of the tactile sensation by confronting both sides in the infraoral area

After an exhaustive evaluation, the therapist interprets the information to build the therapeutic program. A useful strategy to choose adequate exercises is to match the degree of the exercises according with the phase of the nerve recovery:

 First level exercises in denervation phase: the therapist performs the movements for the patient due to the lack of activation of the facial musculature. The mean objective is to reduce the compensatory hyperkinesis on the non-affected side.

- · Furthermore, educating the patient to manage the outcomes is necessary (e.g. teaching how protect the affected eye, drinking from the glass and not from the straw. ecc).
- · Second degree exercises when the motor activity is emerging: the therapist assists the patient during the muscles activation. The motor unit's recruitment has to be subliminal, avoiding synkinesis on the affected side and decreasing the hyperkinesis on the non-affected side.
- · Third degree exercises during the final network recovery: the patient performs facial movements without assistance. In this phase, the exercise requires to coordinate multiple movements to complete an action such as feeding, verbal communication, spontaneous mimicry as a support for verbal language (i.e. prosody). This phase puts an end to the "pocker face" (not attivation of the face muscles) and the patient can let go his emotions. The treatment is aimed at the return of communication skills, when the patient own feelings and the movements produced finally match each other.

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