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FACIAL NERVE NEUROPATHIES: CLINICAL-TOPOGRAPHIC CLASSIFICATION, ETIOLOGICAL TYPES, AND PRINCIPLES OF CLINICAL DIAGNOSIS

Pulatova Maftuna Baxtiyarovna
Kimyo International University in Tashkent (KIUT)
3rd-year student, General Medicine
Pulatova06@icloud.com

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Abstract: This article examines facial nerve neuritis (facial paralysis), one of the most common disorders of the peripheral nervous system. The anatomical localization of the facial nerve is described, with particular emphasis on its passage through the Fallopian canal and the role of this anatomical feature in the development of the disease. The article provides a detailed classification of the main clinical types according to etiology, including idiopathic (Bell's palsy), infectious, vascular, and mechanical forms. In addition, the principles of clinical diagnosis are presented, focusing on the identification of the condition and establishment of a clinical diagnosis using simple facial movement tests without the need for complex instrumental examinations.

Keywords: facial nerve, neuritis, Fallopian canal, Bell's palsy, etiology, clinical facial movement tests, facial asymmetry.

INTRODUCTION: ANATOMY OF THE FACIAL NERVE

The facial nerve (VII cranial nerve, n. facialis) consists of two nerves: the proper facial nerve, formed by the axons of motor neurons, and the intermediate nerve (n. intermedius), which contains sensory and autonomic fibers. The nuclei of the solitary tract (nucleus tractus solitarii), the motor nucleus of the facial nerve (nucleus nervi facialis), and the superior salivatory nucleus (nucleus salivatorius superior) are located in the region of the pons.

The facial nerve emerges from the base of the brain at the posterior border of the pons, lateral to the olive, and enters the internal acoustic meatus together with the vestibulocochlear nerve. Within the pyramid of the temporal bone, the nerve passes through the facial canal (Fallopian canal) and exits the skull through the stylomastoid foramen.

Within the facial canal, the nerve forms the geniculate bend (geniculum) and the geniculate ganglion (ganglion geniculi). The geniculate ganglion consists of pseudounipolar cells and belongs to the sensory component of the intermediate nerve.

While passing through the facial canal, the facial nerve gives rise to the following branches:

Branches of the Facial Nerve Within the Facial Canal

Greater petrosal nerve (n. petrosus major) consists of parasympathetic fibers. It exits the facial canal through the hiatus of the greater petrosal nerve onto the anterior surface of the petrous part of the temporal bone, then runs along the corresponding groove and leaves the cranial cavity through the

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foramen lacerum. Together with sympathetic fibers, it enters the pterygoid canal, where it is known as the nerve of the pterygoid canal (n. canalis pterygoidei) and joins the pterygopalatine ganglion.

Chorda tympani (chorda tympani) contains parasympathetic and sensory fibers. The sensory fibers originate from the processes of the pseudounipolar cells of the geniculate ganglion. Its peripheral fibers form taste receptors located in the anterior two-thirds of the tongue and the soft palate. The nerve passes from the facial canal into the tympanic cavity through the canaliculus for the chorda tympani. It then leaves the tympanic cavity through the petrotympanic fissure (fissura petrotympanica) and joins the lingual nerve.

Nerve to the stapedius (n. stapedius) is a motor branch that innervates the stapedius muscle within the tympanic cavity.

Motor Branches of the Facial Nerve

After exiting through the stylomastoid foramen, the facial nerve gives rise to the following motor branches

Posterior auricular nerve (n. auricularis posterior) innervates the occipital belly of the occipitofrontalis muscle and the auricular muscles.

Digastric branch (r. digastricus) innervates the posterior belly of the digastric muscle.

Motor Branches of the Facial Nerve

Stylohyoid branch (r. stylohyoideus) innervates the stylohyoid muscle.

Afterward, the facial nerve enters the parotid gland, where it divides into several branches and forms the parotid plexus (plexus intraparotideus). The following branches arise from this plexus:

Temporal branches (rr. temporales) innervate the auricular muscles, the frontal belly of the occipitofrontalis muscle, and the orbicularis oculi muscle.

Zygomatic branches (rr. zygomatici) innervate the orbicularis oculi muscle and the zygomaticus major muscle.

Buccal branches (rr. buccales) innervate the zygomaticus major and minor muscles, levator labii superioris muscle, buccinator muscle, levator anguli oris muscle, orbicularis oris muscle, as well as the nasal and risorius muscles.

Marginal mandibular branch (r. marginalis mandibularis) innervates the muscles that depress the lower lip and angle of the mouth, as well as the mentalis muscle.

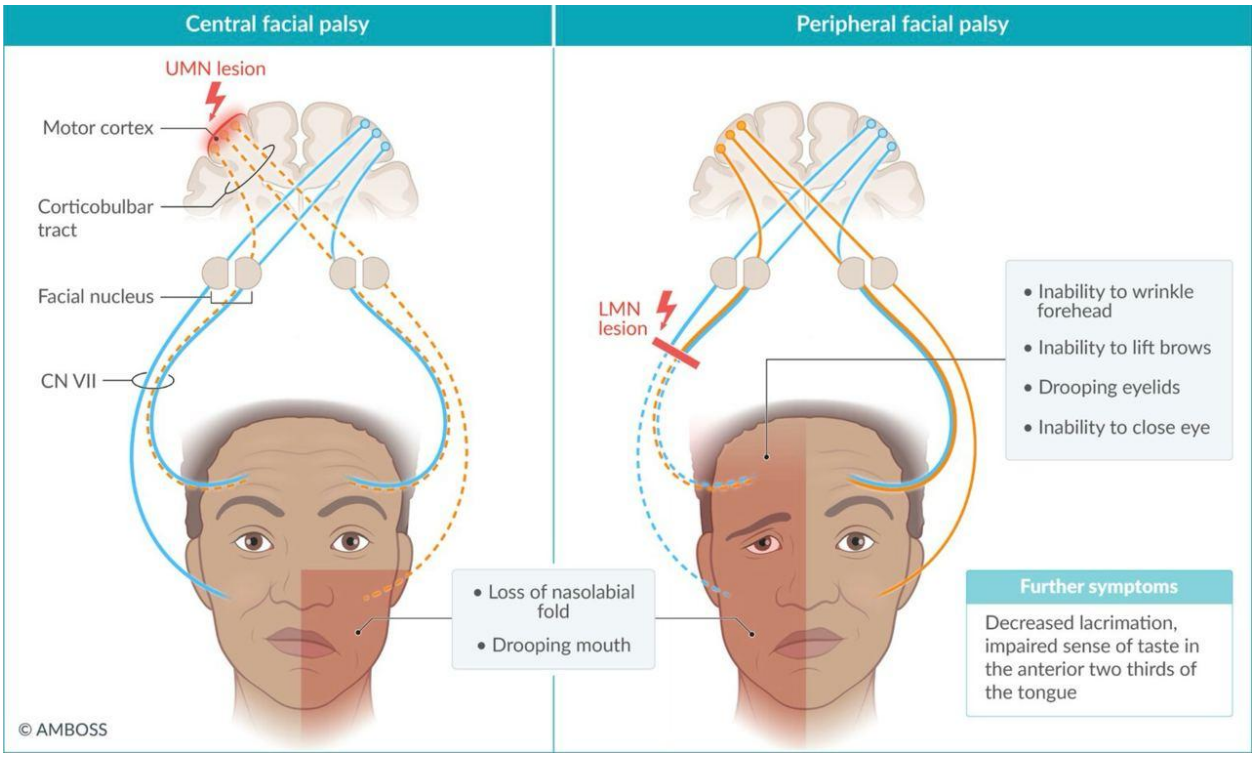
Cervical branch (r. colli) innervates the platysma muscle.

Why Is the Facial Nerve Particularly Susceptible to Injury?

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The reason lies in the fact that the Fallopian canal is very narrow. If the nerve becomes even slightly swollen for any reason, it can quickly become compressed against the bony walls of the canal. As a result, the transmission of nerve impulses along the facial nerve may be impaired, leading to temporary facial paralysis (loss of facial muscle movement).

ETIOLOGICAL TYPES



CENTRAL PARALYSIS

Causes:

Usually caused by damage to the central nervous system, such as a stroke (in the right or left cerebral hemisphere), brain tumors, or traumatic brain injury.

Symptoms:

Facial paralysis is partial, affecting only the lower part of the face, such as the lips and jaw.

Movements of the eyes and forehead are preserved; for example, the patient can wrinkle the forehead or close the eyes.

Symptoms occur on the contralateral side: if the right cerebral hemisphere is affected, symptoms appear on the left side of the face.

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Mechanism:
The muscles of the upper face receive bilateral cortical innervation, which allows movements of the forehead and eyes to remain preserved in cases of central nervous system lesions.

PERIPHERAL PARALYSIS

Causes:
Direct damage to the facial nerve, such as Bell's palsy, infections (e.g., herpes simplex), tumors, or traumatic injuries.

Symptoms:

Complete paralysis of one side of the face, involving both the upper and lower facial muscles.

Inability to close the eye or wrinkle the forehead.

Loss of movement of the lips and jaw.

Dry eye due to impaired lacrimal gland function.

Drooping of the corner of the mouth.

Mechanism:
In peripheral nerve injury, the facial nerve is affected along its course, resulting in paralysis of both the upper and lower facial muscles.

BELL'S PALSY

Main Causes (Etiology)

Viral infection: Reactivation of the latent *Herpes simplex virus type 1 (HSV-1)* within the facial nerve, particularly when immune function is weakened.

Hypothermia (cold exposure): Exposure to air conditioning or cold drafts may cause vasospasm of the blood vessels supplying the facial nerve.

Anatomical factor: The facial nerve passes through the narrow bony Fallopian canal, so even slight nerve swelling can lead to compression, resulting in a tunnel-syndrome-like mechanism.

Clinical Manifestations

Symptoms usually develop suddenly, most often within 24–48 hours, and affect one side of the face:

Facial asymmetry: Drooping of the corner of the mouth with deviation toward the healthy side.

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Lagophthalmos ("rabbit eye"): Inability to completely close the eye on the affected side, accompanied by excessive tearing or dryness.

Loss of forehead wrinkles: Inability to raise the eyebrow on the affected side.

Additional symptoms: Pain behind the ear and impaired taste sensation in the anterior part of the tongue.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Clinical tests: The patient is asked to raise their eyebrows, close their eyes tightly, and show their teeth.

Differentiation from stroke: In a stroke (central paralysis), the forehead muscles remain functional, allowing the patient to wrinkle the forehead. In Bell's palsy, the entire affected side of the face, including the forehead, is paralyzed.

Prognosis

In 80–85% of patients, facial movements recover completely within 1–3 months when appropriate treatment is initiated promptly and correctly.

INFECTIOUS NEUROPATHIES

Herpes simplex virus (HSV-1): It is considered one of the most common infectious causes of Bell's palsy. The virus may reactivate and spread along the nerve, causing acute inflammation and edema.

Varicella-zoster virus (VZV): Facial nerve neuropathy caused by this virus is known as Ramsay Hunt syndrome. It is characterized by facial paralysis accompanied by painful vesicular eruptions (herpes zoster) on the auricle and external auditory canal. This type of

neuropathy generally has a more severe clinical course and may be associated with a less favorable recovery than Bell's palsy.

Epstein-Barr virus (EBV): In the setting of infectious mononucleosis, EBV may cause unilateral or bilateral autoimmune inflammation of the facial nerve.

Assessment of the Degree of Facial Nerve Involvement (Facial Asymmetry)

In clinical practice, the House–Brackmann grading system is widely used to objectively assess the degree of facial asymmetry and paralysis. This scale is divided into six grades:

Grade	Degree of Dysfunction	Clinical Presentation
Grade I	Normal function	All facial functions are normal and symmetrical.

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Grade II	Mild dysfunction	The face is symmetrical at rest. Mild asymmetry is observed during movement, and the eye closes completely.
Grade III	Moderate dysfunction	Mild asymmetry may be observed at rest. Eyebrow elevation is weak, but the eye can be completely closed with effort.
Grade IV	Moderately severe dysfunction	Noticeable facial asymmetry at rest. Forehead movement is absent, and the eye cannot be completely closed.
Grade V	Severe dysfunction	Marked facial asymmetry is present even at rest. Facial movements are minimal, and eye closure is extremely difficult or nearly impossible.
Grade VI	Complete paralysis	Complete loss of voluntary movement and muscle tone in the facial muscles, resulting in total facial paralysis and atonia.

CONCLUSION

This article examined the anatomy of the facial nerve, its associated disorders, and the main approaches to their clinical diagnosis. The following conclusions were reached:

Anatomical factor: The facial nerve passes through the narrow Fallopian canal within the temporal bone. Therefore, even slight inflammation or nerve edema can cause compression, resulting in impaired facial movement and paralysis.

Importance of differentiation from stroke: When facial asymmetry occurs, it is essential to distinguish peripheral facial nerve paralysis from central paralysis caused by stroke. If the patient can wrinkle the forehead, central facial paralysis may be suspected. In contrast

inability to move the forehead together with incomplete eye closure is characteristic of peripheral facial nerve involvement, such as Bell's palsy.

Etiological factors: According to the information analyzed, common factors include exposure to cold or drafts, as well as reactivation of latent herpes viruses when immune function is weakened.

Clinical assessment: Even in the absence of sophisticated diagnostic equipment, simple facial movement tests-such as raising the eyebrows, closing the eyes, and smiling-can provide important clinical information. The House–Brackmann grading system can also be used to determine the severity of facial nerve dysfunction on a six-grade scale.

Prognosis: Early recognition and timely initiation of appropriate treatment are important for favorable recovery. According to the information presented, 80–85% of patients may achieve

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complete recovery of facial movements within 1–3 months when treatment is initiated appropriately and promptly.

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