

Current Approaches to the Clinical Anatomy of the Facial Nerve

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Abstract

The facial nerve is the seventh cranial nerve with a mixed character, containing motor, parasympathetic, general sensory, and special sensory fibers. It has a broad functional spectrum, from the control of facial muscles to taste sensation, salivary and tear secretion, and sensory innervation of the ear region. Facial nerve pathologies are among the frequently encountered neurological problems in medical practice, with Bell's palsy being the most common cause of peripheral facial paralysis. The complex anatomical course of the facial nerve, from its origin in the brainstem to its arrival at the facial muscles, is studied in multiple anatomical segments, each with its own unique clinical significance. Recent advances in imaging technologies and electrophysiological diagnostic methods have significantly expanded our knowledge of facial nerve anatomy and pathologies. This study comprehensively presents the embryological development, detailed anatomical course, functional characteristics, clinical significance, diagnostic approaches, and treatment strategies of the facial nerve in light of current literature.

Keywords: Facial nerve, anatomical segments, facial paralysis, Bell's palsy, hemifacial spasm

Nervus Facialis'in Klinik Anatomisine Güncel Yaklaşımlar

Özet

Nervus facialis, motor, parasempatik, genel duyuşal ve özel duyuşal lifler içeren mikst karakterli yedinci kranial sinirdir. Mimik kaslarının kontrolünden tat duyuşuna, tükürük ve gözyaşı sekresyonundan kulak bölgesinin duyuşal innervasyonuna kadar geniş bir fonksiyonel spektruma sahiptir. Nervus facialis patolojileri, tıbbi pratikte sık karşılaşılan nörolojik problemler arasında yer almakta olup, Bell paralizi en sık periferik fasiyal paralizi nedenidir. Nervus facialis beyin sapından çıkışından yüz kaslarına ulaşmaya kadar olan karmaşık anatomik seyri, birden fazla anatomik segmentte incelenmektedir ve her segment kendine özgü klinik öneme sahiptir. Son yıllarda görüntüleme teknolojilerindeki gelişmeler ve elektrofizyolojik tanı yöntemlerindeki ilerlemeler, nervus facialis anatomisi ve patolojileri hakkındaki bilgilerimizi önemli ölçüde genişletmiştir. Bu çalışmada, nervus facialis'in embriyolojik gelişimi, detaylı anatomik seyri, fonksiyonel özellikleri, klinik önemi, tanısal yaklaşımlar ve tedavi stratejileri güncel literatür ışığında kapsamlı olarak sunulmaktadır.

Anahtar kelimeler: Nervus facialis, anatomik segmentler, yüz felci, Bell felci, hemifasiyal spazm

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INTRODUCTION

The facial nerve, known as the seventh cranial nerve (CN), is one of the cranial nerves with the most complex anatomy. It has a mixed character due to the presence of visceromotor, viscerosensitive, and somatosensitive fibers (Arıncı & Elhan, 1995; Moore & Dalley, 2018). It originates from the nuclei located in the pons section of the brainstem and reaches the facial region after passing through the temporal bone. The facial nerve performs many functions, including controlling mimic muscles, tear and saliva secretion, taste sensation, and sensory innervation of the ear region (Kochhar et al., 2016; Marur et al., 2014).

Facial nerve (CN VII) pathologies are among the most frequently encountered neurological disorders in medical practice and can significantly affect patients' quality of life. Bell's palsy (idiopathic facial paralysis), with an annual incidence of 20–30 per 100,000, is the most common cause of peripheral facial paralysis, and its lifetime risk in the general population is approximately 1% (Peitersen, 2002; Steinhäuser et al., 2022). In acute facial nerve dysfunctions, traumatic and iatrogenic causes constitute a significant portion of acquired paralyses (Hohman & Hadlock, 2014; Fliss et al., 2022).

Facial nerve dysfunction, in addition to aesthetic disorders resulting from the loss of function of facial muscles, can lead to serious functional and psychosocial consequences. Corneal damage that may develop as a result of the inability to fully close the eyelid, feeding difficulties due to oral sphincter insufficiency, articulation disorders, and facial asymmetry are the most frequently encountered clinical problems of these pathologies (Gilden, 2004; May & Schaitkin, 2000). Furthermore, it has been reported that the frequency of depression is increased and mood and quality of life scores are significantly decreased in patients with severe facial dysfunction (Ishii et al., 2018).

The facial nerve's close relationship with numerous anatomical structures along its intracranial and extracranial course makes it extremely important for both basic sciences and clinical disciplines. In various specialties such as neurology, otolaryngology, plastic and reconstructive surgery, radiology, and physical therapy and rehabilitation, a correct understanding of the facial nerve's anatomical organization and functional integrity directly impacts diagnostic and treatment success (Seneviratne & Patel, 2023).

Rapid advancements in imaging technologies in recent years have led to significant progress in the evaluation of facial nerve pathologies. High-resolution magnetic resonance imaging (MRI) and computed tomography (CT) techniques offer detailed anatomical examination along the intracranial and extracranial course of the facial nerve (Ho et al., 2015; Ottaiano et al., 2023). CT provides superior resolution, particularly in the evaluation of bony structures such as the temporal bone and fallopian tube. MRI, on the other hand, allows for better contrast imaging of the nerve itself, surrounding soft tissues, and its relationships with cerebrospinal fluid. These two imaging methods always complement each other in the evaluation of the facial nerve.

The aim of this compilation is to create a comprehensive resource by bringing together current and evidence-based information on the anatomy, embryological development, and clinical implications of the facial nerve.

The information presented is intended to guide both researchers working in the basic sciences and physicians who use this information in clinical practice.

Embryological Development

The facial nerve develops through complex cellular and molecular interactions, beginning in the early embryonic period. Its embryological origin is the second branchial (pharyngeal) arch, which consists of a mesenchymal structure rich in neural crest cells. Each branchial arch is innervated by a specific cranial nerve, playing a decisive role in the development of the muscle, bone, and connective tissue components of that arch (Larsen et al., 2001).

Around the fourth week of embryonic development, motor neurons of the facial nerve begin to differentiate from the neural tube at the level of the rhombencephalon. These motor neurons emerge from the ventrolateral surface of the brainstem and extend towards the mesenchyme of the second branchial arch. During the same period, neural crest cells, which will form the sensory component of the facial nerve, migrate to form the geniculate ganglion (Guthrie, 2007; Ottaiano et al., 2023; Dođmuş, 2024).

The embryological development of the facial nerve is regulated by numerous transcription factors and signaling pathways. In particular, Hox and Pax genes, and molecules such as Sonic hedgehog and retinoic acid, are critically important for the correct segmental specification and target tissue orientation of motor neurons (Trainor & Krumlauf, 2001). The incorporation of neural crest cells into the vessel walls during migration along the pharyngeal arch arteries plays a fundamental role in the normal morphogenesis of the aortic arch and the large vessels that develop from it (Kochhar et al., 2016).

Functional Anatomy

The facial nerve is a mixed-character cranial nerve containing four main functional components: motor, parasympathetic (general visceral efferent), general somatic afferent, and special visceral afferent fibers (Arıncı & Elhan, 1995; Moore & Dalley, 2018; Ottaiano et al., 2023). Its possession of special visceral efferent (SVE) motor fibers, responsible for innervating facial muscles, is clinically significant.

The motor fibers of the facial nerve originate from the facial nerve motor nucleus located in the ventral pontine tegmentum of the pons. This nucleus is situated deep within the base of the fourth ventricle and is divided into four sub-nuclei (dorsal, intermediate, lateral, and medial) exhibiting somatotopic organization (Davis et al., 1956; Ottaiano et al., 2023). The upper portion of the motor nucleus receives bilateral corticobulbar innervation, while the lower portion receives innervation only from the contralateral hemisphere. This unique innervation pattern forms a fundamental anatomical basis for the differential diagnosis of central and peripheral facial paralyses (Gilden, 2004; Urban et al., 2001).

Motor fibers, after exiting the nucleus, travel dorsally, making a characteristic curve around the abducens nerve nucleus and forming a bulge at the base of the fourth ventricle known as the facial colliculus. This structure is considered a distinctive anatomical landmark in axial magnetic resonance imaging sections of the brainstem (Ottaiano et al., 2023).

The motor branches of the facial nerve innervate all of the mimic muscles (the frontalis, orbicularis oculi, corrugator supercilii, procerus, nasalis, orbicularis oris, zygomaticus major and minor, risorius, platysma muscles, and other perioral muscles). In addition to these muscles, the stapedius muscle, posterior belly of the digastric muscle, and stylohyoid muscle are also motor innervated by the facial nerve (Arıncı & Elhan, 1995; Marur et al., 2014).

The parasympathetic fibers of the facial nerve originate from the superior salivary nucleus and lacrimal nucleus located in the pons. Preganglionic parasympathetic fibers travel within the intermediate nerve to reach target glands (Kochhar et al., 2016; Ottaiano et al., 2023). These fibers have peripheral distribution via two main pathways (the major petrosal nerve and the chorda tympani). The major petrosal nerve is a branch originating from the geniculate ganglion and carries secretomotor fibers to the nasal and palatal glands, primarily the lacrimal gland. This nerve, which travels along the superior surface of the petrous portion of the temporal bone, passes through the foramen lacerum and joins the deep petrosal nerve to form the pterygoideal canal (Vidian nerve). Fibers reach target glands postganglionic after synapsing in the pterygopalatine ganglion (Arıncı & Elhan, 1995; Toulgoat et al., 2013).

The chorda tympani branches off from the mastoid segment of the facial nerve, passes through the tympanic cavity, and leaves the base of the skull via the petrotympanic fissure to join the lingual nerve. Parasympathetic fibers from this pathway synapse in the submandibular ganglion and provide secretomotor innervation to the submandibular gland and sublingual gland (Ho et al., 2015).

Special visceral afferent fibers of the facial nerve carry taste sensation from the anterior 2/3 of the tongue and the soft palate. These fibers join the facial nerve peripherally via the chorda tympani. The intermediate nerve (Wrisberg nerve) is a component of the facial nerve containing sensory and parasympathetic fibers, and it arises from the brainstem between the motor root and the vestibulocochlear nerve. The cell bodies of first-order sensory neurons are located in the geniculate ganglion, while their central extensions terminate in the nucleus tractus solitarius in the pons (Gasser, 1967; Ottaiano et al., 2023). General somatic afferent fibers carry sensation from the external acoustic meatus, a limited portion of the auricle, and the retroauricular area (Ramsay–Hunt zone). These fibers also travel within the intermediate nerve, and their first-order neurons are localized in the geniculate ganglion (Sataloff et al., 2014).

Anatomical Segments and Course

The facial nerve is studied by dividing it into six anatomical segments (intracranial, meatal, labyrinthic, tympanic, mastoid, and extratemporal) (Arıncı & Elhan, 1995; Toulgoat et al., 2013).

The intracranial segment covers the portion of the facial nerve from its exit from the brainstem to its entry into the internal acoustic canal is approximately 15–20 mm long. This segment runs within the cerebellopontine angle cistern in the posterior cranial fossa. The facial nerve arises from the anterolateral surface of the pontomedullary junction as two separate roots anteromedially to the vestibulocochlear nerve (CN VIII). These

roots are the larger motor root and the smaller intermediate nerve, which contains sensory and parasympathetic fibers (Seneviratne & Patel, 2023).

The region where the facial nerve exits the brainstem is called the root exit zone and is of critical clinical importance, especially in the pathogenesis of hemifacial spasm due to vascular compression (Seneviratne & Patel, 2023; Ottaiano et al., 2022). In high-resolution MRI, especially 3D-CISS or FIESTA sequences are preferred in the evaluation of the cisternal segment (Hatipoğlu et al., 2007).

The meatal segment corresponds to the course of the facial nerve within the internal acoustic canal and is approximately 7–8 mm long. This segment is located within the petrous part of the temporal bone. Within the internal acoustic canal, the facial nerve is localized in the anterosuperior quadrant and is separated from the superior vestibular nerve by a bony projection known as Bill's bar (Toulgoat et al., 2013; Ottaiano et al., 2023). The location of the facial nerve in the inner ear canal is important for surgical and radiological evaluation. In this area, the facial nerve is in close proximity to the cochlear and vestibular nerves (Ho et al., 2015).

The labyrinthine segment encompasses the portion of the facial nerve extending from the internal acoustic canal fundus to the geniculate ganglion, measuring approximately 3–5 mm in length. Located within the fallopian tube, this segment is considered the narrowest and therefore most vulnerable part of the facial nerve. At this level, the nerve travels between the cochlea and the vestibulum, making a sharp angle anterior to the lateral semicircular canal before reaching the geniculate ganglion; this anatomical turning point is called the first genu. Due to the limited bony canal volume of the labyrinthine segment, the nerve is easily compressed in cases of edema or inflammation. Therefore, nerve damage is most frequently reported in this segment in inflammatory facial paralyses such as Bell's palsy and Ramsay-Hunt syndrome (Arıncı & Elhan, 1995; Moore & Dalley, 2018; Peitersen, 2002; Gilden, 2004).

The geniculate ganglion is the sensory ganglion of the facial nerve, located at the distal end of the labyrinthine segment, in the first genu of the facial canal. This ganglion contains the cell bodies of the first pseudounipolar neurons belonging to the general somatic afferent and special visceral afferent (SVA) fibers of the facial nerve (Sataloff et al., 2014; Ottaiano et al., 2023). The most important branch, the petrosus major nerve, branches off from the geniculate ganglion (Ottaiano et al., 2023).

The tympanic segment extends from the geniculate ganglion to the pyramidal eminence and is approximately 8–11 mm long. It runs horizontally along the medial wall of the tympani cavity. In this segment, the facial nerve is located superior to the oval window and inferior to the lateral semicircular canal (Arıncı & Elhan, 1995; Moore & Dalley, 2018). The tympanic segment is the most common site for facial canal dehiscence. It has been reported that approximately 30% of normal individuals may have a thin or absent bony covering in this segment. This increases the risk of facial nerve injury during middle ear surgeries and otological infections (Kochhar et al., 2016; Ottaiano et al., 2023). Destructive middle ear pathologies such as cholesteatoma can also lead to facial nerve erosion and paralysis in this segment (Ho et al., 2015).

The mastoid segment extends from the pyramidal eminence to the stylomastoid foramen and is approximately 10–14 mm long. It runs vertically medial to the mastoid air cells. Clinically important branches, such as the stapedial nerve and chorda tympani, originate from this segment (Toulgoat et al., 2013; Ho et al., 2015). The stapedial nerve provides motor innervation to the stapedius muscle and forms the efferent limb of the acoustic reflex (Arıncı & Elhan, 1995; Moore & Dalley, 2018). The chorda tympani separates from the mastoid segment, passes through the middle ear, exits the base of the skull via the petrotympanic fissure, and carries parasympathetic fibers from the anterior two-thirds of the tongue, related to taste, and to the submandibular and sublingual glands (Arıncı & Elhan, 1995; Ho et al., 2015; Ottaiano et al., 2023).

The extratemporal segment is the portion of the facial nerve that forms its course within the parotid gland after exiting the stylomastoid foramen, and it is approximately 15–20 mm long. After exiting the stylomastoid foramen, the nerve enters the parotid gland and divides into two main branches: the temporofacial trunk and the cervicofacial trunk. These trunks then further divide into five terminal branches known as the temporal ramus, zygomatic ramus, buccalis ramus, marginal mandibulae ramus, and colli ramus, providing motor innervation to the facial muscles (Seneviratne & Patel, 2023; Triantafyllou, et al., 2024).

Extratemporal Branches

The extratemporal segment of the facial nerve is one of the most anatomically varied and surgically critical segments. The course of the nerve within the parotid gland, its branching pattern, and the connections between terminal branches can vary between individuals (Poutoglidis et al., 2022). These variations are particularly important for the preservation of the facial nerve during parotid surgery, facial reconstruction, and aesthetic procedures.

Within the parotid gland, the facial nerve is considered the primary anatomical structure separating the superficial and deep lobes of the gland. The length of the extratemporal segment averages 15–20 mm, and in this section, the nerve branches into terminal branches (Kochhar et al., 2016; Marur et al., 2014).

In the classic branching pattern, the facial nerve bifurcates into two main trunks within the parotid gland: the temporofacial trunk and the cervicofacial trunk. These two trunks then divide into five branches: the ramus temporalis, ramus zygomaticus, ramus buccalis, ramus marginalis mandibulae, and ramus colli. These branches provide motor innervation to the facial mimic muscles, forming the basis of facial expression (Triantafyllou, et al., 2024).

However, the extratemporal branching pattern may not always exhibit this classic structure. In a systematic review and meta-analysis conducted by Triantafyllou et al. (2024), the extratemporal branching patterns of the facial nerve were examined in detail and various variations were identified. Accordingly, the classic pattern characterized by bifurcation is the most common structure, with a reported prevalence of 94.1%. Trifurcation and plexiform branching patterns, although rarer, are among the clinically significant variations (Triantafyllou, et al., 2024).

Anastomoses of varying degrees can be observed between terminal branches in the extratemporal segment. These connections allow nerve fibers to pass from one branch to another, forming a complex neural network and contributing to partial compensation for functional loss in the event of possible branch damage. The presence of a single anastomosis between the temporofacial and cervicofacial trunks is the most common pattern among those described, reported with a prevalence of approximately 23.1% (Triantafyllou et al., 2024). The surgical importance of the extratemporal segment is also increased by age-related anatomical differences. While in adults the facial nerve is relatively protected by bony structures such as the tip of the mastoid process, the tympanic ring, and the mandibular ramus, in children under two years of age the nerve follows a more superficial course, increasing the risk of injury during trauma and surgical procedures (Myckatyn & MacKinnon, 2004).

Facial Nerve Pathologies

Facial nerve pathologies are neurological conditions frequently encountered in clinical practice and significantly affect the quality of life of patients. The most common clinical conditions among these pathologies are peripheral and central facial paralyses. The anatomical localization of the lesion directly determines the distribution of clinical findings and the prognosis (Peitersen, 2002; Gilden, 2004; May & Schaitkin 2000).

Peripheral facial paralysis develops as a result of lesions occurring at the level of the facial nerve nucleus of the facial nerve or at any point in its distal course. Motor function loss is seen in all or part of the ipsilateral facial muscles. In the clinical picture, all mimic muscles, including the forehead muscles, are affected (Peitersen, 2002).

Bell's palsy is the most common among peripheral facial paralyses. With an annual incidence of approximately 2–3 per 10,000 people, this condition can occur in all age groups, but is most common between the ages of 15 and 45, and is observed with similar frequency in both sexes (Peitersen, 2002). Although the etiopathogenesis of Bell's palsy is not fully understood, viral infections are the most widely accepted mechanism. In particular, herpes simplex virus type 1 and herpes zoster virus can be latent in the geniculate ganglion and initiate the inflammatory process after reactivation (Murakami et al., 1996).

Clinically, Bell's palsy usually begins acutely and presents with unilateral facial paralysis. Common findings include weakness in all ipsilateral facial muscles, brow ptosis, lagophthalmos, hyperacusis, impaired taste sensation, and decreased lacrimation and salivary secretion (Murakami et al., 1996; Gilden, 2004). The prognosis is favorable in most cases, with complete or near-complete recovery within 3–6 months in approximately 70–85% of patients. However, 15–30% of patients may develop permanent facial asymmetry, synkinesis, and muscle contractures (Peitersen, 2002).

Central facial paralysis results from lesions occurring along the corticonuclear tract, which forms the supranuclear course of the facial nerve. In this table, marked paralysis is observed in the lower facial muscles opposite the lesion, while the upper facial muscles (especially the frontalis muscle) are generally spared due

to bilateral cortical innervation. The most common cause of central facial paralysis is cerebrovascular events. In addition, multiple sclerosis, intracranial tumors, and brainstem lesions can also lead to central facial paralysis. This distinction between the upper and lower facial muscles in clinical examination is one of the key diagnostic clues in differentiating between central and peripheral lesions (Urban et al., 2001; Gilden, 2004).

In acute idiopathic peripheral facial paralysis, corticosteroid treatment initiated within the first 72 hours of symptom onset forms the basis of the current approach. In a large series of 1,220 patients by Steinhäuser et al. (2022), it was reported that the rates of improvement were significantly increased in patients who received acyclovir along with corticosteroids in the acute phase. In cases where facial nerve integrity is compromised due to traumatic or iatrogenic causes, end-to-end nerve anastomosis performed within the first 72 hours is considered the gold standard approach. Fliss et al. (2022) reported that repairs performed up to the first 6 months after injury can be effective in preserving facial muscle tone, but after 12 months, irreversible muscle atrophy necessitates resuscitation techniques such as static sling or free muscle transfer (gracilis). In cases requiring nerve grafting, the sural nerve or great auricular nerve are most frequently preferred.

Physical therapy and rehabilitation approaches are an integral part of treatment in both acute and chronic phases. In a systematic review by Khan et al. (2022), facial neuromuscular retraining programs based on facial exercises and biofeedback therapies applied in front of a mirror were shown to contribute to the reduction of synkinesis and the increase of functional recovery, especially in chronic cases. The effectiveness of electrical stimulation, however, is still debated. Yoo et al. (2023) showed in animal experiments that electrical stimulation accelerated nerve regeneration by increasing brain-derived neurotrophic factor (BDNF) levels. However, it is emphasized that inappropriate stimulation that causes muscle contraction in humans may increase the risk of synkinesis, therefore subthreshold (sensory level) stimulation should be preferred.

Blood Vessels and Nerves

The facial nerve is supplied by various arterial sources. In the intracranial segment, it is supplied by the AICA; in the intratemporal segment, by the stylomastoid artery and its petrous branches; and in the extratemporal segment, by the posterior auricular artery and branches of the facial artery (Arıncı & Elhan, 1995; Moore & Dalley 2018).

The venous drainage of the facial nerve varies along its course. In the intracranial segment, it is drained by the petrous sinuses; in the intratemporal segment, by the pterygoid plexus; and in the extratemporal segment, by the facial vein and the retromandibular vein (Arıncı & Elhan, 1995).

Lymphatic drainage around the facial nerve is provided via the parotid lymph nodes, mastoid lymph nodes, and deep cervical lymph nodes. Pathologies of these lymph nodes can affect facial nerve function (Ho et al., 2015).

CONCLUSION

The facial nerve is one of the cranial nerves with the most complex anatomy. Containing motor, parasympathetic, general sensory, and special sensory fibers, this mixed nerve performs numerous functions,

from controlling facial muscles to taste sensation. Understanding the anatomical course of the facial nerve from its origin in the brainstem to its connection with the facial muscles is clinically important.

The current literature demonstrates that a thorough understanding of the anatomy and clinical functions of the facial nerve is valuable for accurate diagnosis, prevention of complications, and effective treatment planning. The information presented in this review serves as a resource for clinicians and researchers working in the fields of anatomy, neurology, ENT, plastic surgery, radiology, and physical therapy. Further elucidation of the nerve's segmental structure, branching patterns, and clinical variations will contribute to the development of more personalized approaches in the diagnosis and treatment of facial nerve-related diseases in the future.

Conflict of Interests

Authors declare no conflicts of interest

AUTHOR CONTRIBUTIONS

Planning: A.K.; Literature review: A.K.; Writing: A.K., A.E.Ç. Critical reading: A.S.

REFERENCES

- Arıncı, K. & Elhan, A. (1995). *Anatomi* (1. Cilt). Güneş Kitabevi.
- Boahene, K. D., Byrne, P., & Ishii, M. (2018). The importance and psychology of facial palsy: Diagnostic and therapeutic management. *Otolaryngologic Clinics of North America*, 51(6), 1011-1019. <https://doi.org/10.1016/j.otc.2018.07.015>
- Davis, R. A., Anson, B. J., Budinger, J. M., & Kurth, L. E. (1956). Surgical anatomy of the facial nerve and parotid gland based upon a study of 350 cervicofacial halves. *Surgery, Gynecology & Obstetrics*, 102(4), 385-412.
- Doğmuş, S. (2024). Clinical Anatomy of the Facial Nerve with Fallopian Canal in Terms of Surgical Landmarks and Variations. In G. Açar & A. E. Çiçekcibaşı (Eds.), *Head and Neck Anatomy with Surgical Landmarks* (pp. 117-141). Livre de Lyon. <https://doi.org/10.5281/zenodo.12667653>
- Fliss, E., Yanko, R., Zaretski, A., Tulchinsky, R., Arad, E., Kedar, D. J., Fliss, D. M., & Amir, A. (2022). Facial nerve repair following acute nerve injury. *Archives of Plastic Surgery*, 49(4), 501-509. <https://doi.org/10.1055/s-0042-1755602>
- Gasser, R. F. (1967). The development of the facial nerve in man. *Annals of Otology, Rhinology & Laryngology*, 76(1), 37-56. <https://doi.org/10.1177/000348946707600104>
- Gilden, D. H. (2004). Clinical practice. Bell's palsy. *New England Journal of Medicine*, 351(13), 1323-1331. <https://doi.org/10.1056/NEJMcp041120>
- Guthrie, S. (2007). Patterning and axon guidance of cranial motor neurons. *Nature Reviews Neuroscience*, 8(11), 859-871. <https://doi.org/10.1038/nrn2254>
- Hatipoğlu, H. G., Durakoglugil, T., Ciliz, D., & Yüksel, E. (2007). Comparison of FSE T2W and 3D FIESTA sequences in the evaluation of posterior fossa cranial nerves with MR cisternography. *Diagnostic and Interventional Radiology*, 13(2), 56-60.
- Ho, M. L., Juliano, A., Eisenberg, R. L., & Moonis, G. (2015). Anatomy and pathology of the facial nerve. *American Journal of Roentgenology*, 204(6), W612-W619. <https://doi.org/10.2214/AJR.14.13444>
- Hohman, M. H., & Hadlock, T. A. (2014). Etiology, diagnosis, and management of facial palsy: 2000 patients at a facial nerve center. *The Laryngoscope*, 124(7), E283-E293. <https://doi.org/10.1002/lary.24542>

- Khan, A. J., Szczepura, A., Palmer, S., Bark, C., Neville, C., Thomson, D., & Leveque, J. L. (2022). Physical therapy for facial nerve paralysis (Bell's palsy): An updated and extended systematic review of the evidence for facial exercise therapy. *Clinical Rehabilitation*, 36(11), 1424-1449. <https://doi.org/10.1177/02692155221116834>
- Kochhar, A., Larian, B., & Azizzadeh, B. (2016). Facial nerve and parotid gland anatomy. *Otolaryngologic Clinics of North America*, 49(2), 273-284. <https://doi.org/10.1016/j.otc.2015.10.002>
- Larsen, W. J., Sherman, L. S., Potter, S. S., & Scott, W. J. (Eds.). (2001). *Human embryology* (3rd ed.). Churchill Livingstone.
- Marur, T., Tuna, Y., & Demirci, S. (2014). Facial anatomy. *Clinics in Dermatology*, 32(1), 14-23. <https://doi.org/10.1016/j.clindermatol.2013.05.022>
- May, M., Schaitkin, B. M., & Shapiro, A. M. (2000). Facial nerve disorders in newborns and children. In M. May & B. M. Schaitkin (Eds.), *The facial nerve* (pp. 339-365). Thieme Medical Publishers.
- Moore, K. L., & Dalley, A. F. (2018). *Clinically oriented anatomy* (8th ed.). Wolters Kluwer.
- Murakami, S., Mizobuchi, M., Nakashiro, Y., Doi, T., Hato, N., & Yanagihara, N. (1996). Bell palsy and herpes simplex virus: Identification of viral DNA in endoneurial fluid and muscle. *Annals of Internal Medicine*, 124(1), 27-30. https://doi.org/10.7326/0003-4819-124-1_Part_1-199601010-00005
- Myckatyn, T. M., & Mackinnon, S. E. (2004). A review of facial nerve anatomy. *Seminars in Plastic Surgery*, 18(1), 5-11. <https://doi.org/10.1055/s-2004-823119>
- Ottaiano, A. C., Gomez, G. D., & Freddi, T. D. A. L. (2023). The facial nerve: Anatomy and pathology. *Seminars in Ultrasound, CT and MRI*, 44(2), 71-80. <https://doi.org/10.1053/j.sult.2022.11.001>
- Peitersen, E. (2002). Bell's palsy: The spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. *Acta Oto-Laryngologica*, 122(7), 4-30. <https://doi.org/10.1080/000164802760370736>
- Poutoglidis, A., Paraskevas, G. K., Lazaridis, N., Georgalas, C., Vlachsis, K., Markou, K., & Piagkou, M. (2022). Extratemporal facial nerve branching patterns: Systematic review of 1497 cases. *The Journal of Laryngology & Otology*, 136(12), 1170-1176. <https://doi.org/10.1017/S0022215122002286>
- Sataloff, R. T., & Sataloff, J. B. (2014). *Embryology & anomalies of the facial nerve and their surgical implications* (2nd ed.). Jaypee Brothers Medical Publishers.
- Seneviratne, S. O., & Patel, B. C. (2023). Facial nerve anatomy and clinical applications. In StatPearls [Internet]. StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK554456/>
- Steinhäuser, J., Volk, G. F., Thielker, J., Geitner, M., Kutenreich, A. M., Klingner, C. M., Dobel, C., Guntinas-Lichius, O., & Volk, G. F. (2022). Multidisciplinary care of patients with facial palsy: Treatment of 1220 patients in a German facial nerve center. *Journal of Clinical Medicine*, 11(2), 427. <https://doi.org/10.3390/jcm11020427>
- Toulgoat, F., Sarrazin, J. L., Benoudiba, F., Pereon, Y., Auffray-Calvier, E., Dumas-Duport, B., Toulgoat, F., & Desal, H. A. (2013). Facial nerve: From anatomy to pathology. *Diagnostic and Interventional Imaging*, 94(10), 1033-1042. <https://doi.org/10.1016/j.diii.2013.06.013>
- Trainor, P. A., & Krumlauf, R. (2001). Hox genes, neural crest cells and branchial arch patterning. *Current Opinion in Cell Biology*, 13(6), 698-705. [https://doi.org/10.1016/S0955-0674\(00\)00268-8](https://doi.org/10.1016/S0955-0674(00)00268-8)
- Triantafyllou, G., Tsiouris, C., Chrysanthou, I., Paschopoulos, I., Tsakotos, G., Kalamatianos, T., Cappabianca, S., Panagiotopoulos, V., Grigoropoulos, A., & Filippou, D. (2024). Extratemporal facial nerve interconnections and trunk's variability: A systematic review with meta-analysis. *Diagnostics*, 14(17), 1862. <https://doi.org/10.3390/diagnostics14171862>
- Urban, P. P. (2002). The course of cortico-facial projections in the human brainstem. *Otology & Neurotology*, 23, S2-S3.
- Yoo, M. C., Kim, J. H., Kim, Y. J., Jung, J., Kim, S. S., Kim, S. H., & Chung, S. Y. (2023). Effects of electrical stimulation on facial paralysis recovery after facial nerve injury: A review on preclinical and clinical studies. *Journal of Clinical Medicine*, 12(12), 4133. <https://doi.org/10.3390/jcm12124133>