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THE TRIGEMINAL (V) AND FACIAL (VII) CRANIAL NERVES: Head and Face Sensation and Movement

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Psychiatry (Edgemont) 2010;7(1):25–31

ABSTRACT

There are close functional and anatomical relationships between cranial nerves V and VII in both their sensory and motor divisions. Sensation on the face is innervated by the trigeminal nerves (V) as are the muscles of mastication, but the muscles of facial expression are innervated mainly by the facial nerve (VII) as is the sensation of taste. This article briefly reviews the anatomy of these cranial nerves, disorders of these nerves that are of particular importance to psychiatry, and some considerations for differential diagnosis.

INTRODUCTION

Connoisseurs and wine experts intuit that there are interactions between somato-sensation (cranial nerve V) on the tongue and "taste" itself (cranial nerve VII). Recently, the interaction between the sensory parts of cranial verves V and VII has been illuminated.[1] For example, electrophysiological studies reveal that the trigeminal nerve (V), which innervates somato-sensation on the tongue, modulates the gustatory (taste) neurons arising from cranial nerve VII at the level of the solitary nucleus (medulla and lower pons) of cranial nerve VII.[1,2] Within the motor system, although the muscles of mastication are innervated by the trigeminal nerve (V), the muscles of

FUNDING: There was no funding for the development and writing of this article.

FINANCIAL DISCLOSURE: The authors have no conflicts of interest relevant to the content of this article.

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KEY WORDS: trigeminal nerve, trigeminal neuralgia, tic doloureaux, facial nerve, taste, Parry-Romberg syndrome, Bell's palsy, Sturge-Weber syndrome

facial expression are innervated mainly by the facial nerve (VII). The close functional and anatomical relationships between cranial nerves V and VII in both their sensory and motor divisions have induced us to discuss them together in this article.

CRANIAL NERVE V: THE TRIGEMINAL NERVE

Findings in psychiatric conditions. The corneal reflex, which involves trigeminal nerve afferents and facial nerve efferents, was found reduced in 30 percent and absent in eight percent of patients with schizophrenia who were chronically hospitalized.[3] Generally, the reflex is absent in states of sedation and coma[4] and after damage to the contralateral hemisphere.[5] In the elderly, it is often absent, especially among those with cerebrovascular disease.[6] A hyperactive masseteric (jaw-jerk) reflex is commonly seen in dementia and many neurologic conditions but has no clear diagnostic significance.

Trigeminal neuralgia. The most frequent disorder of the trigeminal nerve is trigeminal neuralgia (tic douloureux), and the severity of the pain sometimes generates a referral for a psychiatric consultation.[6] Trigeminal neuralgia can be idiopathic, but it often is caused by compression, demyelination,[7] or other injury of the trigeminal nerve root entry zone at the level of the pons or by pressure from an adjacent artery or vein.[8–10] Trigeminal neuralgia primarily affects the elderly, with a 3:2 preponderance in women. The pain is unilateral, tends to involve the second and third divisions of the sensory part of the nerve (maxillary and mandibular), and is intense enough to cause the patient to grimace (tic). There are initiating or trigger points. There is no sensory or motor "loss" per se. If trigeminal neuralgia is preceded or accompanied by hemifacial spasm, this may indicate that there is a tumor, aneurysm, or arteriovenous malformation compressing both the trigeminal (V) and facial (VII) nerves. Trigeminal neuralgia can also be associated with glossopharyngeal neuralgia (in the tonsillar region, cranial nerve IX).[11]

Medical management of trigeminal neuralgia is usually the initial treatment of choice. Microvascular decompression neurosurgery is sometimes recommended for persistent trigeminal neuralgia if the pain does not respond to medication (anticonvulsants, tricyclic antidepressants). In surgical cases, when there is an interneural vein that travels between the motor and sensory branches at the nerve root entry zone, this vein can be removed, relieving pressure on the nerve. In cases where this cannot be done (where the vein is bisecting a sensory branch of the nerve, for example), selective trigeminal nerve rhizotomy is an alternate approach to treatment.[8]

Sturge-Weber syndrome. Sturge-Weber syndrome (also called encephalofacial or encephalotrigeminal angiomatosis) is a neurocutaneous syndrome that is characterized by facial port-wine stains in the trigeminal nerve distribution, plus open angle glaucoma, and vascular lesions in the ipsilateral brain and meninges. The syndrome occurs sporadically and with equal frequency in male and female genders, and probably is due to a mutation during embryogenisis that caused a disruption in local angiogenesis.

In addition to issues surrounding self-image for which the psychiatrist may be consulted, the central nervous system involvement caused by the intracerebral vascular lesion sometimes results in focal "temporal lobe" seizures, with related ictal and postictal behavioral disturbances.[12]

ANATOMICAL RELATIONSHIPS OF THE TRIGEMINAL NERVE

The trigeminal nerve (V) is the largest cranial nerve, and it has both a sensory and a motor division. The motor division of the trigeminal nerve, which has its own nucleus located in the pons, innervates the "muscles of mastication" and also the tensor muscle of the tympanic membranes of the ear.

The trigeminal sensory nucleus (the substantia gelatinosa), in contrast to the motor nucleus, extends from the midbrain through the medulla and innervates sensation in the head and face. The best known disorder of the sensory division is trigeminal neuralgia, which has been described above.

Both the motor and sensory divisions leave the brainstem at the side of the pons, accompanied by the facial nerve (VII) and also cranial nerve VIII or the acoustic nerve. These three nerves pass through the so-called "cerebellopontine angle" together.[13] This is why an acoustic neuroma (CN VIII) can also affect the trigeminal nerve (V).

Trigeminal sensory ganglion. The trigeminal sensory ganglion receives three divisions of input that travel backward from the sensory receptor sites of the face: the ophthalmic, maxillary, and mandibular divisions. The ophthalmic part of the trigeminal nerve supplies sensation to the cornea, ciliary body, lachrymal glands, conjunctiva, nasal mucosa, and the skin of the nose, eyelid, and forehead. The maxillary part of the trigeminal nerve innervates the middle third of the face, the side of the nose, the lower eyelid, and upper teeth. The mandibular part of the trigeminal nerve supplies sensation to the lower third of the face, the anterior two-thirds of the tongue, the oral mucosa of the mouth, and the lower teeth. Infections and some malignancies (e.g., nasopharyngeal carcinoma or squamous cell carcinoma of the skin) can involve these peripheral divisions of the trigeminal nerve.[14]

Meckel's cavity. Inside the skull, the trigeminal sensory ganglion is very near the internal carotid artery in the posterior portion of the cavernous sinus in a cerebrospinal fluid (CSF)-filled sac called "Meckel's cavity." The cavernous sinus is a collection of veins between the temporal bone and the sphenoid bone, lateral to the sella turcica.

One-third of patients with intracavernous carotid aneurysms have trigeminal sensory ganglion manifestations because of the close approximations of these structures.[15,16]

Meckel's cavity also is a frequent site of metastases, which also can present as trigeminal neuropathy due to a cavernous sinus mass. Also, an expanding pituitary adenoma also can spread laterally through the cavernous sinus to secondarily cause a mass effect on the trigeminal sensory ganglion. Such a patient will present with sensory changes on face (including pain), cognitive changes, headaches, and/or behavioral changes. There also are primary tumors of Meckel's cavity that involve the trigeminal ganglion, such as trigeminal schwannoma.

Root entry zone. The sensory part of the trigeminal nerve just as it enters the pons is called the root entry zone. At this level, vascular anomalies, such as arteriovenous malformations and tumors, and inflammatory or infectious conditions, such as sarcoidosis, viral encephalitis, herpes, and Lyme disease, can affect cranial nerve V.

THE BRAINSTEM

Spinal sensory trigeminal tract. All tracts from the sensory trigeminal ganglion project to nuclei in the brainstem.

In the brainstem, the sensory part of the trigeminal nerve ganglion has three nuclei. The fibers of one of them (spinal sensory) carry pain and temperature sensation from the face. The spinal sensory trigeminal tract extends into the cervical cord. This explains why some patients with upper cervical disc herniation or occlusion of the vertebral artery can present with trigeminal sensory neuropathy.

Multiple sclerosis, glioma, and infarction are the most common cervical cord or brainstem lesions that cause trigeminal symptoms. The rare patient has developed herpes encephalitis from a retrograde extension of a herpes simplex infection from the trigeminal nerve ganglion to the brainstem.[17]

Facial motor activity is often abnormal in neuropsychiatric conditions. Facial movements that are impaired on command but intact during spontaneous expressive movement reflect pyramidal tract pathology, while the opposite dissociation can reflect pathology in several other cortical and subcortical systems.[19]

CLINICAL EXAMINATION OF THE TRIGEMINAL NERVE

Observation. Normally, there should be no jaw tremor, involuntary chewing, or trismus (clenching of teeth).

Motor division. The patient should be able to symmetrically and tightly clench the teeth, and the mandible should be midline. If there is motor weakness, the jaw will deviate to the weak side and central median incisors will not be aligned.

Reflexes. To perform the jaw jerk reflex, which tests both motor and sensory divisions of cranial nerve V, place your finger on the tip of patient's jaw and tap your finger lightly with a reflex hammer. Increased symmetrical closure rate reflects an upper motor neuron lesion in the same way that hyperactivity of other muscle stretch reflexes suggests an upper motor neuron lesion.

Sensation. The patient should be able to feel the touch of a sterile sharp object on the oral mucosa, jaw, cheek, and forehead. To test the corneal reflex, the cornea of one eye is touched with a cotton wisp, and normally there should be a bilateral blinking response. Tactile insensitivity that splits the midline and also bilaterally asymmetric vibratory sensation are often considered psychogenic or hysterical signs, but the evidence does not support this.[18]

CRANIAL NERVE VII: THE FACIAL NERVE

Findings in psychiatric conditions. Facial motor activity is often abnormal in neuropsychiatric conditions. Facial movements that are impaired on command but intact during spontaneous expressive movement reflect pyramidal tract pathology, while the opposite dissociation can reflect pathology in several other cortical and subcortical systems.[19] Diffuse horizontal wrinkling of the forehead in schizophrenia can give an appearance of surprise.[20] Forehead wrinkling in depression ("omega sign") is localized to the lower medial forehead.[21]

Subtle facial asymmetry is common without demonstrable pathology. Facial asymmetry or other signs of facial motor weakness are seen in 3 to 5 percent of patients with schizophrenia, compared with less than one percent of healthy people.[22–24] Unipolar depression may be characterized by exaggerations of normal asymmetries in emotional expression, which could potentially mislead the examiner, but these asymmetries vary with the specific expression.[25]

Bell's palsy. The lower motor neuron lesion of cranial nerve VII is referred to as Bell's palsy. In Bell's palsy, symptoms start acutely. Pain behind the ear may precede paralysis by 24 to 48 hours. There can be a transient (up to two weeks) loss of the sensation of taste. One side of the face becomes paralyzed (for both voluntary and involuntary movement), and the forehead is affected as much as the lower face. Incomplete paralysis in the first week is the most favorable prognostic sign. The incidence of Bell's palsy is higher in persons with diabetes. Usually Bell's palsy is idiopathic, but other causes include Lyme disease (from the tic-borne

spirochete *Borrelia burgdorferi*) or herpes simplex.

There are case reports of body dysmorphic disorder being triggered by Bell's palsy.[26–38] In one case,[26] a boy of 15 developed Bell's palsy and despite resolution of the paralysis he became increasingly self-absorbed and socially isolated, stating that he had severe facial and skin deformities. At the age of 17 he attempted suicide and was hospitalized.

Other disorders that affect the motor functioning of cranial nerve VII include tumors that invade the temporal bone, fracture of the temporal bone, Ramsay-Hunt syndrome (which is herpes zoster of the geniculate gangion that presents with severe facial palsy associated with a vesicular eruption in the external auditory canal), acoustic neuromas, and dilatations of the basilar artery from aneurysms, leprosy, and infectious mononucleosis. Infectious mononucleosis can present with multiple or single cranial palsies of acute onset, with bilateral facial paralysis being the most common combination.[29] Myasthenia gravis can affect the motor function of cranial nerve VII.

Irritative lesions (including acoustic neuroma) can result in hemifacial spasm, and this also can be transient or permanent sequelae of Bell's palsy. Also, some elderly patients develop an involuntary recurrent spasm of both eyelids as an isolated phenomenon. Meige's syndrome is an uncommon disorder that includes involuntary clenching of the jaw and squinting of the eyes.

Differential diagnosis. A rare condition that may be part of the differential diagnosis in women who present with apparent stigmata of remote, partially resolved Bell's palsy is the unusual syndrome of facial hemiatrophy.[30–33] In facial hemiatrophy, which is not due to a cranial nerve VII lesion, there is disappearance of fat in the dermal and subcutaneous tissues on one side of the face. Facial hemiatrophy occasionally presents with an associated severe facial pain caused by displacement of the trigeminal sensory nerves resulting in trigeminal neuralgia (cranial nerve V). Facial hemiatrophy is actually a form of lipodystrophy.

The emotional impact of facial deformities. Patients who possess (or believe they possess) facial deformities are often severely impacted. As the late Lucy Gealy, a woman with a postoperative facial deformity due to surgery for a malignancy, explained: "I spent five years of my life being treated for cancer, but since then I've spent 15 years being treated for nothing other than looking different from everyone else. It was the pain from that, from feeling ugly, that I always viewed as the great tragedy of my life. The fact that I had cancer seemed minor in comparison."[34]

ANATOMICAL RELATIONSHIPS OF THE FACIAL NERVE (CRANIAL NERVE VII)

Relationship to the ear. Because of its close approximation to the temporal bone, the peripheral part of the facial nerve (VII) is involved in many conditions that affect that bone, which often also affect the ear. These conditions include congenital anomalies, degenerative disorders, infections, and neoplasms.

Embryologic development. The facial nerve develops embryologically from tissue that also gives rise to the acoustic nerve (cranial nerve VIII). Since it leaves the brainstem at the pontomedullary junction near the acoustic nerve (cranial nerve VIII), a vestibular schwannoma can affect the facial nerve as well as the acoustic nerve. The motor division of the facial nerve develops embryologically near the middle ear and eventually elongates and travels through a canal in the temporal bone near the structures of the ear. Incomplete development of this canal may contribute to the facial palsies sometimes associated with otitis media. The sensory fibers of the facial nerve (nervus intermedius), which respond to taste, also have their cell bodies in a sensory ganglion located near the inner ear.[35]

CRANIAL NERVE VII: SUMMARY OF THE FOUR COMPONENTS

Cranial nerve VII originates in four nuclei in the pons and medulla. These nuclei all combine to travel, via the internal auditory meatus, to the geniculate ganglion.

1. The somatic motor component, which innervates the muscles of facial expression and the stapedius muscle of the ear, originates in the pons at the facial nucleus. It circles around cranial nerve VI.
2. The visceral motor component, which innervates the lacrimal, submaxillary, and submandibular glands, originates in the medulla at the superior salivatory nucleus.
3. The somatic sensory component of cranial nerve VII, which innervates external ear sensation, originates in the medulla at the level of the spinal nucleus of cranial nerve V.
4. The visceral sensory component, which innervates taste for the anterior two thirds of the tongue, originates in the medulla at the solitary tract nucleus.

SENSORY AND SECRETOMOTOR DIVISIONS OF CRANIAL NERVE VII

The sensory fibers of the facial nerve, called the chorda tympani nerve, respond to taste input from the taste buds of the tongue. The cell bodies of these fibers are in a sensory ganglion located near the inner ear (called the geniculate ganglion). Rather than entering the skull with the facial nerve, the chorda tympani travels separately. It passes between the malleus and the incus bones of the ear and enters from the skull separately, which is why taste is sometimes spared even though other branches of the facial nerve are affected in Bell's palsy. The chorda tympani has its own nucleus of cell bodies in the medulla, called the nucleus solitarius.

Secretomotor fibers of cranial nerve VII innervate the sublingual and submaxillary glands. These fibers originate from the salivary

nucleus, which is located in the pons, near the motor nucleus.

EXAMINATION OF CRANIAL NERVE VII

Observation and motor function. Inspect the face for droop or asymmetry. Ask the patient to look up, so that the forehead wrinkles, and observe if there is a loss of wrinkling on one side. Push down on each side of the forehead. Strength will be relatively preserved in an upper motor neuron lesion, because of bilateral innervation of the upper part of the facial musculature.

Ask patient to hold shut both eyes and compare the strength of closure on each side. Observe nasolabial folds during voluntary movement. Observe the patient frowning, showing teeth, and puffing out the cheeks. In addition, observe for facial asymmetry during spontaneous facial expression (most often smiling in response to humor or good news).

Muscle stretch reflexes involving the facial nerve can be elicited, but tend to be more prominent in neurologic disease. These "primitive reflexes" include the snout (tapping on the upper lip causes reflex contraction of the orbicularis oris so that the lips protrude) and glabellar (tapping between the eyebrows causes reflex contraction of the orbicularis oculi, resulting in a blink). They are not useful in localizing lesions or assessing facial nerve function.

LOCALIZING A LOWER MOTOR NEURON CRANIAL NERVE VII LESION

If the lesion is at the stylomastoid foramen (termination of facial canal, where the facial nerve leaves skull), all muscles of facial expression are paralyzed. The corner of mouth droops. Creases and skin folds are effaced. The forehead is unfurrowed on the side of the lesion. The palpebral fissure is widened. The eyelids will not close. The lower lid sags. Tears spill over onto the cheek. However, taste is intact (because the chorda tympani nerve (taste) enters the skull in a different place than the rest of the facial nerve).

If there is hyperacusis (increased auditory volume in an affected ear), this is due the stapedius muscle in the middle ear being affected. The stapedius muscle functions to dampen ossicle movements, which normally decreases volume. If there is cranial nerve VII nerve damage, this muscle is paralyzed. Because the branch of the seventh cranial nerve that goes to the stapedius muscle begins very proximally, hyperacusis due to seventh cranial nerve lesions indicates a lesion close to the nerve's origin in the brainstem rather than more peripheral. Nevertheless, it is still a lower motor neuron lesion, because the lesion is not affecting the corticbulbar tract.

UPPER MOTOR NEURON LESIONS OF CRANIAL NERVE VII

The motor nucleus of cranial nerve VII is in the pons, lateral to the abducens nerve (cranial nerve VI). The fibers of the motor division (mostly) cross at some level in the central nervous system, and so injuries to either the cerebral cortex or upper brainstem (both of which affect the corticobulbar tract) result in paresis of the lower part of the face opposite to the side of the central nervous system lesion. However, since both cerebral hemispheres innervate the superior part of the face, central nervous system lesions spare the forehead muscle. This sort of injury is called an "upper motor neuron" lesion of cranial nerve VII, as it involves the corticbulbar pathway from the motor cortex.

The motor or facial nuclei also receive projections the extrapyramidal system and from the frontal lobe, which control emotional expression. This dual innervation of the facial nucleus of cranial nerve VII may explain the phenomenon of paresis of voluntary facial expression when there still is involuntary movement associated with emotional states.

SUMMARY

There are close functional and anatomical relationships between cranial nerves V and VII in both their sensory and motor divisions. The motor division of the trigeminal nerve innervates the "muscles of mastication," while the best known disorder of the trigeminal sensory division is "trigeminal neuralgia." Facial expression is innervated by the facial nerve (VII) and is often abnormal in neuropsychiatric conditions. Facial movements that are impaired on command but intact during spontaneous expressive movement reflect pyramidal tract pathology, while the opposite dissociation can reflect pathology in several other cortical and subcortical systems. Simple bedside observations and tests can quickly aid the psychiatrist in determining whether a lesion exists involving cranial nerves V and/or VII.

REFERENCES

1. Felizardo R, Boucher Y, Braud A, et al. Trigeminal projections on gustatory neurons of the nucleus of the solitary tract: a double-label strategy using electrical stimulation of the chorda tympani and tracer injection in the lingual nerve. *Brain Res*. 2009;1288:60–68.
2. Hummel T, Iannilli E, Frasnelli J, et al. Central processing of trigeminal activation in humans. *Ann NY Acad Sci*. 2009;1170:190–195.
3. Runeberg J. Die neurologie der schizophrenie. *Acta Psychiatrica et Neurologica*. 1936;11:523–547.
4. Cruccu G, Leardi MG, Ferracuti S, Manfredi M. Corneal reflex responses to mechanical and electrical stimuli in coma and narcotic analgesia in humans. *Neurosci Lett*. 1997;222:33–36.
5. Berardelli A, Accornero N, Cruccu G, et al. The orbicularis oculi response after hemispheral damage. *J Neurol Neurosurg Psychiatry*. 1983;46:837–843.
6. Phansalkar S, Stress M, Gaviria M. Psychiatry consultation service in neurosurgery. *Surgical Neurology*. 2002;57:204–208.

7. Marinkivic S, Gibo H, Todorovic V, et al. Ultrastructure and immunohistochemistry of the trigeminal peripheral myelinated axons in patients with neuralgia. *Clin Neurol Neurosurg*. 2009;111:795–800.
8. Helbig GM, Callahan JD, Cohen-Gadol AA. Variant intraneural vein-trigeminal nerve relationships: an observation during microvascular decompression surgery for trigeminal neuralgia. *Neurosurgery*. 2009;65:958–961.
9. Penarrocha M, Mora E, Bagan JV, et al. Idiopathic trigeminal neuropathies: a presentation of 15 cases. *J Oral Maxillofac Surg*. 2009;67:2364–2368.
10. Lecky BR, Hugher RA, Murray NM. Trigeminal sensory neuropathy. A study of 22 cases. *Brain*. 1987;110:1463–1485.
11. Adams RD, Victor M. *Principles of Neurology*, *Third Edition*. New York: McGraw-Hill Book Company; 1985.
12. Madaan V, Dewan V, Ramaswamy S, Ashis S. Behavioral manifestations of Sturge-Weber Syndrome: A case report. *J Clin Psychiatry*. 2006;8:198–200.
13. Kaufman DM. *Clinical Neurology for Psychiatrists*, *Sixth Edition*. Philadelphia, PA: Saunders Elsevier; 2007.
14. Kamel HAM, Toland J. Trigeminal nerve anatomy, pictorial essay. *Am J Roentgenol*. 2001;176:247–251.
15. DeMarco JK, Hesselink JR. Trigeminal neuropathy. *Neuroimaging Clin N Am*. 1993;3:111.
16. Majorie CBLM, Verbeeten B, Dol JA, et al. Trigeminal neuropathy: evaluation with MR imaging. *Radiographics*. 1995;15:795–811.
17. Tien RD, Dillon WP. Herpes trigeminal neuritis and rhombencephalitis on Gd-DTPA-enhanced MR imaging. *Am J Neuroradiol*. 1990;11:413.
18. Rolak LA. Psychogenic sensory loss. *J Nerv Ment Dis*. 1988;176:686–687.
19. Lishman WA. Clinical assessment In: Lishman WA (ed.) *Organic Psychiatry: The Psychological Consequences of Cerebral Disorder*, *Third Edition*. Malden, MA:Blackwell Science;1998.
20. Stoddart WHB. *Mind and Its Disorders: A Textbook for Students and Practitioners of Medicine*. London, United Kingdom: HK Lewis;1926.
21. Greden JF, Genero N, Price HL. Agitation-increased electromyogram activity in the corrugator muscle region: a possible explanation of the "Omega sign?" *Am J Psychiatry*. 1985;142:348–351.
22. Lemke VR, Neurologische befunde bei schizophrenen. *Psychiatrie Neurologie und Medizinische Psychologie*. 1955;7:226–229.
23. Griffiths TD, Sigmundsson T, Takei T, et al. Neurological abnormalities in familial and sporadic schizophrenia. *Brain*. 1998;121:191–203.
24. Braun CMJ, Lapierre D, Hodgins S, et al. Neurological soft signs in schizophrenia: Are they related to negative or positive symptoms, neuropsychological performance, or violence? *Arch Clin Neuropsychology*. 1995;10:489–509.
25. Yecker S, Borod JC, Brozgold A, et al. Lateralization of facial emotional expression in schizophrenic and depressed patients. *J Neuropsychiatry Clin Neurosci*. 1999;11:370–379.
26. Gabbay V, O'Dowd MA, Weiss AJ, Asnis GM. Body dysmorphic disorder triggered by medical illness? *Am J Psychiatry*. 2002;159:493.
27. Rajarethinam RP, Abelson JL, Himle JA. Acute onset and remission of obsessions and compulsions following medical illnesses and stress. *Depress Anxiety*. 2000;12:238–240.
28. Buhlman U, Etcoff NL, Wilhelm S. Facial attractiveness ratings and perfectionism in body dysmorphic disorder and obsessive-compulsive disorder. *J Anxiety Disord*. 2008;22:540–547.
29. Nager GT, Proctor B. Anatomic variations and anomalies involving the facial canal. *Otolaryngol Clin North Am*. 1991;24:531.
30. Nager GT, Proctor B. Anatomic variations and anomalies involving the facial canal. *Otolaryngol Clin North Am*. 1991;24:531.
31. Robinson LK, Hoyme HE, Edwards DK, Jones KL. Vascular pathogenesis of unilateral craniofacial defects. *J Pediatr*. 1987;111:236–239.
32. Kumar AA, Kumar RA, Shantha GP, Aloogopinathan G. Progressive hemifacial atrophy-Parry Romberg syndrome presenting as severe facial pain in a young man: a case report. *Cases J*. 2009;6776.
33. Poswillo D. The pathogenesis of the first and second branchial arch syndrome. *Oral Surg Oral Med Oral Pathol*. 1973;35:302–328.
34. Grealy L. *Autobiography of a Face*. New York: HarperCollins Publishers;1994.
35. Courbille J. The nucleus of the facial nerve: the relation between cellular groups and peripheral branches of the nerve. *Brain*. 1966;1:338.