EVALUATING THE CRANIAL NERVES, PART IV

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INTRODUCTION

As mentioned in previous articles, a thorough neurological examination includes evaluation of a patient's mental status and speech, motor system, sensory system, reflexes, and cranial nerves. Parts I, II, and III dealt with Cranial Nerves I through VI; this article will present an overview of Cranial Nerve VII, which innervates the facial, scalp, and neck muscles, certain glands, and a portion of the tongue. Some cranial nerves contain only sensory fibers; those that contain both sensory and motor fibers are called mixed nerves.

CRANIAL NERVE VII

Facial Nerve (Mixed)

The motor portion of the facial nerve originates in the pons and terminates in facial, scalp, and neck muscles and the lacrimal, sublingual, submandibular, nasal, and palatine glands (Figure 1).

The sensory portion originates from the taste buds found on the anterior two-thirds of the tongue. After passing through the geniculate ganglion, a swollen portion of the facial nerve that contains unipolar cells, the sensory fibers travel to a nucleus in the pons, which sends fibers to the thalamus. The thalamus, in turn, relays fibers to the gustatory portion of the cerebral cortex.

The sensory portion also sends deep general sensations from the face, and proprioceptive sensations from facial and scalp muscles.

Action of the Motor Portion

The chief action of the motor portion of the facial nerve is to control those muscles of facial expression and the glands that control secretion of tears and saliva.

The facial expression muscles surround every facial aperture, such as the eyes, mouth, nostrils, and external

auditory canals. Every facial movement (excluding mandibular movements, which are controlled by CN V) is controlled by CN VII.

A lesser action of the facial nerve is control of the stapedius muscle to dampen excessive movement of the ossicles (in the ear) during loud sounds. In addition, other branches of the facial nerve innervate the auricular muscles, the posterior belly of the digastric muscles, and the stylohyoid muscles.

Sensory Portion

The sensory portion of the facial nerve consists of taste fibers from the anterior two-thirds of the tongue, the floor of the mouth, the soft palate, some general sensation fibers from the external acoustic meatus and auricular concha, and some proprioceptive fibers that carry sensations of deep pressure and position sense from the face and scalp.

SOME DISORDERS OF VII

About three-fourths of facial nerve lesions fall into the Bell's Palsy type of disorder, which is characterized by paralysis of the facial muscles, loss of taste, and problems with closing the eyelids (when patients attempt to close their eyelids, the eyeball on the affected side may turn upward, a movement known as Bell's phenomenon).

Bell's Palsy, or peripheral facial paralysis, can be caused by a number of agents, including chills of the face, middle ear infections, tumors, fractures, hemorrhages, meningitis, infectious diseases, etc. The disorder may occur at any age, but is more common in the second to fifth decade of life.

Other, more rare, disorders make up the other one-fourth of facial nerve lesions and fall outside the scope of this discussion.

Symptoms and Signs of Bell's Palsy

Variations in the symptoms and signs of Bell's Palsy occur depend on the actual location of the lesion. The part of the facial nerve that controls the muscles of the upper part of the face receives fibers from both cerebral

Figure 1: A very simplified schematic of the facial nerve and some of the muscles it innervates.

hemispheres. Therefore, an upper motor neuron (UMN) lesion of the facial nerve will demonstrate a gradient of involvement that completely paralyzes only the lower muscles of the face. A lower motor neuron (LMN) lesion, on the other hand, will paralyze all the muscles on the affected side of the face.

LMN lesions cause flaccid paralysis of musculature. The lower eyelid droops and tears flow from the unprotected eye. The mouth also droops and drools. Depending on the actual location of the lesion, loss of taste on the anterior two-thirds of the tongue may also occur, as well as increased sensitivity to sound (hyperacusis) from paralysis of the stapedius muscle (mentioned earlier).

CLINICAL TESTS

Motor Examination

The facial nerve innervates all muscles of facial expression. The astute examiner begins an inspection upon meeting and talking with the patient, noting the play of facial muscles and any asymmetry of facial movements or blinking, and asymmetry and depth of the nasolabial folds. Note that other disorders such as muscular dystrophy, parkinsonism, and even depression can reduce facial movement, a condition called “masked facies.” Clinically important muscles to test include the frontalis, orbicularis oculi, orbicularis oris, buccinator, and platysma:

1. The frontalis muscle controls the forehead. To test, the patient is asked to wrinkle up his forehead, as in looking upward at the ceiling.
2. The orbicularis oculi is the sphincter muscle surrounding the eyeball. The patient is asked to close his eyes as tightly as possible; the examiner tries to force them open.
3. The orbicularis oris is the sphincter muscle surrounding the mouth. The patient is asked to close his lips as tightly as possible.
4. The buccinator is a major cheek muscle that compresses the cheek (as in the act of blowing air) and allows the cheek to cave in (as in the act of sucking). The patient is asked to pull back the corners of his mouth, as if smiling.
5. The platysma draws the outer part of the lower lip down and backward as if pouting. The patient is asked to pull down the corners of his mouth.

Sensory Examination

The only clinically testable sensory portion of the facial nerve is taste. For routine clinical examination, the examiner needs to test only one of the four tastes: salty (salt), sweet (sugar), sour (citric acid), or bitter (quinine).

Since the anterior two-thirds of the tongue is affected with lesions of the facial nerve, it's probably best to use salt or sugar, which are detected on that portion of the tongue. Place a small amount of the test substance on the patient's stuck-out tongue, massage it in slightly with a tongue depressor, and have the patient identify the taste. Test each side of the tongue separately after rinsing.

Is it an UMN or LMN?

Trace the nerve pathways in Figure 2 to see why the upper and lower portions of the face are affected differently by UMN or LMN lesions.

Patient A in the figure was asked to close his eyes tightly and show his teeth by smiling. Not shown is equal forehead movement when asked to look up. Lack of wrinkling around the right eye shows weak eyelid closure. The mouth retraction on the right was paralyzed. The gradient of paralytic involvement indicates a left UMN lesion.

Patient B was also asked to close his eyes tightly and smile. Not shown is unilateral left paralysis of the forehead muscles and lack of wrinkling when asked to look up. The left eye droops and fails to shut. The left mouth

Figure 2: Shows the gradient innervation of the facial nerve. Upper facial muscles are bilaterally innervated, whereas lower facial muscles are unilaterally innervated. "A" represents a left UMN lesion. Note that the left frontalis muscle is still innervated via the right corticobulbar tract, but the right buccinator and platysma are paralyzed. "B" represent a left LMN lesion. Note that all the muscles on the left side are paralyzed: the left eye remains open and droops, and the left side of the mouth droops and can't be drawn back.

retraction is paralyzed. Total unilateral paralysis of all facial muscles indicates a left LMN lesion.

PROGNOSIS

In most cases, partial or complete recovery occurs in a few weeks up to a couple of years (for older patients), although contractures may develop on the involved side. Recurrence sometimes happens. The patient should keep his face warm and avoid exposure to wind and dust. Eyedrops and an eyepatch may be necessary to prevent the eyeball from drying. Heat and gentle, upward massage may hasten recovery and maintain muscle tone.

Every-other-day electrical stimulation after the first couple of weeks may help prevent muscle atrophy.

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