

BELL'S PALSY

The seventh cranial nerve (Cranial N. VII) is commonly referred to as the facial nerve. Its motor root emerges from the posterior border of the pons, just lateral to the inferior olive through the medial side of the cerebellopontine angle, to leave the cranium through the internal acoustic meatus. Its sensory root originates in the geniculate ganglion, passes through the internal acoustic meatus and finally penetrates the medulla. It is a complex nerve, containing both efferent and afferent elements from both the autonomic and the central nervous systems. Its motor division is made up of motor neurons that not only innervate skeletal muscle derived from embryonic branchial arch mesoderm, but also innervate both smooth muscle and various glands. Its sensory division innervates the proprioceptive sensory organs in various muscles and associated tendons, as well as sensory receptors for touch, pain, temperature sensibility in the facial skin, and chemoreceptors in the mucous or serous membranes that play a role in taste sensation.

The facial nerve innervates the muscles of the face and scalp, as well as the stapedius, stylohyoid, the posterior belly of the digastric and the anterior two thirds of the tongue. It also innervates the external auditory meatus, as well as the submandibular, sublingual, and lacrimal glands. Consequently, it controls facial expression, the secretion of saliva and tears, aids in the perception of taste, and provides for the perception of pain and temperature from the external auditory meatus.

The portion of the facial nerve that is most important to us here (for the most part) is the portion that innervates the facial muscles. It leaves the skull through the stylomastoid foramen, branching to become the temporofacial and cervicofacial nerves, which in turn sub-branch to ultimately reach the appropriate end organs in the various facial muscles.

Bell's palsy (peripheral facial paralysis or prosopoplegia) is generally caused by neuritis or compression of the facial nerve by soft tissue swelling, usually as it passes through the stylomastoid foramina in association with the mastoid process. Such soft tissue swelling may have resulted from chilling of the face, middle ear

infection, tumors, fractures, meningitis, hemorrhage, infectious disease, and other less common causes.

The symptoms of *Bell's palsy* occur on the same side of the face as the lesion. The number of symptoms and their severity depend on the extent of the lesion and its precise site along the facial nerve. If the lesion occurs outside the stylomastoid foramen, the corner of the mouth will droop and draw to the opposite side, deep facial sensation will be lost. The patient may not be able to whistle, wink, close the involved eye (with attendant tearing), or wrinkle the forehead. If the lesion occurs in the facial canal and involves the chorda tympani nerve, the loss of the sense of taste in the anterior two-thirds of the tongue and a reduction of salivation on the affected side may be added to the above symptoms. If the lesion is higher in the facial canal and involves the stapedius muscle, hyperacusis may also result. If the lesion on the facial nerve involves the geniculate ganglion, the onset is often acute, with pain behind and in the ear accompanying paralysis. Lesion in the internal auditory meatus produces the stereotypic paralysis along with deafness in one ear. Lesion of the facial nerve as it emerges from the pons results in facial paralysis with various neuromuscular complications that depend on the extent of cranial nerve involvement (Cranial Nerves V, VIII, VI, XI, or XII).

Most cases of *Bell's palsy* clear up in thirty days, but in some patients, the paralysis may last for months, or indefinitely, with wide variations in the extent of recovery and the permanency of neuromuscular deficits or defects. The speed and extent of recovery depend on the extent and the speed of reinnervation.

In acute or slowly developing cases of *Bell's palsy*, evidence of soft tissue inflammation may be present in the area over or just anterior to the mastoid process of the skull. The involved area may be slightly swollen and tender to palpation. Confirmatory objective evidence of inflammation may come from differential skin resistance (DSR) survey (the skin resistance right over the inflammation will be relatively higher than that of the surrounding area). Inflammation may also be present over and around either of apertures through

which branches of the fascial nerve make their way through the jaw.

The inflamed zone(s) should first be established through DSR survey and a visual inspection (to establish the presence of swelling) of the involved mastoid and proximal jaw areas.

Treatment

If Inflammation is present, it may be successfully treated with phonophoresis of non-steroidal anti-inflammatories. If applied early enough, the developing swelling (if fluid) may be halted and the paralysis curtailed, reversed, or prevented.

Application:

- Preset the ultrasound unit to deliver a 1 MHz pulsed waveform, at 1.8 W/cm^2 . Ultrasound the inflamed zone, utilizing an effective non-steroidal anti-inflammatory as a coupling agent, for six minutes. This

procedure is designed to soften the adhesions that may be present.

- Manipulate the inflamed zone to eliminate any adhesions that may be present.
- After 30 minutes have elapsed, following the first ultrasound procedure, ultrasound the inflamed zone a second time.

Five or six sessions may be required for successful treatment. Success in the acute situation, before full paralysis has occurred, is defined as a full halt in the progress of the paralysis, restoration of full nerve function, and no immediate return of the syndrome.

Chronic facial nerve paralysis is often treatable through the use of electromyometric feedback for neuromuscular reeducation. If the motor element of the nerve is intact, it may be possible to achieve near or full recovery of motor function, even if the condition is several years old.