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Facial Paralysis

Diagnosis and Management

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Keywords

• Facial nerve palsy • Facial nerve paralysis • Bell's palsy • Synkinesis • Lagophthalmos • Facial reanimation

Key points

- Patients with facial nerve palsy require systematic examination to make a prompt diagnosis and direct appropriate treatment planning.
- Causes of facial nerve palsy include idiopathic Bell's palsy, infection, neoplasm, trauma, congenital origin, and nonmedical or iatrogenic injury.
- Facial examination should evaluate the 4 distinct facial regions or zones (forehead, eyes, midface, and mouth) for static, dynamic, and synkinetic findings.
- Patients with slowly progressive facial paralysis (>72 hours) or paralysis that does not involve the entire hemiface require MRI and further evaluation.
- Long-term management of the paralyzed face benefits from a broad variety of surgical and nonsurgical interventions.

INTRODUCTION

Facial nerve function is essential not only for facial movement and expression, but also for proper sensory functioning of the middle ear, salivation, taste, lacrimation, and ocular surface protection. Facial paralysis creates substantial disabilities, including slurred speech, difficulty eating and drinking, loss of facial expression, and depression. The eye can become dry and painful, resulting in corneal ulceration, perforation, endophthalmitis, and blindness. Knowledge of the nerve's unique intracranial, extracranial, and facial anatomy explain why

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cranial nerve VII is unusually susceptible in comparison with the other cranial nerves. Management requires a correct diagnosis, which can usually be made by the astute clinician based on history and physical examination. Systematic evaluation of the 4 primary facial zones helps to ensure comprehensive evaluation and documentation in the acute setting and over the long course of recovery [1,2]. These facial zones are the forehead, eyes, midface, and mouth. Similarly, the periocular region should be evaluated systematically, using 4 periocular microzones as a method of organization. These microzones are eyebrow, upper eyelid, ocular surface, and lower eyelid. Acute and chronic treatment plans should be tailored based on etiology and degree and rate of recovery.

CONTENT

Facial nerve anatomy

The facial nerve (cranial nerve VII) originates from its nucleus in the lower pons, giving off ventral and dorsal tracts. The ventral tract decussates once within the brainstem to innervate the contralateral lower face. The dorsal tract, conversely, crosses several times so that each side innervates both the ipsilateral and contralateral upper face. The nerve exits the brainstem ventrally at the pontomedullary junction through the internal acoustic meatus. Just proximal to the geniculate ganglion, cranial nerve VII emits the greater superficial petrosal nerve along which parasympathetic fibers travel through the pterygopalatine ganglion to the lacrimal gland to control reflex tear production. The facial nerve then courses through a long, narrow canal in the temporal bone measuring 20 to 30 mm, the longest such transit of any cranial nerve [3]. It emerges from the stylomastoid foramen before entering the parotid gland, where it bifurcates into upper and lower divisions and subsequently into 5 major branches: frontal (also called temporal), zygomatic, buccal, marginal mandibular, and cervical (Fig. 1).

Etiology and presentation

The most common cause of facial nerve palsy in the United States is idiopathic inflammation, first described by Sir Charles Bell in the 1800s and termed Bell's palsy, and it accounts for 62% of new cases [4]. Nerve inflammation and compression within the facial canal leading to ischemia and demyelination is felt to be the mechanism of injury [5]. Higher incidence of herpes simplex virus 1 in endoneural fluid and saliva of affected patients has suggested a correlative relationship [6,7]. The diagnosis of Bell's palsy is one of exclusion, after infection, autoimmune disease, trauma, and compressive lesions have been eliminated by clinical history and physical examination.

Idiopathic Bell's palsy presents with acute onset of unilateral facial paralysis (Fig. 2). Patients are usually between the ages of 15 and 60 years, with rare exceptions [1,2]. Symptoms uniformly peak within 3 days of onset. Indeed, cases that evolve clinically over more than 72 hours suggest a different diagnosis and require MRI (Table 1). The prognosis is good overall, with complete recovery seen in 61% to 94% of patients. The severity of presenting symptoms is inversely related to the recovery potential [8].