Periocular Reconstruction in Patients with Facial Paralysis

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KEYWORDS

• Facial paralysis • Facial nerve • Paralytic lagophthalmos • Eyelid retraction • Ectropion • Exposure keratopathy • Synkinesis

KEY POINTS

- Facial nerve injury often results in orbicularis oculi weakness, which impairs eyelid closure and blink, causing potentially serious ocular consequences.
- Patients with inadequate Bell's phenomenon, corneal anesthesia, and decreased tear production are at high risk for exposure keratopathy in the setting of orbicularis oculi weakness and require a thorough ophthalmologic evaluation.
- If the recovery of facial nerve function is likely, then temporary and conservative measures to provide corneal protection and increase ocular surface lubrication are the mainstays of therapy.
- If the recovery of facial nerve function is unlikely or expected to be prolonged, then surgical rehabilitation of the periocular complex should be considered.
- Current surgical reconstructive procedures are most commonly intended to improve coverage of the eye but cannot restore blink.

INTRODUCTION

Facial nerve injury is one of the most serious complications of parotid diseases and parotid surgery. Transient facial paralysis has been reported in as many as 65% of patients after parotidectomy, whereas permanent facial paralysis has been reported in up to 5% of patients.[1–3] The temporal, zygomatic, and buccal branches of the facial

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nerve innervate the orbicularis oculi muscle, which is the main protractor of the eyelids. Up to 75% of patients with permanent facial nerve injury following parotidectomy also have orbicularis oculi weakness caused by disruption of its innervation.[1]

Impaired function of the orbicularis oculi manifests as incomplete eyelid closure and reduced blink frequency and amplitude.[4] Blink is essential to effective tear film distribution across the corneal surface. Inadequate blink leads to excessive evaporation of the tear film and desiccation of the cornea. Furthermore, in the setting of orbicularis oculi weakness, the action of the eyelid retractors (levator palpebrae superioris and Müller's muscle for the upper eyelid, and inferior tarsal muscle for the lower eyelid) become more pronounced. This condition manifests as upper and lower eyelid retraction, and widening of the vertical palpebral fissure. Reduced orbicularis oculi tone also means less counteraction against the gravitational pull on the lower eyelid, which may result in paralytic ectropion. Together, these dynamic and static changes of the eyelids lead to increased exposure of the ocular surface. If not managed appropriately, patients may develop corneal epithelial defects, ulcers, perforations, and even endophthalmitis. These ocular complications may cause loss of vision and even loss of the eye. Therefore, it is of paramount importance that patients with facial nerve injury involving the periocular complex undergo a thorough ophthalmologic evaluation and, if indicated, periocular reconstruction.

EVALUATION

A thorough history of the nature and time course of facial nerve injury should be elicited. The likelihood for recovery of facial nerve function should be determined. Patients with a history of facial nerve sacrifice during surgery, facial nerve malignancy, or facial nerve injury longer than 12 months are less likely to recover.[5] The presence of ocular symptoms, including change in vision, eye irritation, pain, foreign body sensation, and tearing, should be documented. A past ophthalmologic medical and surgical history should be obtained. Patients with corneal conditions, including dry eye, have less reserve to withstand increased corneal exposure. Monocular patients with facial paralysis affecting the eye with vision must receive special attention.

Physical examination of the periocular complex should begin with observing the patient at repose with spontaneous blink and then with gentle and forced eyelid closure. The integrity of the main corneal protective mechanisms, including frequency and amplitude of blink, orbicularis oculi strength, Bell's phenomenon, corneal sensation (trigeminal nerve), and tear production, must be assessed (**Table 1**). Bell's phenomenon, not to be confused with Bell's palsy, is the spontaneous upward and outward movement of the eye when an individual attempts to close the eyes. This reflex is present in most patients and is a corneal protective feature for patients with facial paralysis. Visual acuity should be obtained. The ocular surface should be examined with fluorescein dye to identify any corneal epithelial defects or ulcers; conjunctival injection should also be noted because this is often a sign of ocular surface exposure (**Fig. 1**). If one or more of the corneal protective mechanisms are impaired, or if the patient has ocular symptoms, decreased visual acuity, or any signs of ocular surface abnormalities, then an urgent ophthalmology consultation is warranted. An evaluation of the static changes of the periocular complex should also be performed to guide reconstructive management (**Fig. 2**, **Table 2**).

MANAGEMENT

The goal of management of the periocular complex in patients with facial paralysis is primarily to protect the ocular surface and preserve visual function and secondarily to