A Review of the Paradoxical Manifestations from Facial Nerve Injury: Epiphora and Hyperlacrimation

Shreya Bhatt, OMS-II, M.S., Cadynce Peltzer, OMS-II, M.S., Irene Kamel, OMS-II, Nourdeen Hussini, OMS-II, Yuri Zagvazdin, Ph.D., and Mohammadali M Shoja, M.D.

The incidence of facial nerve paralysis is roughly 30 per 100,000 persons annually. Despite being frequently idiopathic in nature, as is commonly seen with Bell's palsy, it may also be caused by infection, trauma, or neoplasm. It manifests with lagophthalmos, partial or total facial paresis, and dry eye due to denervation of the lacrimal gland alongside other ocular abnormalities. In this study, we examine the incidence and mechanisms of epiphora and hyperlacrimation in facial nerve injury by reviewing historical publications dating back to the 19th century as well as recent literature. Several mechanisms have been proposed, including aberrant axonal regeneration known to cause the syndrome of "crocodile tears", ocular irritation due to dry eye with disruption of the tear film resulting in increased reflex lacrimation, and reduced drainage of tears as a result of paralysis of the orbicularis oculi muscle and malpositioning of the eyelids. The mechanisms of excess tearing in some types of facial nerve lesions, for example in patients with certain brainstem tumors, remain unknown. Understanding the pathophysiology of epiphora and hyperlacrimation is crucial in guiding management of patients presenting with these aforementioned signs of facial nerve injury. Further experimental and clinical studies focusing on the quantification of tear production and precise localization of facial nerve damage via modalities such as the Schirmer test and electromyography will help improve our understanding of these paradoxical manifestations from facial nerve dysfunction.