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Temporal Bone Histopathology Case of the Month
Idiopathic Facial Paralysis (Bell's Palsy)

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Accumulating evidence indicates that idiopathic facial paralysis (IFP) is caused by a viral inflammation of the proximal portion of the facial nerve in the internal auditory canal (1). The proposed early location of the viral agent, such as herpes simplex 1, herpes zoster virus, and other members of the Herpesvirinae subfamily, has been found in the geniculate ganglion. Herpes simplex or varicella virus DNA has been recovered from the geniculate ganglion in temporal bones of patients demonstrating IFP. However, the majority of imaging studies using enhanced magnetic resonance imaging have indicated that the earliest and most proximal area of enhancement of IFP is in the meatal portion of the facial nerve (2). This location of enhancement is consistent with the intraoperative surgical observations of Fisch and Esslen (3), who proposed that the maximal swelling of the facial nerve occurs proximal to the meatal foramen. On the basis of these observations, they recommended surgical decompression of the meatal foramen and the labyrinthine segment of the facial nerve as the treatment of IFP.

Recent temporal bone observations indicate that the meatal ganglion (MG) of the facial nerve demonstrates ganglion cell degeneration in addition to satellite cell and inflammatory cell infiltration in patients with IFP (4). The input to the MG is carried over the greater superficial petrosal nerve from taste receptors in the soft palate and oropharynx. This region of the pharynx is replete with viral and bacterial organisms in the overall population. It may account for the very high incidence of elevated herpes simplex virus antibodies in the general population worldwide. Figures estimating this incidence in the 70%–80% range by the age of 30 years are common. Therefore, the introduction of a neurotrophic virus (NT) such as herpes simplex virus or zoster through the oropharynx into the MG where latency is established represents a logical explanation for the neuroradiologic findings in IFP. This is the scenario for reactivation of neurotrophic virus in the MG by a stressful event at some later time in the patient's life. The treatment of IFP with

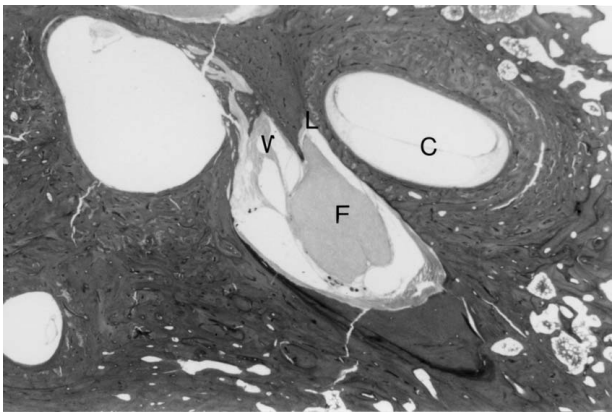

FIG. 1. Low-power view of the internal auditory canal shows marked swelling of the facial nerve (*F*) proximal to the labyrinthine segment of the fallopian canal (*L*). *V*, superior division of the vestibular nerve; *C*, basal turn of cochlea.

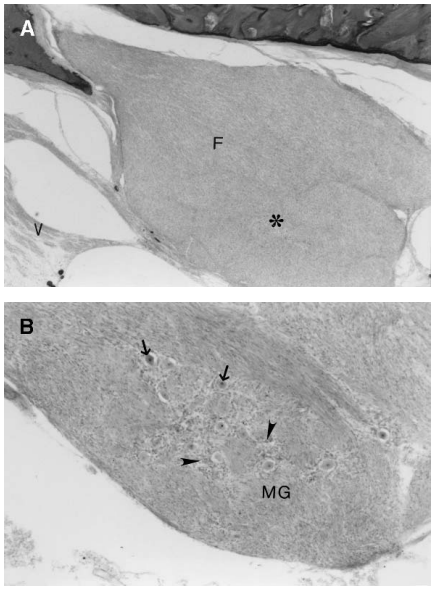

FIG. 2. **A:** The meatal ganglion (*) is located in the edematous portion of the facial nerve (*F*). *V*, vestibular nerve. **B:** The meatal ganglion (*MG*) is shown in this high-power view in which a dense satellite and inflammatory cell infiltrate (*arrowheads*) surround ganglion cells (*arrows*).

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antiviral and with antiinflammatory (steroids) agents has produced superior results to the natural history of IFP or surgical decompression of the facial nerve.

The temporal bone presented here is from a patient with untreated facial paralysis of an unknown period of time. The temporal bone demonstrates marked swelling and edema of the facial nerve proximal to the MG (Fig. 1). The MG contains degenerated and intact ganglion cells surrounded by a heavy infiltration of satellite and inflammatory cells (Fig. 2). The geniculate ganglion appears normal, containing neither degenerated ganglion cells nor an increased density of satellite cells.

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