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Viral Neuropathies in the Temporal Bone

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Contents

VII Preface

XI Acknowledgment

Chapter 1

1 The Biology of Neurotropic Viruses
Gacek, R.R. (Mobile)

Chapter 2

12 Neuroanatomy of the Nerves in the Temporal Bone
Gacek, R.R. (Mobile)

Chapter 3

32 Meatal Ganglionitis: A Pathologic Correlate in Idiopathic Facial Paralysis
Gacek, R.R.; Gacek, M.R. (Mobile)

Chapter 4

54 Vestibular Neuronitis: A Viral Neuropathy
Gacek, R.R.; Gacek, M.R. (Mobile)
Preface

A number of otologic disorders have mystified clinicians over the years. These have been referred to as ‘idiopathic’ indicating lack of a known cause. Although animal models are useful in elucidating basic physiologic mechanisms, recurrent neuropathies (vestibular, facial) of the temporal bone (TB) are unique to humans. Therefore, human TB specimens represent the best source of information providing insight into the pathology of these neuropathic disorders.

For hundreds of years, Bell’s palsy (IFP) and Ménière’s disease (MD) have been regarded as idiopathic. Although displaced otoconia have been implicated in the mechanism of benign paroxysmal positional vertigo, the precise stimulus for degenerated otoconia has also been unknown (idiopathic). Only vestibular neuronitis was assumed to be an inflammatory disorder of the vestibular nerve because of its clinical association with viral-type illnesses and supported by serologic evidence of elevated viral antibodies.

The description of endolymphatic hydrops (EH) in TB from patients with the clinical symptoms of MD [1, 2] provided the impetus for a long series of investigations into the concept of obstruction in longitudinal flow of endolymph to the endolymphatic sac. The theory received support from the experimental demonstration of EH following obstruction of the endolymphatic duct in some animals (guinea pig, gerbil, rabbit) [3, 4]. However, failure to produce EH in nonhuman primates [5] and the absence of vertigo in the successful animal models of EH detracted from the EH theory of MD and accounted for the equivocal results obtained by treatments designed to reduce endolymph.
In a similar way, the previous concept of IFP held that an ischemic event leads to edema of the facial nerve and compression within the surrounding bony canal. Surgical decompression to relieve intraneural pressure did not achieve superior results compared to no treatment in a large number of consecutive patients with IFP [6]. Molecular amplification of herpes simplex virus 1 by PCR on vestibular nerves (ganglia) from patients with MD [7] and IFP [8] supports a viral role in these idiopathic disorders.

We have demonstrated in human TB specimens from patients with IFP, MD, vestibular neuritis and benign paroxysmal positional vertigo a pattern of degenerative changes in the facial nerve (meatal ganglion) and vestibular nerve (and ganglion) which is similar to morphologic changes in herpes zoster of the trigeminal nerve. This evidence has been summarized in the series of reports contained in this volume of *Advances in Otorhinolaryngology*.

Harold F. Schuknecht, MD, predicted a viral cause for MD in his discussion of delayed EH, a form of MD. ‘Assuming that viral labyrinthitis can occur in infants as a subclinical disease that results in delayed endolymphatic hydrops, we may have an explanation for the cause of Ménière’s disease. Viewed in this context the disease entity known as delayed endolymphatic hydrops becomes the missing link in understanding the pathogenesis of Ménière’s disease’ [9]. We dedicate this series of studies to the memory of H.F. Schuknecht whose life-long professional passion was the TB.

Armed with this concept of pathogenesis for the recurrent vestibulopathies, the variable features and unpredictable nature of the ‘three faces’ of vestibular ganglionitis can be understood. An antiviral approach is warranted but will require substantive changes in present-day antiviral pharmaceuticals.

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References


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