Chapter 1

Introduction
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In 1861 Prosper Menière, a French physician, presented a paper in which he described a series of patients with episodic vertigo, hearing loss and tinnitus. Menière concluded that vertigo originates from diseases of the inner ear. Until then, vertigo was thought to be a cerebral symptom similar to epileptic seizures.

At present Menière’s disease is known as a chronic illness characterised by disabling attacks of vertigo, fluctuating hearing loss, tinnitus and often a sensation of aural fullness. In 1938, Hallpike and Cairns and also Yamakawa presented histological findings of temporal bones from patients with Menière’s disease. The common pathological feature was a distension of Reissner’s membrane. Since then an endolymphatic hydrops, which is an excess of endolymph volume, has been generally accepted as the histopathological substrate of Menière’s disease. However, there is also evidence that Menière’s disease may exist without an endolymphatic hydrops (Frayssé et al., 1980). Furthermore, an endolymphatic hydrops may be present without symptoms of Menière’s disease (Vasama and Linthicum, 1999).

The precise mechanism of endolymph volume regulation and pathophysiology of an endolymphatic hydrops remain enigmatic. Various etiologic factors regarding the endolymphatic hydrops have been proposed, including functional or anatomic obstruction of endolymphatic flow, malabsorption of endolymph, genetic defects, vasodilatation, allergy, viral infection and autoimmunity (Paparella and Djalilian, 2002). Furthermore a tiny organ in the inner ear, called the endolymphatic sac, is generally believed to represent one of the primary loci for endolymph volume regulation (Kimura and Schuknecht, 1965; Rask-Andersen et al., 1999).

Studies on experimentally induced endolymphatic hydrops have extensively been performed over the years. The guinea pig is most often chosen as experimental animal model for it has an inner ear closely resembling the human. In the chronic model, the endolymphatic sac is surgically ablated resulting in an endolymphatic hydrops within several days to weeks (Kimura and Schuknecht, 1965). An obvious disadvantage of this model is the destruction of an organ which is essential for inner ear homeostasis.

The model of an acute endolymphatic hydrops involves micro-injection of artificial endolymph into scala media of the cochlea. This refined model leaves the inner ear intact and is moreover verifiable. First introduced by Kitahara (1982), this model provides a useful research tool for examination of the immediate effects of an endolymphatic hydrops on the inner ear.

In this thesis, changes in morphology and cochlear function during and after induction of an acute endolymphatic hydrops are presented. Besides increasing our knowledge regarding pathophysiological mechanisms in Menière’s disease, this thesis also contributes to our knowledge of fundamental processes in the inner ear.

Objectives of this study

In this thesis, changes in morphology and cochlear function during and after induction of an acute endolymphatic hydrops are evaluated. Light microscopy, transmission electron microscopy and
orthogonal-plane fluorescence optical sectioning microscopy (OPFOS) demonstrate morphological characteristics of the cochlea and endolymphatic sac. Distortion products in otoacoustic emissions (DPOAE) and cochlear microphonics (CMDP) were assessed to evaluate cochlear function.

Chapter 2 describes the morphological study of the endolymphatic sac’s epithelia and luminal filling after induction of an acute endolymphatic hydrops.

Chapter 3 describes the evaluation of cochlear function in the acute endolymphatic hydrops model by measuring of the $2f_1-f_2$ DPOAE.

Chapter 4 describes the effects of inner ear pressure changes on the $2f_1-f_2$ DPOAE. The changes in inner ear pressure were induced by injection of artificial perilymph and withdrawal of native perilymph.

Chapter 5 describes the effects of injection of artificial perilymph into scala tympani on the $2f_1-f_2$ DPOAE, $f_2-f_1$ and $2f_1-f_2$ CMDP.

Chapter 6 describes the effects of injection of artificial endolymph into scala media on the $2f_1-f_2$ DPOAE, $f_2-f_1$ and $2f_1-f_2$ CMDP.

Chapter 7 describes the effects of cumulative injections of artificial endolymph into scala media. During and after a “catastrophic” endolymphatic hydrops, both cochlear function and morphology were assessed.

References


