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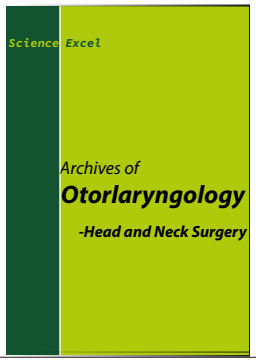
Editorial



The Critical Role of Reissner's Basement Membrane in Meniere's Disease

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Endolymphatic hydrops refers to pathologic distention of the membranous labyrinth [1]. Such hydropic distention constitutes the histologic correlate of the clinical entity known as Meniere's Disease [2]. When these distended membranes rupture they are thought to release potassium ions into the surrounding perilymph. Such potassium ions are suspected to result in a neuronal depolarization that induces the clinical symptom of vertigo in Meniere's disease patients [3]. It should be noted that some authorities question this causal relationship [4]. However, the efficacy of labyrinthectomy in permanently eliminating the vertigo attacks argues strongly for the labyrinth to be the source of the clinical symptoms [5] while leaving open the question of the exact pathophysiological mechanism.

This hydropic membrane distention is not uniform across the labyrinth but preferentially affects the Reissner's membrane of the cochlea duct and the saccular membrane [6]. Optical microscopy reveals that Reissner's membrane and the saccular membrane of the pars inferior labyrinth are much thinner than those of the pars superior chambers. These vestibular cochleo-saccular membranes have a gross optical thickness of ~5 microns in contrast to the membranes of the canal system that have a gross optical thickness of ~25 microns. It should also be noted that the membranes of the pars superior canal system have an additional layer of the fibrous long-spaced collagen superimposed on their perilymphatic surface [7]. This additional layer is what adds to the five-fold greater thickness of the superior labyrinth membranes and their greater structural resistance to distention and rupture [8].

Electron microscopy reveals that Reissner's membrane and the saccular membrane are similar in structure. Both consist of a single layer of epithelial basement membrane of amorphous appearance with minor thickness irregularity. These basement membranes support epithelial cells on the endolymphatic surface and mesothelial cells on the perilymphatic surface [7]. This basement membrane tissue has been identified as a form of collagen designated as Type IV. Type IV collagen has been found to exhibit a mesh-like configuration of fibers randomly oriented within the plane of the membrane rather than aligned in parallel [9].

This random structural arrangement of type IV collagen fibers appears to account for the prominent viscoelastoplastic behavior of these membranes. This behavior, typical of polymers, has been illustrated with a mesh of precipitated collagen [10] and subsequently used to as a model of the cochlea-saccular membranes [11]. This viscoelastic behavior permits these membranes to readily distend preferentially in response to pressure [8] and thereby accommodate modest fluctuations in endolymphatic volume, thereby defending the physiological motion mechanics of the canal system which are dependent on their geometric morphology [12].

Such a random fiber pattern may also be well suited to resist a trans-mural force due to raised endo-lymphatic pressure in contrast to the highly collimated fiber arrangement found in tendons which would logically be better suited to resist longitudinal forces. In fact, normal basement membrane is reported to exhibit surprising mechanical strength given its diminutive size [13]. Electron microscopy of the basement membrane in experimentally induced cochleo-saccular hydrops in otherwise normal guinea pigs has demonstrated that the normal basement membrane of the pars inferior chambers is able to withstand extreme distention without overt rupture as the membrane thins out, only developing microfenestrations at extreme levels of distention [14] .

This stands in contrast to specimens from human cases of Meniere's disease where ruptures of the Reissner's or saccular

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membranes may occur early in the distensile process [15]. Analysis of labyrinthectomy specimens in cases of Meniere's Disease has identified pathological thickening of this basement membrane in the region of the macula [16] suggesting a direct patho-physiologic disturbance in the basement membrane functioning in this disease.

The nature of the disturbance in basement membrane functioning remains unclear but several potential mechanisms are under scrutiny. For example, it should be noted the basement membrane can be the focal target in Alport's syndrome and Goodpasture's syndrome. In Alport's syndrome gene mutations are implicated in the structural malformation of the basement membrane collagen itself, whereas in Goodpasture's syndrome antibodies are directed at structures hidden the basement membranes [17]. A compelling illustration of basement membrane pathology is well demonstrated in pemphigoid where the epithelial BM stains intensely and selectively with immuno-reagents [18]. Finally, defects in basement membrane have also been documented in deficiency states wherein an absence of nidogen or perlecan has been found to cause loss of mechanical strength [19] .

These foregoing features therefore suggest that a process is at work that weakens the normal basement membrane in the pars inferior labyrinth and makes it more susceptible to pressure induced distention and rupture. Taken together these features suggest that pathological functioning of the Type IV collagen of Reissner's basement membrane as well as that of the saccule may play a central role in lesion formation in endolymphatic hydrops and its clinical counterpart Meniere's disease.

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