

Original Article

The Effect of Collagen Viscoelastoplasticity on Reissner's Membrane Displacement: A Graphic Analysis

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BACKGROUND: The aim of this study is to analyze the effect of collagen viscoelastoplasticity on the bulge displacement of Reissner's membrane that is observed in endolymphatic hydrops and Meniere's disease.

METHODS: Viscoelastoplastic load–deformation characteristics for Reissner's membrane were based on a reported collagen polymer model of the cochleo-sacculus membranes. The projected bulge displacements of Reissner's model membrane at key distention points were quantified trigonometrically and plotted graphically.

RESULTS: Initial deformation is characterized by a membrane laxity with substantial stretch at low tension with projected bulge displacement of Reissner's membrane approaching 30%. Intermediate deformation is characterized by a linear membrane stiffness with projected bulge displacement of Reissner's membrane in the range of 30-40%. Terminal deformation is characterized by reduced stiffness with a disproportionate increase in membrane stretch with projected bulge displacement of Reissner's membrane reaching a critical value of 50%, indicating a hemi-circular profile with imminent risk of rupture.

CONCLUSION: This collagen model of membrane viscoelastoplasticity demonstrates that at low pressure significant degrees of bulge displacement up to 30% can occur that may be reversible. The narrower 30-40% range of membrane displacement is one of the increasing deformity but without risk of rupture. Greater displacements approaching 50% indicate that the membrane is reaching a critical hemi-circular configuration with impending rupture.

KEYWORDS: Endolymphatic hydrops, Meniere's disease, Reissner's membrane

INTRODUCTION

Meniere's disease is characterized by attacks of vertigo, unilateral hearing loss, and tinnitus that lasts for a matter of hours. The underlying pathology was first identified in 1938 in patients suffering from a severe form of the disease wherein a marked dilation of the labyrinth membranes was found. In the cochlea portion of the inner ear, it was found that Reissner's membrane bulges pathologically into the scala vestibuli in Meniere's disease as shown in Figure 1.¹ Endolymphatic transmural pressure was presumed to be the driving force behind this membrane displacement. However, the association between transmural pressure and membrane stress near rupture is not known for humans with Meniere's disease. Such pressure elevation may be related to a constricted endolymphatic drainage system in affected individuals or possibly to overactivity in the secretory tissues of the labyrinth. The exact fluid mechanics remain to be elucidated.²

The reactive biomechanics of membrane deformation that give rise to such lesion development also remain unclear. A recently reported model of the cochleo-sacculus membranes³ was based on the reported deformation behavior of a matrix of precipitated collagen fibrils.⁴ This model membrane exhibited a viscoelastoplastic distensile behavior characterized by a sigmoid load–deformation curve and strain rate sensitivity. This was found to be qualitatively similar to the non-linear distensile behavior of human Reissner's membrane in vitro.⁵ The objective of the current study was to demonstrate how the model membrane's viscoelastoplastic properties translate into the hydroptic membrane displacements that are characteristic of Meniere's disease.

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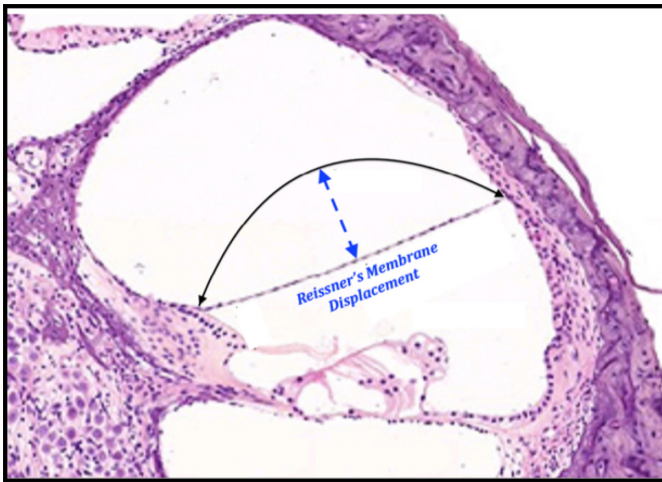


Figure 1. Reissner's membrane bulge displacement was measured at the midpoint.

MATERIALS AND METHODS

The projected sagittal displacements of Reissner's membrane were based on the recently described collagen model noted above. For that model, the collagen matrix was strained in a controlled manner in a tensometer as shown in Figure 2 (used with permission).⁴

The projected sagittal displacements of Reissner's membrane at the midpoint, as shown in Figure 1, were quantified trigonometrically and plotted graphically at key distention points. The formula that relates the magnitude of the midpoint sagittal displacement of Reissner's membrane to the degree of membrane arc length is given in Equation (1):⁶

$$L = [\pi(x^2 + y^2) \arcsin(2xy/(x^2 + y^2))] \div 180(y), \tag{1}$$

where *L* is the arc length of Reissner's membrane, *x* is one-half the initial length of Reissner's membrane (*RM*₀), and *y* is the midpoint sagittal displacement of Reissner's membrane.

This equation is cumbersome to use for the direct calculation of displacement values of Reissner's membrane. However, an online calculator is available⁷ when the initial and distended values of Reissner's membrane are known. Values at key distention points along the viscoelastoplastic load–deformation curve from the model membrane

were used to calculate the corresponding sagittal displacement values of Reissner's model membrane. Such calculated displacements were then depicted graphically in order to clarify how membrane distention might translate into lesion progression in Meniere's disease. Also, it should be noted that the zones designated as toe, linear, and failure in the model reference study⁴ have been relabeled viscoelastic, elastoplastic, and plastic in the current report to emphasize their functional significance.

RESULTS

Table 1 shows stress, strain, and calculated displacement values at key points along the load–deformation curve of a collagen model of Reissner's membrane.

These results are shown graphically in Figure 3. This figure illustrates how the viscoelastoplastic load–deformation curve of the model collagen membrane translates into the progressive histologic displacement of Reissner's membrane when strained at a rate of 38% per minute. This figure shows that at the beginning under zero stress (point A), Reissner's membrane is neither distended nor displaced from its normal anatomic position. At the distention limit of the initial viscoelastic region (point B), a low-stress level of 1.4 kPa has induced a substantial bulge displacement of 29%. At the inflection (point C) in the center of the intermediate elastoplastic transition region, stress is more than doubled to 3.4 kPa, but the bulge displacement is only fractionally higher at 36%, indicating a greater stretch resistance of the membrane. Such stretch resistance continues until the onset of the terminal plastic region where a stress of 5.2 kPa has induced a bulge displacement of 43% (point D). With a small additional increase in stress to 6.3 kPa, stretch resistance decreases in the plastic range until bulge displacement is exactly 50% (point E). Shortly after this, stress actually decreases to 6.0 kPa as the membrane starts to shred (point F), reaches a final bulge displacement of 52%, and then ruptures abruptly at 60% displacement (point G).

DISCUSSION

Summary of Results

Initial membrane laxity in the viscoelastic region is associated with substantial and likely reversible bulge displacements in Reissner's membrane of up to 29%. Increased membrane stiffness in the elastoplastic transition region is associated with a limited increase in bulge displacement up to 43%, and there is no apparent risk of membrane

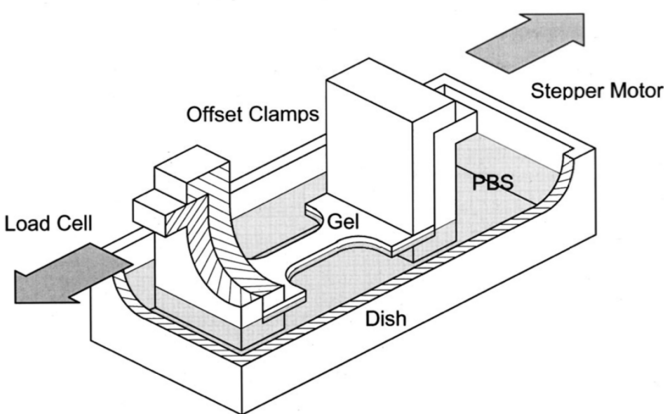


Figure 2. Tissue tensometer schematic showing an "I"-shaped collagen gel specimen, immersed in a physiologically balanced saline solution "PBS," clamped at both ends, and subjected to controlled distention.

Table 1. Model Stress and Strain Values with Calculated Bulge Displacements of Reissner's Membrane at Selected Points Along the Viscoelastoplastic Load–Deformation Curve

Selected Points (Lettered)	Stress (kPa)	Strain (mM/mM)	Displacement (% <i>RM</i> ₀)
Starting point "A"	0	0.0	0
Viscoelastic limit "B"	1.4	0.21	29
Elasto-plastic midpoint "C"	3.4	0.32	36
Plastic onset point "D"	5.2	0.43	43
Peak point "E"	6.3	0.57	50
Shredding begins "F"	6.0	0.61	52
Full rupture "G"	0	---	60

Strain refers to the lengthwise increase in an overall stretch of Reissner's membrane, while displacement refers to the degree of the bulge of Reissner's membrane at the midpoint.

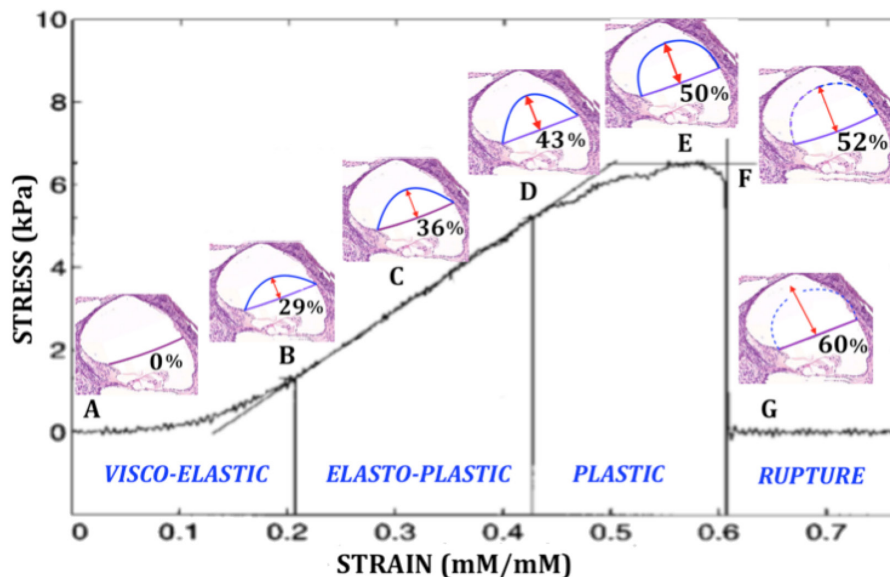


Figure 3. This figure shows the degree of histologic membrane bulge displacement at key points along the viscoelastoplastic load–deformation curve of Reissner's membrane model. The load–deformation curve is based on an in vitro collagen matrix⁴ as described in the text.

rupture. Beyond 43%, membrane stiffness starts to decrease with disproportionate increases in membrane distension and bulge displacement. At peak stress of 6.3 kPa in the plastic region, membrane bulge displacement has reached 50%, indicating a critical hemi-circular configuration, and the risk of rupture is imminent.

Analysis of Results

One of the noteworthy results of this study is that Reissner's model membrane can displace a surprising degree up to 29% at very low stress levels, suggesting that it is very pliant at low degrees of endolymphatic pressure. Moreover, this limited degree of distention is theorized to be associated with spongy distortion of the collagen fiber meshwork in the basal lamina, rather than covalent bond stretching within the collagen molecules.⁸ These findings suggest that early hydropic displacement of Reissner's membrane in the range of less than 30% is viscoelastic and may be reversible. Such reversibility would suggest that because of membrane recoil, there might be a paucity of low-amplitude hydropic lesions found in the temporal bone histology of patients with Meniere's disease.

Moderate displacements in the range of 30–43% correlate with a transition from an alteration in a collagen mesh configuration to covalent bond strain and crosslink disruption with the attendant molecular flow. These findings imply that Reissner's membrane deformations at the beginning of this range are elastic, then as the central inflection point is reached, some degree of plastic deformation begins, and toward the end of the range, the displacements become increasingly plastic and irreversible, and fiber shredding begins. Such incremental plastic displacements are probably cumulative over a lifetime and can be expected to be evidence for unruptured hydropic lesions in temporal bone histology of Meniere's disease.

Greater displacements beyond 43% appear to be fully plastic and correlate with collagen fiber disruption. This process accelerates as membrane bulge reaches a critical value of 50% and then results in complete rupture shortly thereafter at 52% in the model. This suggests that acute covalent bond rupture is the predominant mode of

failure affecting all fibers in a near synchronous way, with initial fiber shredding progressing to a full rupture.

The illustrated findings occurred at a reported strain rate of 38% per minute in the collagen model. Higher strain rates might be expected to induce covalent bond disruption at lower strain levels and thus be associated with earlier onset of incipient membrane failure. At lower strain rates, progressive disruption of cross-linkages between collagen fibrils might be expected to predominate over covalent bond rupture leading to a greater plastic distention of the membrane antecedent to rupture. Thus, in cases of chronic Meniere's disease, temporal bone histology that reveals the presence of ruptures in the face of a low displacement of Reissner's membrane may suggest an acute exacerbation at a high strain rate due to covalent bond disruption while severe distention without rupture argues for a much slower strain rate causing an unrelenting process based on progressive collagen fiber cross-linkage disruption. The conceptual key to reconciling the various degrees of membrane displacement with risk of rupture would appear to lie with the strain rate factor.

Comparison of Results with Published Data

Low-amplitude bulge displacements of Reissner's membrane in the viscoelastic range of less than 30% are seldom seen in isolation in cases of Meniere's disease, suggesting that this degree of displacement may be fully elastic and reversible.⁹ Intermediate-amplitude displacements consist of moderate degrees of Reissner's membrane bulge without rupture in the 30–43% range. These have been designated as a danger zone¹⁰ not because rupture is imminent but because they represent the gradual onset of irreversible plastic deformation of the underlying polymer structure that approaches the plastic zone of impending failure. Such lesions are commonly seen in Meniere's disease specimens.⁹ High-amplitude displacements in the 40–50% range that approach a hemi-circular configuration are at risk of incipient rupture, have ruptured, or have been buttressed by contact with the otic capsule thereby forestalling membrane rupture. The actual risk of rupture associated with such a hemi-circular configuration to Reissner's membrane as identified in this study closely

conforms to a previous theoretical analysis of risk.¹¹ In that study, such a hemi-circular configuration was found to constitute a stress nadir in membrane pressure-bulge function. Distention beyond this "critical point" was found to lead to an exponential increase in membrane stress levels and potential for rupture.

Strain rate effects in the collagen membrane on which the model is based are also of interest.⁴ At lower strain rates, a doubling of rate from 19% to 38% per minute increased stiffness only by 5.7% (from 15.7 kPa to 16.6 kPa). At higher strain rates, a doubling of rate from 192% to 385% increased stiffness by 25.3% (from 20.9 kPa to 25.0 kPa). These data are consistent with other reports of collagen sensitivity to strain rate, wherein such sensitivity is minimal at low strain rates but gets progressively greater above a certain threshold in strain rates.¹²

Also of interest in the reported data for the collagen membrane on which the model is based is the fact that for all reported strain rates the failure strains were approximately the same at 55-59%. This is consistent with membrane rupture just beyond the critical point of membrane displacement where the membrane has assumed a hemispherical profile and stress starts to rise exponentially.

Critical Analysis of Methods

It has been wryly observed that all models are wrong, but some are useful.¹³ An in vitro matrix of precipitated collagen as cited herein is used as a membrane model of viscoelastoplasticity in the Reissner's membrane displacement calculations. The membrane can be expected to thin out to a variable extent during testing in vitro. However, the size of the tissue specimen is microscopic, and more refined measurements addressing this issue have not been reported. As a consequence, engineering stress is used as a first approximation in assessing the membrane biomechanics as the specimen distends.

The model membrane appears to be reasonably accurate, in that it does exhibit viscoelastoplastic behavior consistent with other biological polymers, namely a sigmoid load-deformation curve and strain-rate dependency.¹² It also appears to emulate the load-deformation behavior of actual human Reissner's membrane.⁵

The strain rate associated with the model load-deformation curve is 38% per minute. This strain rate is suspected to exceed the strain rates encountered in naturally occurring Meniere's disease, where lesions evolve over years, not minutes and hours. However, it is possible that acute exacerbations of Meniere's disease may be associated with suddenly higher strain rates and thereby mirror the results presented herein. Overall, the results of these calculations suggest that the degree of Reissner's membrane displacement found in cases of Meniere's disease may have heuristic value as an indicator of impending rupture risk with mild displacements at low risk and hemi-circular displacements at elevated risk. However, any quantitative assessment of the relationship between endolymph volume rate of increase and the strain rate of Reissner's membrane is beyond the scope of this report.

CONCLUSION

This collagen model of Reissner's membrane, when strained at a controlled rate of 38% per minute, demonstrates that low degrees of

bulge displacement of less than 30% are viscoelastic and potentially reversible. The narrow 30-40% middle range of membrane displacement is found to be elastoplastic with increased stiffness but with little risk of rupture. Greater displacements approaching 50% are fully plastic and indicate that the membrane is nearing a critical hemi-circular configuration with the risk of impending rupture. Higher strain rates may anticipate earlier membrane failure at lower distention, while lower strain rates may portend greater distention with delayed risk of membrane failure.

Ethics Committee Approval: No ethical committee approval was necessary as this is a theoretical study and did not involve any human subjects or animals.

Informed Consent: N/A.

Peer-review: Externally peer-reviewed.

Declaration of Interests: The author has no conflict of interest to declare.

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