Doctoral Thesis

Fluid-mechanical model for vestibular responses to sound in presence of a superior canal dehiscence

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Patients with a superior canal dehiscence (SCD) in the inner ear suffer from events of dizziness and vertigo in response to sound, also known as Tullio phenomenon (TP). To date, the mechanisms behind TP remain obscure. When risky surgical interventions appear to be the only means to cure the symptoms, it may be worthwhile to study the underlying mode of operation and possibly devise less invasive methods.

Approaching this medical condition from an engineering perspective, we are faced with an intertwined system of fluids (endolymph and perilymph), elastic structures (membranous labyrinth, cupula, dehiscence) and rigid bone (temporal bone, stapes). In accordance with the so-called ‘third window theory’, we assume that the vibrating stapes causes abnormal perilymph pulsations towards the pathologic ‘window’ in the superior canal of the balance sense. Based on this assumption, we developed a computational model in order to resolve fluid-structure interactions which we expect to arise from such a coupled system.

The simulation results confirm our hypothesis, revealing the occurrence of wave propagation phenomena along the deforming membranous canal. More specifically, we note that two substantially different flows are evolving. First, the deforming labyrinth causes pulsations of the endolymph which lead to rapid vibrations of the cupula in phase with the sound stimulus. Second, these primary pulsations feature a static component, the so-called steady streaming, such that endolymph is continuously driven through the canals in (mostly) ampullofugal direction. Reaching a quasi-steady balance with the opposing cupula, the latter maintains a constant deflection amplitude. Both findings are in agreement with clinical observations on the cupula response in patients with SCD.

Carrying out a sensitivity study, we were able to obtain an analytical fit to match our simulation results in a relevant range of parameters. We coupled the inner-ear dynamics to the corresponding eye response (vestibulo-ocular reflex). The results reveal a ‘sweet spot’ for TP within the audible spectrum which largely coincides with patient data. We found that the underlying mechanisms originate primarily from Reynolds stresses in the fluid, which are weakest in the lower sound spectrum. Additionally, natural variations in the membrane stiffness and the stapes motility are observed to shift the sweet spot. Waves become evanescent above 4-6kHz, such that we cannot expect vestibular responses in that range.
Cover: Illustration of the dehiscent superior canal within the vestibular system of the inner ear; see also Fig. 3.1 on p. 30

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FLUID-MECHANICAL MODEL
FOR
VESTIBULAR RESPONSES TO SOUND
IN PRESENCE OF A
SUPERIOR CANAL DEHISCENCE

A thesis submitted to attain the degree of
DOCTOR OF SCIENCES of ETH ZURICH
(Dr. sc. ETH Zurich)

presented by
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Prof. Dr. L. Kleiser, examiner
Prof. Dr. D. Obrist, co-examiner
Prof. Dr. J. Dual, co-examiner

2015
Abstract

Patients with a superior canal dehiscence (SCD) in the inner ear suffer from events of dizziness and vertigo in response to sound, also known as Tullio phenomenon (TP). To date, the mechanisms behind this phenomenon remain obscure. When risky surgical interventions appear to be the only means to cure the symptoms, it may be worthwhile to study the underlying mode of operation and possibly devise less invasive methods.

Approaching this medical condition from an engineering perspective, we are faced with an intertwined system of fluids (endolymph and perilymph), elastic structures (membranous labyrinth, cupula, dehiscence) and rigid bone (temporal bone, stapes). In accordance with the so-called ‘third window theory’, we assume that the vibrating stapes causes abnormal perilymph pulsations towards the pathologic ‘window’ in the superior canal of the balance sense. Based on this primary assumption, we developed a computational model in order to resolve fluid-structure interactions which we expect to arise from such a coupled system. We discretize our computational domain using the Finite-Volume Method and solve the fluid motion with the Navier-Stokes equations in the Arbitrary Lagrangian-Eulerian (ALE) formulation for moving grids. The dynamics of the embedded membranous labyrinth is considered by a linear-elastic shell model and coupled to the adjacent fluids by an iterative procedure using Aitken relaxation. The cupula dynamics follows a volume-based formulation. Our model is implemented in C++ by a tailor-made code using the open source libraries of OpenFOAM and Armadillo.

The simulation results confirm our hypothesis, revealing the occurrence of wave propagation phenomena along the deforming membranous canal. More specifically, we note that two substantially different flows are evolving. First, the deforming labyrinth causes pulsations of the endolymph which lead to rapid vibrations of the cupula, both in phase with the sound stimulus. Second, these primary pulsations feature a static component, the so-called steady streaming, such that endolymph is continuously driven through the canals in (mostly) ampullofugal direction. Reaching a quasi-steady balance with the opposing cupula, the latter maintains a constant deflection amplitude. Both findings are in agreement with clinical observations on the cupula response in patients with SCD.

Carrying out a sensitivity study and employing dimensional analysis, we are able to obtain an analytical fit to match our simulation results.
in a relevant range of parameters. Through the vestibulo-ocular reflex, the eyes are moving at a speed and direction which corresponds to the cupula displacement and the plane of the superior canal, respectively. We thus coupled the inner-ear dynamics to the corresponding eye motion by means of lumped parameters. The results reveal a ‘sweet spot’ for the Tullio phenomenon within the audible spectrum which largely coincides with patient data from the literature. We find that the underlying mechanisms originate primarily from Reynolds stresses in the fluid, which are weakest in the lower sound spectrum. Additionally, natural variations in the membrane stiffness and the stapes motility are observed to shift the sweet spot on the frequency scale. Waves become evanescent at frequencies above about 4 – 6kHz, such that we cannot expect vestibular responses in that range.

Kurzfassung

Patienten, die eine Dehiszenz im oberen Bogengang des Innenohrs aufweisen, leiden gewöhnlich an Schall-induzierten Drehschwindelattacken, auch bekannt als Tullio-Phänomen. Bisher konnten dessen Hintergründe nicht ausreichend geklärt werden. Da die zur Behandlung notwendigen Operationen sehr risikoreich sind, könnte es sich lohnen, die Funktionsweise des Phänomens zu erforschen, um aus den gewonnenen Erkenntnissen möglicherweise weniger invasive Verfahren abzuleiten.

Indem wir das Krankheitsbild aus der Sichtweise eines Ingenieurs betrachten, identifizieren wir zunächst ein verwobenes System aus Flüssigkeiten (Endolymphe und Perilymphe), elastischen Strukturen (häutiges Labyrinth, Cupula, Dehiszenz) sowie festem Knochen (Schläfenbein, Steigbügel). In Einklang mit der sogenannten ‘Theorie des dritten Fensters’ vermuten wir, dass der Steigbügel die Flüssigkeitssäule der Perilymphe zum krankhaften ‘Fenster’ hin über das Gleichgewichtsorgan hinweg in Schwingung versetzt. Aufbauend auf dieser Grundannahme haben wir ein Rechenmodell entwickelt, das es ermöglicht, Fluid-Struktur-Interaktionen zu erfassen, welche wir in einem derartig gekoppelten System erwarten. Wir diskretisieren unseren Rechenbereich mittels der Finite-Volumen-Methode und lösen die Flüssigkeitsbewegungen mit den Navier-Stokes-Gleichungen anhand einer Formulierung für bewegliche Rechengitter (‘Arbitrary Lagrangian Eulerian’-Methode). Die Dynamik des eingebetteten, häutigen Labyrinths wird durch ein linear-
elastisches Schalenmodell berücksichtigt und mit den umgebenden Flüs-
sigkeiten über eine iterative Prozedur gekoppelt, die auf der Aitken-
Relaxation beruht. Die Dynamik der Cupula orientiert sich am Verdrän-
gungsvolumen. Für unser Modell entwickelten wir einen massgeschnei-
derten C++-Code, der die frei zugänglichen Bibliotheken von OpenFOAM
und Armadillo benutzt.

Die Simulationsergebnisse bestätigen unsere Hypothese, indem sie
Wellenausbreitungsphänomene entlang der sich deformierenden Kanal-
haut aufzeigen. Dabei treten zwei grundlegend verschiedene Strö-
mungsarten auf. Erstens verursacht die bewegliche Kanalhaut eine
pulsierende Bewegung der Endolymphe, welche in schnellen Cupula-
Vibrationen in Phase mit der Schallanregung resultiert. Zweitens be-
werken diese Primärschwingungen eine statische Strömung, so dass die
Endolymphe kontinuierlich in (meist) ampullofugaler Richtung durch
den Kanal bewegt wird. Sobald sich ein quasi-stationäres Gleichgewicht
mit den gegengerichteten Cupula-Kräften einstellt, kann letztere eine
konstante Auslenkung aufrechterhalten. Beide Erkenntnisse stehen in
Einklang mit klinischen Beobachtungen zur Cupula-Reaktion in Patien-
ten mit SCD.

Mittels Dimensionsanalyse und einer Parameterstudie fanden wir
eine analytische Darstellung, welche unsere Simulationsergebnisse
in einem sinnvollen Parameterbereich gut abbildet. Aufgrund
des Vestibulo-Okular-Reflexes bewegen sich die Augen mit einer
Geschwindigkeit und Richtung, welche durch die Cupula-Auslenkung
und die Ebene des oberen Bogengangs bestimmt ist. Hierfür koppel-
ten wir die Innenohr dynamik mit den zugehörigen Augenbewegungen
anhand von in Reihe geschalteten Übertragungsfunktionen. Die Ergeb-
nisse zeigen die Existenz eines Bereichs maximaler Schallsensitivität,
welcher sich mit verfügbaren Patientendaten weitgehend deckt. Die zu
Grunde liegenden Mechanismen werden primär von Reynoldsspannungen
verursacht, welche im niederer Frequenzbereich besonders schwach sind.
Zudem verschieben natürliche Variationen der Steifigkeit des häutigen
Labyrinths sowie der Steigbügelempfindlichkeit die maximale Schallsen-
sitivität in einen anderen Frequenzbereich. Die Wellenausbreitung ver-
schwindet komplett in einem Frequenzbereich oberhalb von ca. 4 – 6kHz,
so dass wir dort keine Reaktionen des Gleichgewichtsorgans erwarten
können.
Acknowledgments

In the course of this project I was supported by numerous people to whom I would like to express my sincerest gratitude.

I was very fortunate to be supervised by Prof. Leonhard Kleiser who provided me with excellent working conditions. He generated a fruitful environment of many liberties, trust and perseverance. I greatly enjoyed our weekly meetings which prodded me to scientific diligence, always accompanied by a good dose of humor.

Most of all, I am indebted to the outstanding guidance of Prof. Dominik Obrist. Not only that he is exceptionally knowledgeable in the inter-disciplinary field of computational biofluiddynamics, he also understands to share his insight in a manner that is both stimulating and enriching. His door was always open for discussions, and his presence made my doctoral journey an unforgettable experience. I deeply admire his amicable style of guidance, ever maintaining a good balance of professionalism and friendship.

Furthermore, I acknowledge the constructive discussions with Prof. Jürg Dual who acted as a co-examiner. His expertise in the dynamics of vibrating structures was the ideal complement to the otherwise fluid-dynamical and medical background of this project.

Addressing a human pathology from an engineering perspective, the cooperation with clinical scientists was essential to the success of this project. I am especially grateful to Dr. Stefan Hegemann from the University Hospital Zurich who generously took the time to introduce me to the Tullio phenomenon and its patients. It also was a major honor to collaborate with Prof. Ian Curthoys from the University of Sydney who would frequently exchange ideas with me on various pathologies of the balance sense.

The code development would not have been possible without the great support from the OpenFOAM community. First of all, I would like to thank Prof. Hrvoje Jasak for admitting me to his precious summer school at the University of Zagreb in 2012. I will never forget these two weeks of intense hands-on programming under the supervision of notably Prof. Željko Tuković whom I deeply respect for his ingenious work. Furthermore, I thank our local ‘self-help support group’ which meets bi-weekly to discuss OpenFOAM-related issues. I am particularly grateful to Prof. Heng Xiao, Dr. Michael Wild, Dr. Wolfgang Wiedemair, Dr. Yvonne Reinhardt and Adrien Lücker.
During my stay at the Institute of Fluid Dynamics (IFD), I felt surrounded by an atmosphere of collegiality, encouragement and friendship. Many thanks to all of you! It was a great pleasure to share my office with Dr. Tobias Luginsland and Dr. Michael John who never tired to fight daily routines with humor and spontaneity. Owing to the tenacity of my sparring partner Andreas Müller, even the most stressful weeks could be mastered pleasantly by incorporating sports into our life at ETH Zurich.

I am much obliged to the staff at IFD, especially to Hans-Peter Caprez who supplied me with immediate support when technical computer problems had arisen. Furthermore I am very grateful to Bianca Maspero, officially the secretary, but secretly the ever-smiling soul of the institute. Thanks to our librarian, Sonia Atkinson, not only each Christmas party became a memorable event.

Thanks also go out to all my Bachelor and Master students who had their own share in the successful outcome of this project, most notably Carl-Friedrich Benner who accompanied me during the final phase of my thesis. Through them I began to develop my own way of supervision and guidance.

I would like to express my uttermost gratitude to my parents, Inge and Franz. With their unconditional love, they followed me throughout the many steps of my education and gave me the necessary freedom to prosper and mature. I am very proud to call Maximilian my brother whose unbroken backing has formed a deep foundation of trust.

Finally, I want to address a few words to the person who owns my heart. Only her loving care and continuous support made this greatest endeavor of mine possible in the first place. She has shown me what matters most in life, and never ceases to delight me by her amiable nature. Sighi, this work is dedicated to you.

Zürich, April 2015

Bernhard Johann Grieser

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### List of Abbreviations and Symbols

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### Medical Glossary

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List of Abbreviations and Symbols

This list is structured in six sections, with each section first listing symbols, then Roman and calligraphic characters in alphabetical order, followed by letters of the Greek alphabet.

Abbreviations

<table>
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<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ALE</td>
<td>Arbitrary Lagrangian Eulerian</td>
</tr>
<tr>
<td>BCH</td>
<td>Bone Conduction Hyperacusis</td>
</tr>
<tr>
<td>CT</td>
<td>Computed Tomography</td>
</tr>
<tr>
<td>dB</td>
<td>Decibel (logarithmic unit of SPL)</td>
</tr>
<tr>
<td>DILU</td>
<td>Diagonal Incomplete-LU</td>
</tr>
<tr>
<td>EL</td>
<td>Endolymph</td>
</tr>
<tr>
<td>FSI</td>
<td>Fluid-Structure Interaction</td>
</tr>
<tr>
<td>FVM</td>
<td>Finite-Volume Method</td>
</tr>
<tr>
<td>GAMG</td>
<td>Geometric-Algebraic Multi-Grid</td>
</tr>
<tr>
<td>HC</td>
<td>Horizontal/Lateral Semicircular Canal</td>
</tr>
<tr>
<td>HL</td>
<td>Hearing Loss</td>
</tr>
<tr>
<td>HOT</td>
<td>Higher Order Terms</td>
</tr>
<tr>
<td>HS</td>
<td>Hennebert Sign</td>
</tr>
<tr>
<td>ML</td>
<td>Membranous Labyrinth</td>
</tr>
<tr>
<td>PBiCG</td>
<td>Preconditioned Bi-Conjugate Gradient</td>
</tr>
<tr>
<td>PC</td>
<td>Posterior Semicircular Canal</td>
</tr>
<tr>
<td>PISO</td>
<td>Pressure-Implicit with Splitting of Operators</td>
</tr>
<tr>
<td>PL</td>
<td>Perilymph</td>
</tr>
<tr>
<td>SC</td>
<td>Superior/Anterior Semicircular Canal</td>
</tr>
<tr>
<td>SCC</td>
<td>Semicircular Canal</td>
</tr>
<tr>
<td>SCD</td>
<td>Superior Canal Dehiscence</td>
</tr>
<tr>
<td>SPL</td>
<td>Sound Pressure Level (in dB)</td>
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### List of Abbreviations and Symbols

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<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>TP</td>
<td>Tullio Phenomenon</td>
</tr>
<tr>
<td>VEMP</td>
<td>Vestibular Evoked Myogenic Potential</td>
</tr>
<tr>
<td>vHIT</td>
<td>Video Head Impulse Test</td>
</tr>
<tr>
<td>VOR</td>
<td>Vestibulo-Ocular Reflex</td>
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### Indices

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<td>II</td>
<td>‘Passive’ arm, as defined in Fig. 3.1</td>
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<table>
<thead>
<tr>
<th>Notation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>(·)'</td>
<td>Dimensionless quantity</td>
</tr>
<tr>
<td>(·)₀</td>
<td>Initial/default/unperturbed value of (·)</td>
</tr>
<tr>
<td>(·)ₓ</td>
<td>Related to the cupula motion</td>
</tr>
<tr>
<td>(·)ₑ</td>
<td>Related to the EL motion</td>
</tr>
<tr>
<td>(·)ₓ̂</td>
<td>Related to the fluid (EL/PL) motion</td>
</tr>
<tr>
<td>(·)ᵧ</td>
<td>Related to the grid motion</td>
</tr>
<tr>
<td>(·)ₓ</td>
<td>Related to the horizontal eye motion</td>
</tr>
<tr>
<td>(·)ₖ</td>
<td>Related to the Korteweg theory</td>
</tr>
<tr>
<td>(·)ₚ</td>
<td>Related to the PL motion</td>
</tr>
<tr>
<td>(·)ₛ</td>
<td>Related to the solid (ML) motion</td>
</tr>
<tr>
<td>(·)ₜ</td>
<td>Related to the torsional eye motion</td>
</tr>
<tr>
<td>(·)ᵥ</td>
<td>Related to the vertical eye motion</td>
</tr>
<tr>
<td>(·)ᵣ</td>
<td>Value of (·) at the ML interface</td>
</tr>
<tr>
<td>(·)ᵥ</td>
<td>Related to viscous effects</td>
</tr>
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### Mathematical notations

<table>
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<tr>
<th>Notation</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>‾(·)</td>
<td>Averaged over one period of time</td>
</tr>
<tr>
<td>ũ(·)</td>
<td>Unrelaxed quantity during Aitken subiterations</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>(·)</td>
<td>One-dimensional numerical vector</td>
</tr>
<tr>
<td>Symbol</td>
<td>Description</td>
</tr>
<tr>
<td>--------</td>
<td>-------------</td>
</tr>
<tr>
<td>( \cdot )</td>
<td>Two-dimensional numerical matrix</td>
</tr>
<tr>
<td>( \Delta (\cdot) )</td>
<td>Difference between two states of ( \cdot )</td>
</tr>
<tr>
<td>( \nabla \cdot (\cdot) )</td>
<td>Divergence</td>
</tr>
<tr>
<td>( \nabla (\cdot) )</td>
<td>Gradient</td>
</tr>
<tr>
<td>( \nabla^2 (\cdot) )</td>
<td>Laplacian</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>( b )</td>
<td>Vector quantity (bold font)</td>
</tr>
<tr>
<td>( b_x )</td>
<td>Axial component of vector ( b )</td>
</tr>
<tr>
<td>( b_r )</td>
<td>Radial component of vector ( b )</td>
</tr>
<tr>
<td>( \mathcal{F}(\cdot) )</td>
<td>Function representing the fluid solver (Fig. 4.3)</td>
</tr>
<tr>
<td>( i )</td>
<td>Imaginary unit</td>
</tr>
<tr>
<td>( J_0(\cdot) )</td>
<td>Bessel function of first kind and zeroth order (App. A)</td>
</tr>
<tr>
<td>( n )</td>
<td>Surface normal</td>
</tr>
<tr>
<td>( S(\cdot) )</td>
<td>Function representing the solid solver (Fig. 4.3)</td>
</tr>
<tr>
<td>( (\cdot)_t )</td>
<td>Temporal derivative</td>
</tr>
<tr>
<td>( (\cdot)^n_i )</td>
<td>Evaluated at time ( t=t^n ) during ( i^{th} ) subiteration</td>
</tr>
<tr>
<td>( (\cdot)</td>
<td>_x )</td>
</tr>
<tr>
<td>( (\cdot)_x )</td>
<td>Spatial derivative</td>
</tr>
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**Numerical parameters**

<table>
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<td>Integral operator, def. in (4.5)</td>
</tr>
<tr>
<td>( B )</td>
<td>Integral operator, def. in (4.5)</td>
</tr>
<tr>
<td>( \varepsilon )</td>
<td>Solution vector, def. in (4.2)</td>
</tr>
<tr>
<td>( G \equiv )</td>
<td>Coefficient matrix, def. in (4.12)</td>
</tr>
<tr>
<td>( I \equiv )</td>
<td>Identity matrix</td>
</tr>
<tr>
<td>( K \equiv )</td>
<td>Coefficient matrix, def. in (4.13)</td>
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<tr>
<td>( M \equiv )</td>
<td>Coefficient matrix, def. in (4.8a)</td>
</tr>
<tr>
<td>( N \equiv )</td>
<td>Coefficient matrix, def. in (4.8b)</td>
</tr>
<tr>
<td>( N )</td>
<td>No. of computational elements (Fig. 4.1)</td>
</tr>
<tr>
<td>Symbol</td>
<td>Definition</td>
</tr>
<tr>
<td>--------</td>
<td>-----------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>$N_\perp$</td>
<td>No. of non-orthogonality corrections (Fig. 4.2)</td>
</tr>
<tr>
<td>$q$</td>
<td>Right-hand-side vector, def. in (4.3a)</td>
</tr>
<tr>
<td>$\text{res}_i$</td>
<td>Aitken residual at $i^{th}$ substep, def. in (4.25)</td>
</tr>
<tr>
<td>$S$</td>
<td>Surface area of a volume element</td>
</tr>
<tr>
<td>$\varepsilon_A$</td>
<td>Aitken residual, def. in (4.30)</td>
</tr>
<tr>
<td>$\varepsilon_p$</td>
<td>Pressure residual in Poisson equation (PISO)</td>
</tr>
<tr>
<td>$\varepsilon_u$</td>
<td>Velocity residual in momentum equation (PISO)</td>
</tr>
<tr>
<td>$\varepsilon_V$</td>
<td>Beat volume residual, def. in (4.32)</td>
</tr>
<tr>
<td>$\varphi$</td>
<td>Surface flux, def. in (4.17)</td>
</tr>
<tr>
<td>$\theta$</td>
<td>Aitken relaxation parameter, def. in (4.26b)</td>
</tr>
<tr>
<td>$\vartheta$</td>
<td>Generalized Crank-Nicolson coefficient, def. in (4.10)</td>
</tr>
</tbody>
</table>

**Dimensional quantities**

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>$A$</td>
<td>Cross-sectional area $[m^2]$</td>
</tr>
<tr>
<td>$C_d$</td>
<td>Linear operator for damper elements $[kg m^{-2} s^{-1}]$</td>
</tr>
<tr>
<td>$C_m$</td>
<td>Linear operator for mass elements $[kg m^{-2}]$</td>
</tr>
<tr>
<td>$C_s$</td>
<td>Linear operator for spring elements $[kg m^{-2} s^{-2}]$</td>
</tr>
<tr>
<td>$c$</td>
<td>Wave speed $[m s^{-1}]$</td>
</tr>
<tr>
<td>$c_t$</td>
<td>Speed of sound in the fluid $[m s^{-1}]$</td>
</tr>
<tr>
<td>$c_L$</td>
<td>Longitudinal wave speed $[m s^{-1}]$</td>
</tr>
<tr>
<td>$d$</td>
<td>Right-hand-side expression in (3.32) $[Pa]$</td>
</tr>
<tr>
<td>$E$</td>
<td>Young’s modulus of membrane $[Pa]$</td>
</tr>
<tr>
<td>$f$</td>
<td>Sound frequency $[Hz]$</td>
</tr>
<tr>
<td>$f_\pi$</td>
<td>Ring frequency $[Hz]$</td>
</tr>
<tr>
<td>$g$</td>
<td>Stapes boundary condition (pressure gradient) $[N m^{-3}]$</td>
</tr>
<tr>
<td>$h$</td>
<td>Membrane thickness $[m]$</td>
</tr>
<tr>
<td>$K_c$</td>
<td>Cupula stiffness ($\Delta p_c/V_c$) $[Pa m^{-3}]$</td>
</tr>
<tr>
<td>$K_s$</td>
<td>Proportionality constant ($\alpha_t/\bar{U}_e$) $[deg m^{-1}]$</td>
</tr>
<tr>
<td>$K_\alpha$</td>
<td>VOR constant ($\alpha_t/V_c$) $[deg m^{-3} s^{-1}]$</td>
</tr>
<tr>
<td>Symbol</td>
<td>Description</td>
</tr>
<tr>
<td>--------</td>
<td>-------------</td>
</tr>
<tr>
<td>$L_1$</td>
<td>Axial location of dehiscence</td>
</tr>
<tr>
<td>$L_2$</td>
<td>Length of semicircular canal</td>
</tr>
<tr>
<td>$p$</td>
<td>Fluid pressure</td>
</tr>
<tr>
<td>$R$</td>
<td>Major radius of endolymph torus</td>
</tr>
<tr>
<td>$r$</td>
<td>EL: radial coordinate</td>
</tr>
<tr>
<td>$r$</td>
<td>ML/PL: absolute membrane position</td>
</tr>
<tr>
<td>$r_0$</td>
<td>Minor radius of endolymph torus</td>
</tr>
<tr>
<td>$r_d$</td>
<td>Radius of dehiscence (Fig. 1.12)</td>
</tr>
<tr>
<td>$r_{SC}$</td>
<td>Minor radius of perilymph torus (Fig. 1.12)</td>
</tr>
<tr>
<td>$T$</td>
<td>Period of time</td>
</tr>
<tr>
<td>$t$</td>
<td>Time coordinate</td>
</tr>
<tr>
<td>$U$</td>
<td>Amplitude of bulk fluid velocity</td>
</tr>
<tr>
<td>$u$</td>
<td>Fluid velocity</td>
</tr>
<tr>
<td>$V_c$</td>
<td>Displaced cupula volume</td>
</tr>
<tr>
<td>$x$</td>
<td>Axial coordinate</td>
</tr>
<tr>
<td>$\alpha_t$</td>
<td>Angular velocity (head or eye)</td>
</tr>
<tr>
<td>$\delta_{\nu}$</td>
<td>Stokes boundary layer thickness (A.3)</td>
</tr>
<tr>
<td>$\epsilon_p$</td>
<td>Pseudo-viscous damping in the perilymph</td>
</tr>
<tr>
<td>$\eta$</td>
<td>Radial membrane deflection</td>
</tr>
<tr>
<td>$\bar{\eta}$</td>
<td>Radial membrane deflection envelope</td>
</tr>
<tr>
<td>$\nu_t$</td>
<td>Kinematic viscosity of fluids</td>
</tr>
<tr>
<td>$\varphi$</td>
<td>Flux</td>
</tr>
<tr>
<td>$\rho$</td>
<td>Density</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>Attenuation length (5.7)</td>
</tr>
<tr>
<td>$\tau$</td>
<td>Time constant</td>
</tr>
<tr>
<td>$\omega$</td>
<td>Angular sound frequency, $\omega = 2\pi f$</td>
</tr>
</tbody>
</table>
## List of Abbreviations and Symbols

### Dimensionless parameters

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$D$</td>
<td>Size of the dehiscence (Fig. 1.12)</td>
</tr>
<tr>
<td>$K$</td>
<td>General model constant (Tab. 5.1)</td>
</tr>
<tr>
<td>$K_0$</td>
<td>Korteweg number, def. in (3.37)</td>
</tr>
<tr>
<td>$\mathcal{L}$</td>
<td>Fluid loading, def. in (3.38)</td>
</tr>
<tr>
<td>$\mathcal{R}$</td>
<td>Ramp function, def. in (C.1)</td>
</tr>
<tr>
<td>$Re$</td>
<td>Reynolds number, def. in (3.39)</td>
</tr>
<tr>
<td>$W$</td>
<td>Pseudo-viscosity correction, def. in (A.5)</td>
</tr>
<tr>
<td>$W_0$</td>
<td>Womersley number, def. in (3.40)</td>
</tr>
<tr>
<td>$\beta$</td>
<td>Perilymph dominance, def. in (3.41)</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>Density ratio, def. in (3.42)</td>
</tr>
<tr>
<td>$\kappa_1$</td>
<td>Dehiscence location, def. in (3.43)</td>
</tr>
<tr>
<td>$\kappa_2$</td>
<td>Length of semicircular canal, def. in (3.44)</td>
</tr>
<tr>
<td>$\nu_s$</td>
<td>Poisson ratio of membrane ($\nu_s = 0.5$)</td>
</tr>
<tr>
<td>$\Pi$</td>
<td>Frequency relative to ring frequency $f_\pi$, def. in (5.21)</td>
</tr>
<tr>
<td>$\phi$</td>
<td>Perilymph fraction entering the cochlea (Fig. 1.12)</td>
</tr>
<tr>
<td>$\Psi_L$</td>
<td>Attenuation length w.r.t. canal length $L_2$, def. in (5.8)</td>
</tr>
<tr>
<td>$\Psi_\lambda$</td>
<td>Attenuation length w.r.t. wavelength $\lambda$, def. in (5.9)</td>
</tr>
</tbody>
</table>
Medical Glossary

A list of medical terms with reference to their text location.

Inner Ear

Afferents  Carriers of electric nerve signals (p. 3)
Dehiscence  Lack of bone above one of the → Semicircular Canals which acts as a → Third Window of the inner ear (see Fig. 1.8)
Dura Mater  A layer of connective tissue that covers the → Temporal Bone (see Fig. 3.1)
Endolymph  Lymphatic fluid within the → Membranous Labyrinth of the inner ear (see Fig. 1.4)
Membranous Labyrinth  Elastic structure of similar shape as the surrounding cavity inside the → Temporal Bone (see Fig. 2.2)
Perilymph  Lymphatic fluid between → Membranous Labyrinth and → Temporal Bone (see Fig. 1.4)
Temporal bone  The bone that hosts the inner ear (see Fig. 1.4)
Third Window  See → Dehiscence

Vestibular System

Ampulla  Widened section at the end of each → Semicircular Canal which hosts the → Cupula
Anterior Canal  Alternative expression for the → Superior Canal
Common Crus  Common arm of → Posterior and → Superior Canal
Cupula  Gelatinous membrane which hosts sensory hair cells to detect angular motion
Horizontal Canal  The → Semicircular Canal which coincides with the horizontal plane of the head
Lateral Canal  Alternative expression for the → Horizontal Canal
Macula  Sensory entity which detects linear accelerations; located in the →Utricle and →Sacculus
Otoconia Calcite crystals sitting on top of the →Maculae
Posterior Canal Vertically oriented →Semicircular Canal
Sacculus Hosts the saccular →Macula and is of similar function and shape as the →Utricle
Semicircular Canal Slender cavity of semicircular shape inside the →Temporal Bone
Superior Canal Vertically oriented →Semicircular Canal
Utricle Hosts the utricular →Macula and is filled with →Endolymph
Vestibule →Perilymph-filled cavity to which the →Semicircular Canals are connected

Detailed visualizations of the vestibular system are given in Figs. 1.4 and 1.5.

Cochlea

Apex Innermost turn of the cochlear spiral where →Scala Vestibuli and →Scala Tympani concur
Basilar Membrane Elastic membrane of axially varying stiffness (see Fig. 1.2); performs a spectral decomposition of the acoustic sound signal
Organ of Corti Sensory organ which actively amplifies and converts the motion of the →Basilar Membrane into electric nerve signals (see Figs. 1.2, 1.4)
Scala Vestibuli →Perilymph-filled, coiled duct from the →Vestibule to the cochlear →Apex (see Fig. 1.2)
Scala Tympani →Perilymph-filled, coiled duct from the cochlear →Apex to the →Round Window (see Fig. 1.2)
### Middle Ear

**Incus**
First middle-ear ossicle; connects to the
→ *Tympanic Membrane*

**Malleus**
Second middle-ear ossicle; creates a lever arm between the → *Incus* and the → *Stapes*

**Oval Window**
Elastic membrane covering the opening between the middle ear and the vestibular → *Perilymph*; excited by the oscillating → *Stapes* footplate

**Round Window**
Elastic membrane covering the opening between the middle ear and the cochlear → *Perilymph*

**Stapes**
Third middle-ear ossicle; connects to the inner ear

**Tympanic Membrane**
Separates the external auditory canal from the middle ear

All listed entries for the middle ear appear in Fig. 1.3 on p. 5

### Other

**Hennebert Sign**
Pressure-induced vertigo (see p. 14); named after Hennebert (1911)

**Nystagmus**
Saccadic eye movements (see p. 14): slow-phase eye motions are followed by fast reset maneuvers of the eye

**Superior Canal Dehiscence**
Pathologic condition of a non-existent or markedly thin roof above the → *Superior Canal* (see Fig. 1.8); first classified by Minor et al. (1998); see → *Dehiscence*

**Tullio Phenomenon**
Sound-induced vertigo; named after Tullio (1929)

**Valsalva Maneuver**
Pressure application to the inner ear windows to provoke the → *Hennebert sign* (see p. 14)

**Vestibulo-Ocular Reflex**
Fastest human reflex linking → *Cupula* mechanics to the eye response (vision stabilizer, see p. 7)
Chapter 1

Introduction

With a total span of only about one centimeter, the human inner ear is host to a complex system of intertwining structures. These exhibit multi-physics across a broad bandwidth of time scales, ranging from the detection of slow head rotations on the order of seconds, to high-frequency sound waves of far less than a millisecond period. The physics of the inner ear involves fluid mechanics, solid mechanics, chemical and electrical processes.

Although its mechanisms have been fascinating scientists for more than 150 years, many aspects are still not well understood. This may partly relate to its poor accessibility (e.g. for \textit{in vivo} measurements or surgery), as the inner ear is rather a void carved into bone. Most studies therefore resort to \textit{in vitro}, theoretical or computational models. Such models reduce the anatomical complexity and may result in a decreased predictability. However, they also enable us to isolate and understand certain effects of interest, even beyond the organ’s physiological operation limits.

The present work approaches the inner ear from such a computational modeling perspective. It investigates the case of the \textit{superior canal dehiscence} (SCD) syndrome, a comparably rare pathology in which the roof of the bony encasing of the balance sense is either disrupted or locally dehiscent. Patients who suffer from this condition often report sound-induced vertigo, also known as the \textit{Tullio phenomenon} (TP). A typical scenario would be the patient’s response during exposure to a screaming child: immediate onset of dizziness and vertigo, accompanied by upward-torsional eye motions.

So ‘somehow’ the high-frequency acoustic energy might transfer into the low-frequency domain of the balance sense, invoking a vestibular response beyond its usual operating range. It has also been observed that such responses are very patient-specific, such that some patients may experience symptoms within a different frequency range than others. This indicates that the underlying mechanism most likely remains the same, although the individual manifestations can vary to some extent, e.g. due to different morphologies or material properties of the inner ear.
The computational model developed herein is justified on similar grounds: it does not claim quantitative accuracy down to the individual patient, but it tries to carve out the qualitative TP mechanics and to reveal the key parameters which enable such a deception of the balance sense by sound. The present study is based on ideas developed in the habilitation treatise of Obrist (2011) at the Institute of Fluid Dynamics at ETH Zurich.

In order to understand the need for an engineering approach and to explain the clinical relevance of this thesis (Section 1.4), we begin with an introduction into the anatomy of the inner ear (Section 1.1). It is followed by a short summary of previously employed methods to model the fluid and solid mechanics of the balance sense (Section 1.2). A description of the SCD pathology - along with a literature review on recent computational and experimental findings - is given in Section 1.3.

1.1 Anatomy of the inner ear

The inner ear is located in the temporal bone and is commonly associated with the hearing sense, as the name already suggests. Topographically,
1.1 Anatomy of the inner ear

One part of the inner ear - the cochlea - forms the last unit (‘sound sensor’) in the hearing chain, after the outer ear (‘sound collector’) and the middle ear (‘sound amplifier’), see Fig. 1.1. However, the inner ear also features the balance sense with its characteristic shape of three semicircular canals (SCC), which is able to detect angular and translational motions of the head.

Both organs, hearing and balance, share the same fluid spaces within the cavities of the temporal bone. Together, they consume about a centimeter of space in each direction. The cavities are filled with two water-like, lymphatic fluids - the endolymph (EL) and the perilymph (PL). These differ in their ion content and thus sustain an electric potential across their common interface, the membranous labyrinth (ML). Sensory hair cells can make use of this electric potential, and ultimately stimulate the vestibular or cochlear nerve afferents according to their own mechanical displacement (related to the perceived head motion, or to sound excitation).

The following two sections briefly describe the auditory and vestibular system, respectively.

![Figure 1.2: Structure of the human ear (with focus on the auditory system).](http://www.britannica.com)

1.1.1 Auditory system

Human hearing ranges from frequencies of about 20Hz up to 20kHz, the ultrasound limit at high-pitched tones. Sound is mechanically perceived in the auditory system.
The auditory system consists of the outer ear, the middle ear and the cochlear part of the inner ear (Fig. 1.2). Incoming sound waves travel through the external auditory canal and reach the tympanic membrane, a firmly taut, thin structure which vibrates at the sound frequency. These membrane deflections trigger the lever arm system of the middle-ear ossicles, such that malleus and incus amplify the oscillations towards the stapes, the last middle-ear ossicle.

The middle ear (Fig. 1.3) is filled with air, and its bone encasing features four ‘holes’: one opening towards the outer ear (occluded by the tympanic membrane), two openings towards the inner ear (i.e. the oval and the round window) and the only unobstructed opening into the Eustachian tube (or auditory tube) which allows for pressure equalization through the nasal cavity. When the stapes sets to oscillate, it displaces the perilymph behind the oval window.

As the inner-ear fluids can be considered incompressible media surrounded by temporal bone, the perilymph displacement yields an anti-phased oscillation of the round window membrane by the principle of mass conservation. It creates an oscillating fluid column within the cochlear duct and invokes traveling waves along the basilar membrane which features a monotonically decreasing stiffness with axial distance. Depending on the sound frequency, a specific location along the basilar membrane will exhibit resonance phenomena with maximal membrane-deflection amplitudes and thereby stimulate the local sensory hair cells. From a mathematical point of view, the cochlea may hence be regarded as a ‘real-time Fourier transformer’ by performing a spectral analysis on the energy content of the incoming sound waves.

Details on the cochlear mode of operation as well as further reading on the anatomy of the auditory system can be found in Encyclopædia Britannica Inc. (2015), as well as in Edom (2013).

1.1.2 Vestibular system

The vestibular system consists of three semicircular canals (SCC) of mutually orthogonal orientation, i.e. the horizontal (HC), posterior (PC) and superior (SC) canal (cf. Fig. 1.4). These canals are predominantly filled with perilymph (PL) which merges in the larger cavity of the vestibule, from where it seamlessly shades off into the cochlear scalae of the auditory system. Embedded into the PL, and kept in place by fibrous
1.1 Anatomy of the inner ear

strings, the membranous labyrinth (ML) retains the shape of its host cavity by an elastic structure of three slender ducts that merge into a larger volume (utricle) inside the vestibule, and it is filled with endolymph (EL). The utricle connects to the similarly shaped saccule via the valve of Bast which enables endolymph to be shunted from the cochlear duct through the saccule into the utricle and the canals (Brown et al., 2013). In humans, the endolymph lumen consumes merely a tenth of the total SCC cross-section (Curthoys et al., 1977).

Linear acceleration sensors

The utricle and saccule each host a slightly curved, flat and ellipsoid structure, the utricular or saccular macula, respectively (see Fig. 1.5). These are oriented perpendicularly to each other, and they consist of a soft and gelatinous structure, the otolithic membrane, which is located on the inner surface of the ML walls. On the endolymph side of the otolithic membrane, dense calcite crystals - the so-called otoconia - are imprinted on the surface. As they are heavier than the surrounding media, gravitational as well as inertial forces during translational head movements will cause them to shear the otolithic membrane in opposite direction to the head acceleration. The supple structure bends and deflects sensory hair cells which innervate the otolithic membrane. Upon deflection, these cells change their firing rate on the vestibular nerve.
endings. Featuring a redundant system of many such hair cells with different orientation in space (within the respective macula), the signal decoding of a translational head motion follows vector summation principles (Fitzpatrick & Day, 2004).

Angular acceleration sensors

In the vicinity of the utricle, each SCC in the ML comprises one spherically enlarged section, the ampulla (see Fig. 1.5). Its lumen is completely occluded at all times (Oman & Young, 1972) by a supple, gelatinous
membrane - the so-called cupula. Sensory hair cells (cilia) innervate the cupula and connect to the vestibular afferents. During head rotation, endolymph accelerates with the moving ML walls such that a Stokes flow develops within \[ \text{(Damiano & Rabbitt, 1996)} \]. This flow displaces the elastic cupula and, with it, the embedded cilia accordingly. Similar to the translational receptors of the maculae, a head rotation is perceived by the bent cilia of the cupula which stimulate the vestibular afferents directly. With regard to the present thesis, one may remark at this point that there are other factors which could enable an endolymph flow in the SCCs - and hence invoke the sensation of angular motion (e.g. during caloric reflex tests, as described by \[ \text{Steer et al., in 1967} \]).

**Vestibulo-ocular reflex**

The *vestibulo-ocular reflex* (VOR) is the fastest human reflex, featuring a response time of less than ten milliseconds \[ \text{(Aw et al., 1996)} \]. It acts as a vision stabilizer by making use of the information collected by the balance sense: any rotation of the head will be compensated by an eye motion in the opposite direction of equal angular velocity and allow the fixation of a point in space. If the head accelerates, for instance, clock-
wise about its vertical axis, endolymph moves counterclockwise inside the HC and displaces the cupula. As the instantaneous cupula position correlates directly with the instantaneous angular head velocity, the VOR is wired such that it can control the eye velocity by adjusting the ocular muscles. The VOR may hence be regarded as an unmistakable sign of endolymph motion.

1.2 Mechanics of the vestibular system

The mechanics of an intact vestibular system is exclusively concentrated on the endolymph (EL) domain inside the membranous labyrinth (ML). Its sole purpose is the detection and translation of rotatory and translatory head motions into a nerve signal which can be further processed by the brain or by reflexes like the VOR (Section 1.1.2). It is commonly understood that a head motion results in a relative flow of the endolymph (fluid mechanics) which displaces a gelatinous membrane and its innervating, sensory hair cells (solid mechanics). As the membrane deflections from everyday head maneuvers are roughly proportional to the desirable motion quantity to be measured, the perception mechanism can be broken down into a combination of band-pass type transfer functions of its individual components (fluid and solid), as we will see in the following.

Steinhausen (1933) pioneered by developing a mathematical model for the rotation mechanics of the SCCs which describes the overall conduct of the cupula deflection volume as a function of the angular head acceleration, motivated by the characteristics of an overdamped pendulum. Steinhausen’s model is represented by an ordinary differential equation of second order and requires model constants (i.e. for mass, damper and spring coefficients) to be determined separately. Due to its macroscopic nature, the model does not allow for a clear distinction of fluid (EL) and solid (cupula) mechanics. As the ideas developed in the present thesis demand an isolated view of the two domains, emphasis is laid on them in the following Subsections 1.2.1-1.2.2.

Further details on the biomechanics of the SCCs can be found in a review by Rabbitt et al. (2004) as well as in the habilitation treatise of Obrist (2011).

1.2.1 Fluid mechanics of the endolymph

Inspired by the work of Steinhausen (1933) and based on the Navier-Stokes equations, Van Buskirk & Grant (1973) and later Van Buskirk et al. (1976) formulated a partial differential equation for
1.2 Mechanics of the vestibular system

the motion of the endolymph in response to angular movements of the head. It relies on the assumption that the toroidal shape of the slender ducts in the SCC can be uncoiled to an axisymmetric pipe of a constant diameter across which a flow profile develops, maintaining a balance of excitatory (head acceleration), inertial (endolymph mass), viscous (endolymph viscosity) and restoring forces (cupula). The restoring force of the cupula is modeled as being proportional to the integrated flow through the pipe lumen, which we will later refer to as the cupula volume. Such a linear, volume-based cupula model is a typical idealization when fluid mechanics is the focus of investigation, e.g. in Oman et al. (1987) and Boselli et al. (2013).

An exact solution of the equation of Van Buskirk et al. was derived by Obrist (2008) who also numerically investigated the corresponding eigenvalue spectrum. The spectrum reveals a ‘slow’ mode which relates to the cupula mechanics, and an infinite number of ‘fast’ modes originating from the endolymph mechanics. It turns out that the operating range of the SCCs lies between the slow and the fast modes, and the SCC hence ‘applies’ a band-pass filter on head rotations. As the endolymph properties determine the upper limit, head rotations above approximately 10 Hz will undergo viscous blocking in the slender ducts. Likewise, at the lower limit, slow angular motions below about 0.1 Hz are quickly
canceled out by the restoring forces of the cupula.

More recent approaches by Kassemi et al. (2005), Boselli et al. (2009), Wu et al. (2011), Grieser et al. (2012), Boselli et al. (2013) and Grieser et al. (2014) investigated the fluid mechanics of the SCC numerically by solving the Navier-Stokes (or Stokes) equations in three-dimensional SCC morphologies, revealing a detailed picture of the flow field in response to a characteristic head maneuver. This led to the discovery of further flow features of the endolymph motion: Boselli et al. (2009) uncovered a vortical flow pattern in the utricle, in contrast to the uni-directional Poiseuille flow in the slender ducts. Grieser et al. (2012) found that the typical phase lag between head acceleration and fluid response is on the order of five milliseconds in the slender ducts, and about forty milliseconds in the utricle. They also noted that Coriolis as well as centrifugal forces do not influence the endolymph response significantly. Fig. 1.6 shows a visualization of the endolymph flow from numerical simulations of the horizontal canal (HC) during a slow head maneuver.

It is important to note that most studies treat the ML walls as rigid, i.e. fixed to the temporal bone, regardless of an interaction with the fluid motion. This assumption is motivated by the fact that the ML stiffness is several orders of magnitude greater than the cupula stiffness (details are provided in the following section). Although it can be considered a safe assumption for regular SCC morphologies, Rabbitt et al. (2001) as well as the present work demonstrate that under pathological conditions the elasticity of the ML can be a key factor for influencing endolymph flow and must be accounted for.

### 1.2.2 Solid mechanics of the cupula

The solid mechanics of the cupula is not completely understood yet, and contrary opinions exist about its mode of deflection and the time it takes to restore itself to an undeflected state. These two aspects will be subsequently discussed.

Fig. 1.7 shows a model morphology of the gelatinous part of the cupula (Selva et al., 2009). It is attached to the so-called crista by form closure, and along its perimeter at the apex to the ampulla walls by possibly friction or adhesion mechanisms (Hillman, 1974). This is in contrast to the model by Steinhausen (1933) who describes the motion of the cupula as a revolving door that swings tightly along the apical interface.
at the ampulla walls. Later, [Hillman & McLaren (1979)] observed in bullfrogs that the cupula actually can detach at the apex due to excessive endolymph movements. However, he remarks that this may more likely be a relief mechanism to prevent destruction, and the physiological mode of oscillation resembles more that of a drum membrane.

This so-called sealed diaphragm hypothesis was then confirmed by [Rabbitt et al. (2009)]. They observed in oyster toadfishes that the cupula detached itself from the apex only if the transcupular pressure reached excessive values (by un-physiological means of ML indentation), but normally stays ‘attached around its entire perimeter’. Although the literature has been discordant on the modes of deflection, it is commonly stated that there is no endolymph flowing past the moving cupula, endorsing a volume-displacement approach for the mathematical description of cupula deflections.

![Figure 1.7: Model morphology with characteristic dimensions of the human cupula. Adapted from Fig. 5a in Selva et al. (2009) with permission from IOS press and P. Selva.](image)

A second controversy about the cupula characteristics is the time constant $\tau_c$ with which a physiological deformation decays back to the idle state. As a transcupular pressure builds up, the cupula deflects and accumulates endolymph volume which gives rise to counter-acting stresses within the structure. Stiffer structures will exert stronger forces for the same displaced volume and thus result in a quicker, exponential cupula recovery. Hence the cupula time constant correlates inversely with the volumetric cupula stiffness, henceforth denoted by $K_c$.

Various studies have been performed on $K_c$ and $\tau_c$ during the last decades, and their outcome offers quite a range of values: [Jones et al. (1964)] and [Guedry et al. (1971)] measure the time constant in humans to be 16 s (HC) and 7.2 s (PC, SC), while [Dai et al. (1999)] report it to be 4.2 s on average (ten samples) using a model-based technique. The
latter value was used by Obrist (2008) to derive a volumetric stiffness of 13 GPa/m$^3$.

![Superior canal dehiscence schematic](http://www.earsite.com/what-is-superior-canal-dehiscence)

Figure 1.8: Superior canal dehiscence: schematic visualization of the pathoanatomy. Part of the temporal bone along the roof of the superior canal is dehiscent and opens up to the dura mater (shown in blue) which covers the cranial cavity. Retrieved on January 12, 2015, from http://www.earsite.com/what-is-superior-canal-dehiscence

### 1.3 Superior canal dehiscence

The superior canal dehiscence (SCD) syndrome is a pathological condition of the inner ear which remained unidentified until Minor et al. (1998) connected it to vestibular symptoms in response to sound and pressure stimuli. It refers to an abnormal absence or disruption of the temporal bone which separates the inner-ear fluids from the cranial cavity, cf. Fig. 1.8. Predominantly affected due to its protuberant position within the temporal bone is the superior canal (SC), although dehiscences of horizontal (HC) and, even less likely, posterior canals (PC) have been reported as well (Chien et al., 2011; Cremer et al., 2000a; Erdogan et al., 2011; Krombach et al., 2006).

Section 1.3.1 gives a detailed description of the SCD pathophysiology, along with diagnostic and therapeutic measures to identify and potentially cure SCD patients. The so-called third-window theory is introduced in Section 1.3.2 and draws a connection to the fluid and solid mechanics of the semicircular canals (SCC), based on which a separate discussion on the influence of the SCD on the stapes motility follows in Section 1.3.3.
1.3 Superior canal dehiscence

Almost a century ago, Tullio (1929) discovered in 'fenestrated' pigeons (i.e. with artificially created SCD) that they exhibited vestibular disorders such as vertigo and oscillopsia in response to loud sound. Tullio suspected 'orientation sound reflexes' to be the underlying physical mechanism. Later, when Huizinga (1934) confirmed the findings of Tullio by similar experiments, he distanced himself from Tullio's explanation after demonstrating that the orientation of the sound source had no influence on the reported audio-vestibular symptoms. Continuing the experiments on pigeons, Van Eunen et al. (1943) performed a sensitivity study on the sound intensity thresholds for vestibular responses to a bandwidth of sound frequencies ranging from 100 Hz to 3.2 kHz, and found a peak sensitivity between 400 Hz and 1000 Hz at sound pressure levels (SPL) as low as 70 dB. Wit et al. produced similar results in 1985.

It was not until 1998 when Minor et al. pieced it together: they investigated eight humans who presented with vestibular reactions when exposed to either sound or pressure stimuli. On the evidence from CT images, all subjects revealed a dehiscence of bone covering the roof of the superior canal. In a follow-up study by Carey et al. (2000), a large database of 1000 human temporal bone specimens from 596 adults was re-examined. Carey et al. reported a prevalence of SCD in about 2% of the individuals with either dehiscent or markedly thin (≤ 0.1 mm) bone. Fig. 1.9 shows a typical CT image reconstruction (Fig. 1.9A) from a
patient with superior canal dehiscence, along with a photograph (Fig. 1.9B) taken during surgical repair.

Causes

The causes of SCD are highly debated and of multi-variate nature, as the condition may either be inherited or acquired (Hegemann & Carey, 2011). Among the etiologies of SCD are developmental abnormalities which arise during childhood and usually affect both ears, as the temporal bone undergoes postnatal growth (Hirvonen et al., 2003). If a predisposition towards thin-walled SC roofs exists, it may erode over time due to stresses from the ageing dura mater (visualized in blue, Fig. 1.8), or rupture as a consequence of accidental head trauma (Carey et al., 2000). Chien et al. (2011) lists other factors such as congenital defects or chronic otitis media with cholesteatoma, the latter usually in conjunction with horizontal canal dehiscences (Jang & Merchant, 1997).

Symptoms

According to Chien et al. (2011), typical symptoms of SCD patients involve sound-induced vertigo (Tullio phenomenon), pressure-induced vertigo (Hennebert Sign, Valsalva maneuvers), auditory hearing loss and bone conduction hyperacusis, as highlighted in the following. Symptoms involving vertigo are usually accompanied by saccadic eye movements (nystagmus) with a slow, upward-torsional component that corresponds to the affected canal plane (Minor, 2000).

Cases of the Tullio phenomenon (Tullio, 1929) often report trigger events such as the loud and low-frequent sound of a toilet flush, or the noise from a nearby screaming child. The vestibular response usually persists as long as the disturbing sound is emitted, and then tails off within seconds.

The Hennebert sign (Hennebert, 1911) classifies dizziness resulting from the application of pressure to the middle ear. Similar effects along with eye movements are observed when Valsalva maneuvers are performed, i.e. pressure applied against pinched nostrils or a closed glottis, as these increase the pressure in the middle ear or cranial cavity, respectively.

On top of their vestibular dysfunctions, patients further suffer from auditory hearing loss in the dehiscent ear. When these patients are investigated by pure-tone audiometry, one typically finds an increase of
1.3 Superior canal dehiscence

Air conduction thresholds in the affected ear (Kaski et al., 2012; Minor, 2005).

Another side-effect of SCD is the hypersensitivity to bone-conducted sounds (bone conduction hyperacusis), with patients complaining about hearing their own eyes move (Albuquerque & Bronstein, 2004) or other body sounds from their footsteps or voice (Hegemann & Carey, 2011).

Diagnosis

A series of diagnostic measures is required to distinguish SCD from other pathologies in a patient with similar subsets of symptoms, such as Menière’s disease, Benign Paroxysmal Positional Vertigo (BPPV), Otosclerosis of the middle ear, migraine-associated vertigo or labyrinthine fistulae. In order to detect SCD during differential diagnosis, the following three procedures have become the gold standard.

![Characteristic upward-torsional eye motion related to the Tullio phenomenon (TP) in patients with superior canal dehiscence (SCD) during exposure to sound (sound source in blue) in the affected ear (displayed in red).](image)

A quick way to test for SCD is to investigate if the Tullio phenomenon (TP) is present. The eye motion is qualitatively observed during acoustic stimulation by pure tones of elevated sound pressure level (approximately 100dB SPL). Both ears are stimulated sequentially and at different frequencies through the unilateral application of an earphone. If TP occurs during exposure to sound waves in the affected ear, both eyes will react by performing a slow, upward-torsional motion, followed by quick, saccadic reset movements (nystagmus). During torsion, the superior pole of the eyes moves ‘away’ (cf. Fig. 1.10) from the stimulated, affected ear, corresponding to an ampullofugal (excitatory) motion of the superior cupula (Minor, 2000).
More recently, also recordings of vestibular-evoked myogenic potentials (VEMP) have been used to diagnose a canal dehiscence by measuring the activity of ocular or cervical muscles. These responses could be linked to vestibular activities of the utricle and saccule (Welgampola & Carey, 2010), ruling out potential cochlear origins. Details on this novel approach can as well be found in Brantberg et al. (1999).

Indispensable to a comprehensive diagnosis of SCD is the reconstruction of computed tomography (CT) images of the inner ear in the plane of the superior canal (cf. Fig. 1.9A). As the dehiscence usually stretches out to less than five millimeters, ultra-high-resolution CT scans are necessary to clearly identify the roof of the SC.

Treatment strategies

SCD patients currently face two different options for treatment: Both cases usually follow the so-called middle fossa approach during which the superior surface of the temporal bone is surgically accessed. The first option is to plug the SC completely, which results in a definite loss of vestibular function in the vertical plane of the superior canal. A second procedure tries to maintain the SC functionality by resurfacing the impaired bone. Although the latter approach seems more attractive, it is sided with a high relapse rate (Minor, 2005).

1.3.2 Third-window theory

Minor (2000) put forward the hypothesis that a dehiscence ‘creates a third mobile window into the inner ear’, and paved the way for the so-called third-window theory which has its origins in the early work by Huizinga (1934) and has not been refuted to this day. According to the theory, the perilymph moves due to the presence of the third window and leads to a cupula deflection within the respective canal. Carey et al. (2000) further specify the hypothesis by stating that the dehiscence allows ‘dissipation of the pressure as the membranous labyrinth bulges into the adjacent dura and endolymph flows away from the ampulla’. Carey et al. (2004) later add the possibility that ‘endolymph moves along a path or multiple paths between the stapes and the dehiscence’, and they hence suspect a shearing of the vestibular receptor organs along the trajectory, ultimately resulting in nystagmus. However, they also note that ‘the mechanisms by which acoustic stimuli act on the vestibular end
organs are unclear’, and ‘may differ from the damped endolymph motion associated with head acceleration’.

When [Carey et al. (2004)] measured firing rates of vestibular afferents in fenestrated chinchillas, they found that these animals would respond to loud sound with two types of mechanisms: irregular afferents would phase-lock to the rapid stimulus below sound frequencies of 250 Hz, whereas regular afferents generally underwent a slow, tonic increase in firing rate, up to a certain threshold. In one case of low frequency at 125 Hz, a regular HC afferent showed inhibitory behaviour, corresponding to a reversed, ampullopetal motion of the cupula. The regular afferents responded at sound intensities of \(120 \pm 8\) dB, and the irregular afferents significantly lower at \(92 \pm 16\) dB SPL. It is important to note that although the dehiscence was created by Carey et al. on top of the SC, both SC and HC afferents would respond in the described patterns. It may be remarked here that the ampullae of SC and HC are adjacent entities and linked by a short endolymph pathway via the utricle, cf. Fig. 1.4.

Based on their findings, [Carey et al. (2004)] proposed two mechanisms which could potentially explain the third-window hypothesis. First, an excitation-inhibition asymmetry in the oscillatory EL motion (phasic response pattern of the irregular afferents) might lead to a net flow component (tonic response pattern of the regular afferents). Second, an organ pipe analogy with resonance phenomena might explain the flow reversal at low frequencies.

1.3.3 Stapes motility effects on the perilymph

In the context of the SCD pathology, the frequency-dependent stapes motility is of great importance as it determines the amplitude of the perilymph velocities (cf. Chapter 3.1), based on principles of momentum and mass conservation. In the following, we will first review the literature on the frequency-sensitivity of stapes deflection velocities without SCD, and then complement them with a study that accounts for a dehiscence.

**Frequency-sensitivity without SCD**

[Kringlebotn & Gundersen (1985)] experimented with human cadaveric ears in order to assess the frequency characteristics of the middle ear. They found a peak sensitivity of round-window volume displacements to sounds at around 1 kHz, with an exponential decay towards higher
frequencies. Although Kringlebotn & Gundersen stated that ‘the transfer function of the middle ear cannot be measured in living humans’, Huber et al. (2001) succeeded in measuring the stapes velocity in living humans intra-operatively for the first time in humans using Laser-Doppler interferometry. When comparing their results to cadaveric measurements, they could not find a significant difference. This was recently confirmed in similar experiments by Chien et al. (2009).

Figure 1.11 shows the transfer function of the middle ear, as measured during experiments on different species (living and dead), relating the stapes velocity to the incoming sound pressure level as a function of the sound frequency. For convenience, in the present work this transfer function will be interchangeably referred to as ‘stapes motility’.

![Figure 1.11: Comparison of normalized stapes velocity magnitudes $U_s/p_s(f)$ from different species. Data labeled ‘Human cadaveric’ are from Kringlebotn & Gundersen (1985) and were edited by Chien et al. (2009). Data labeled ‘Human live corrected’ and ‘Human live uncorrected’ are from Chien et al. (2009). Stapes velocities are normalized by acoustic pressures $p_s$. Adapted from Fig. 10 in Chien et al. (2009) who modified it from Fig. 8 in Rosowski et al. (1999).](image)

**Frequency-sensitivity with SCD**

Kim et al. (2013) recently published numerical results from a finite element model of the middle and inner ear in the presence of SCD. They focused on the cochlear fluid pressures and the basilar membrane motion, as they were interested in quantifying the hearing loss in SCD patients.
The dehiscence was modeled by a Dirichlet-type pressure boundary condition, being a commonly used assumption \cite{Obrist2011} to represent the reservoir character of the cerebrospinal fluid volume behind the separating dura mater. \cite{Kim2013} also determined the fraction of the stapes-induced perilymph volume displacement which enters the cochlea and deflects the round window membrane (Fig. 1.12). In accordance with the third-window theory, the remaining perilymph displacement flows into the vestibular pathway(s). With these results at hand, one may deduce that the stapes induces perilymph oscillations which increasingly tend to prefer the vestibular pathways for decreasing frequencies. However, with regard to the measurements by \cite{Kringlebotn1985}, the stapes motility exerts the opposite behaviour for frequencies below 1 kHz (Fig. 1.11): the lower the frequency, the lower the stapes amplitudes. The resulting pathological perilymph motion may hence comprise these two counteracting mechanisms.

1.4 Objectives and Outline

The third-window theory of \cite{Minor2000}, along with first theoretical considerations on the Tullio mechanism by \cite{Carey2004}, offers an interdisciplinary interface for research from an engineering perspective. To date, the remaining puzzle about the key mechanisms behind the Tullio phenomenon has not been solved.

Current clinical treatments focus on restoring the roof of the dehis-
cent canal, a surgical intervention with many risks. What if less invasive cures could be devised by means of changing the underlying mechanism to the favour of the patient? When its key parameters are revealed, would it not be tempting to see if slight changes in them could alleviate the patient’s symptoms?

Numerical studies with a suitable computational model offer the unique chance to do so relatively easily, and sensitivity studies may be performed at the expense of computer resources and to the benefit of the (unscathed) patient. Motivated by the prospect of an impact on human healthcare, this work pursues to identify the mechanism behind the superior canal dehiscence syndrome with a combined fluid-dynamical and mechanical approach.

Based on the ideas that were recently developed at the Institute of Fluid Dynamics (IFD) at ETH Zurich [Obrist, 2011], a novel hypothesis is formulated in Chapter 2 which tries to address the fluid-dynamical implications of the Tullio phenomenon. It involves nonlinear mechanisms of fluid-structure interaction in a strongly coupled environment of components with similar densities: endolymph, membranous labyrinth, and perilymph.

As the frequency spectrum of the coupled system spans three orders of magnitude, Chapter 3 aims at devising a thorough modeling concept to render the complex reality with adequate simplifications. The spatio-temporal modeling of the inner-ear physics intrinsically affects the feasibility of a numerical simulation with regard to its convergence behaviour. In Chapter 4 we lay out our approach to discretize the idealized morphology and the model equations both in time and space, using state-of-the-art numerical methods which will be implemented in a tailor-made code.

With all tools at hand, numerical simulations are carried out and documented in Chapter 5 upon which a discussion of the central aspects follows. By means of lumped-parameter models from prior investigations of the author, the numerical results may then be brought into an applicable context of clinical relevance, connecting endolymph physics to the characteristic eye response patterns.

This thesis closes with concluding remarks on its findings in Chapter 3.2.4 and gives an outlook on how to tap its full potential in follow-up studies.

Parts of this work have been documented in Grieser et al. (2012) as
1.4 Objectives and Outline

well as in conference proceedings: Obrist et al. (2012), Grieser et al. (2014b), Grieser et al. (2014a), and Grieser et al. (2014c). Our lumped-parameter modeling in Chapter 3 is based partly on the results in Grieser et al. (2014c).

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19th European Fluid Mechanics Conference (EFMC-9), Rome

285th GAMM Annual Meeting, Erlangen

328th Bárány Society Meeting, Buenos Aires
Chapter 2

Hypothesis

The two sensory organs of the inner ear share the same fluid spaces of perilymph (PL) and endolymph (EL) which are separated by the elastic walls of the membranous labyrinth (ML). It seems surprising that we can sense motion and hear sound at the same time, given that both actions involve motions of these fluids. Obviously there occur two different types of fluid responses which (usually) do not interact.

![Diagram](image)

Figure 2.1: Temporal scale separation between hearing and balance in healthy adults, adapted from Fig. 20.1 in Obrist (2011) and supplemented with patho-physiological phenomena from patients with superior canal dehiscence (SCD): Tullio phenomenon (TP) from sound, Hennebert sign (HS) from pressure stimuli and bone conduction hyperacusis (BCH) to body sounds. Mechanisms of vestibular and cochlear origin are shaded in green and gray, respectively.

From studies of the vestibular system (Chapter 1.2.1) we know that the endolymph is responsible for balance perception, and that it reacts to head maneuvers only within a certain frequency range. If the maneuvers are faster than about 10 Hz, its inertial forces on the endolymph are inferior to the counter-acting viscous forces, and thus the endolymph does not respond. At slow maneuvers of less than 0.1 Hz, the restoring forces of the cupula suppress a significant endolymph motion.
Investigations on the cochlea (Chapter 1.1.1) show that the perilymph is mainly responsible for sound perception and is limited to our human hearing range from 20 Hz to 20 kHz. We can thus identify a clear gap between the operating ranges for balance and hearing. Sensible head maneuvers are located in the inaudible infrasound limit of the cochlea, and audible sound frequencies are inaccessible to the vestibular system. Fig. 2.1 visualizes this scale separation in the white box labeled ‘healthy adult’.

Some people, however, experience a sensory crosstalk between hearing and balance. On the cochlear side, they may hear their own eyes move (bone conduction hyperacusis), while on the vestibular side, their eyes may move when hearing sound (Tullio phenomenon). These paradox phenomena each belong to a different mechanism, and yet they all have one thing in common: a bone dehiscence of the superior canal (SCD).

It seems as if the ‘hole in the bone’ removes a protective layer which ensures that both inner ear organs work independently. Fig. 2.1 supplements the scale separation in healthy adults with patho-physiological phenomena of SCD patients. It appears that case reports on the Tullio phenomenon (TP) are located in a subrange of the audible spectrum, between approximately 300 Hz and 3 kHz.

Here, we will put forward a hypothesis which tries to address the unknown mechanisms behind sound-induced vertigo in presence of a SCD. Our supposition is based on Carey et al. (2004) who suggested a to-and-fro motion of perilymph due to the vibrating stapes. Starting from this assumption, the following sections break down our hypothesized mechanisms in spectral order: we begin with a wave propagation phenomenon on the milliseconds scale (Section 2.1). By fluid-structure interaction, it induces high-frequency flows in the endolymph which translate, by some nonlinear effects, into steady streaming components within less than a second (Section 2.2), and ultimately lead to a balanced cupula displacement and corresponding eye motion within several seconds (Section 2.3). Our hypothesis aligns with ideas which were developed at the Institute of Fluid Dynamics (IFD) at ETH Zurich (Obrist, 2011).

2.1 Wave propagation along the membranous labyrinth

It is an intriguing question why high-frequency oscillations can stimulate a uni-directional flow response in the low-frequency domain of the
balance sense, simply by adding another ‘mobile window’ to the system. As different mechanisms may be at play, we start with an analysis of the involved components.

The vestibular pathway between the stapes and the dehiscence consists of two incompressible fluids (EL, PL) and their separating wall of the ML. Apart from the most direct path across the ampulla of the superior canal, also other perilymph pathways are conceivably actuated by the stapes motion. However, since the flow resistance increases with the path length, we expect that these do not contribute qualitatively to the resulting lymph flow induced by the dehiscence. As the ML is a supple structure, its elasticity results in a finite wavespeed of the excited, coupled system by means of fluid-structure interaction (FSI). Similar systems of FSI have been studied by Moens (1878) and Korteweg (1878), well-known by their findings on the combined wavespeed in elastic pipes, the so-called Moens-Korteweg wavespeed.

We assume that the compliant walls of the ML are subject to transmural pressure differences which originate from the stapes-induced perilymph oscillations. The transmural loads locally deflect the ML and provoke a flow response of the endolymph and perilymph in its vicinity.
As the overall volume of the inner-ear fluids stays constant, these flows will be compensated by an inverse ML deflection in the near surroundings. This process repeats itself, thereby alternatingly creating displacement minima and maxima which meander along the ML towards the bone dehiscence, with a finite propagation speed which is exclusively determined by material and geometrical properties.

At the same time, the propagating deflections are attenuated along their path by the damping nature of the fluid viscosity, such that they are greatest in the vicinity of the stapes and smallest near the dehiscence. By equalizing the perilymph pressure to the pressure within the cranial cavity, the dehiscence can reflect part of the incoming wave train of membrane deflections, possibly creating interference patterns.

Inside the ‘rhythmically massaged hose’ of the ML, the endolymph is forced to flow back and forth at sound periods, pushing and drawing fluid into and from the utricle and thereby invoking corresponding deflections of the cupula. We assume that these oscillations are causing the phase-locking behaviour of irregular cupula afferents which was reported by [Carey et al., 2004]. Fig. 2.2 visualizes our hypothesis on the wave propagation along the ML.

2.2 Steady endolymph streaming

It has been observed in similar studies on the basilar membrane of the cochlea that there are two nonlinear mechanisms at play which are able to create a non-zero static component in an oscillatory flow: acoustic streaming due to Reynolds stresses [Lighthill, 1992] and steady streaming from FSI nonlinearities in the vicinity of vibrating walls [Edom et al., 2014].

We hypothesize that the endolymph flow which results from traveling waves of radially oscillating ML walls shows an analogous behaviour, i.e. that it features both a static and a dynamic component. Furthermore, the strength and direction of the resulting net flow (steady streaming) may depend on the attenuation of traveling waves. These will be weakly attenuated at low sound frequencies, yet strongly at high frequencies.

As the traveling waves get partly reflected at the location of the dehiscence and at the end of the canal, they can lead to interference patterns of nodes and anti-nodes if weakly attenuated. Such a scenario can occur in a frequency range where the wavelength reaches the length scales of the canal. These waves are most likely incapable of generating
steady streaming, as the energy is ‘trapped’ between the interference
nodes. More importantly, Reynolds stresses in the fluid are generally
weaker at these lower frequencies.

In the other extreme, i.e. at high frequencies, strongly attenuated
waves will ‘die out’ before they displace a significant part of the ML.
Between these frequency limits, traveling waves may form. The sound
spectrum in which the Tullio phenomenon occurs might be related to
these limits (cf. Fig. 2.1).

2.3 Cupula response

The cupula follows the pulsating endolymph flow such that it bulges
back and forth at sound periods, yet also continuously builds up a uni-
directional displacement volume due to the static flow components. As
long as the sound source persists to excite the stapes, a constant stream-
ing force will act on the endolymph, thereby feeding a transcupular
pressure difference. With increasing cupula deflection volume, restor-
ing mechanical forces will counteract the motion such that less net flow
is generated.

Eventually, a balance is reached where no additional streaming can
be produced as the restoring forces are as large as the streaming forces.
At that point, the cupula maintains a constant deflection volume which
oscillates with zero mean mass flux. Due to the vestibulo-ocular reflex,
a constant cupula displacement results in a constant slow-phase eye ve-
locity. This process may explain the tonic behaviour observed in regular
cupula afferents (Carey et al., 2004).

As soon as the sound stimuli disappear, the cupula moves back expo-
nentially to its resting state, and likewise the slow-phase eye velocities
in the vertical and torsional planes decay.

Although clinical data on the Tullio phenomenon report eye responses
largely corresponding to an ampullofugal deflection of the cupula, there
exist few cases of ampullopetal deflections. Such a flow reversal of the
net streaming may be explained by a frequency-dependent dominance of
either the acoustic or the FSI-induced streaming mechanism which can
oppose each other. It might also be associated with a shift in the window
location towards the common crus, such that the perilymph pathway to
the dehiscence gets redirected to oscillate primarily about the utricle
instead.
Chapter 3

Physical Modeling

An adequate physical model for a complex and heterogeneous system such as the inner ear requires a clear definition of the effects which the model should be capable of capturing.

According to the hypothesis in Chapter 2, nonlinear phenomena in the endolymph as well as nonlinearities in the fluid-structure coupling may be responsible for the evolution of steady endolymph streaming to cause sound-induced vertigo. As nonlinearities need to be linearized in numerical solution procedures, costly coupling algorithms are required to ensure convergence. The primary focus of our modeling efforts is to retain as much complexity as necessary in the model for the endolymph, and to simplify the remaining elements (perilymph, membranous labyrinth) of the coupled system as much as possible.

Keeping the numerical implications in mind, Section 3.1 seeks to formulate a physical model which is able to reflect the key features of the vestibular pathway between the stapes and the dehiscence. Based on this modeling concept, the governing equations are derived for each subsystem in Section 3.2. Since we plan to perform a sensitivity study on the Tullio phenomenon at a later stage, a dimensional analysis is carried out in Section 3.3 from which we expect to gain insight into the system characteristics. According to the Buckingham Π theorem, e.g. as in Hornung (2006), a reduced set of dimensionless parameters can be derived which may greatly decrease the computational effort of our study.

Parts of this modeling approach were published in the form of conference proceedings in Grieser et al. (2014b).

3.1 Model formulation

In order to prove the existence of the hypothesized mechanical effects of sound on the dehiscent vestibular organ, it may be sufficient to focus exclusively on pure-tone acoustic stimulations for a relevant range of frequencies. If the suspected effect can be demonstrated by simple
harmonics, it will most likely be present in more complex acoustic waveforms, too. Therefore the stapes motion is modeled as a perfect sinusoid, well-defined by its amplitude (velocity $U_s$) and the sound frequency $f$.

As the stapes vibrates, at least two oscillating fluid columns form in the perilymph (density $\rho_f$): one leads towards the round window via the cochlear scalae, and the other(s) towards the dehiscence along the semicircular canals (SCC).

Our physical model will only consider the shortest vestibular pathway whose centerline coordinate is denoted by $x$. It passes first the ampulla ($x = 0$) of the superior canal (SC) and then leads directly to the dehiscence along the narrow duct (Fig. 3.1). The frequency-dependent admittance $\phi$ of the cochlea in presence of an SCD is considered by predictions from Kim et al. (2013), cf. Fig. 1.12. Apart from that, we do not expect further implications on the balance sense which originate from the cochlear pathway.

We assume that the effects of the dehiscence on the stapes motility
can be neglected. Therefore we may obtain the vestibular perilymph flux simply by subtracting the cochlear share $\phi$ from the total, stapes-induced flux. This leads to vestibular perilymph velocity amplitudes $U_p$ of

$$ U_p(f, \text{SPL}) = (1 - \phi(f)) \cdot \frac{A_s}{A_p} \cdot \frac{U_s}{p_s}(f) \cdot p_s(\text{SPL}), \quad (3.1) $$

where we take into account the lever arm between the stapes footplate area $A_s$ and the perilymph lumen $A_p$. $U_s/p_s$ denotes the normalized stapes velocity amplitudes. These can be measured in healthy adults which are subject to harmonic sound of acoustic pressure $p_s$,

$$ p_s = p_{\text{ref}} \cdot 10^{\text{SPL} \over 20\text{dB}}, \quad (3.2) $$

defined by the sound pressure level (SPL) in logarithmic units of decibel (dB), which is related to a reference pressure of $p_{\text{ref}} = 2 \cdot 10^{-5}$ Pa (auditory threshold). An example for the lumped lever arm model (3.1) is given in Fig. 3.2.

![Figure 3.2: Perilymph velocity magnitudes $U_p$ in the dehiscent superior canal (SC), normalized by the acoustic pressure $p_s$. Data plotted according to (3.1), with a stapes footplate area and perilymph lumen of $A_s = 3.21\text{mm}^2$ (Aibara et al., 2001) and $A_p = 0.93\text{mm}^2$ (Curthoys et al., 1977), respectively. The normalized stapes velocity amplitudes $U_s/p_s(f)$ correspond to data labeled ‘Human cadaveric’ in Fig. 1.11. The cochlear fluid fraction $\phi(f)$ corresponds to data labeled ‘zero pressure’ in Fig. 1.12.](image)

As we focus on purely sinusoidal stapes oscillations, we may assume that the perilymph pulsates likewise at the ‘inlet’ of the SC, with angular
frequency $\omega = 2\pi f$ and velocity amplitude $U_p$ from (3.1). The perilymph velocity and acceleration at the inlet boundary then become

$$u_p|_0(t) = U_p \cos(\omega t)$$,  \hspace{1cm} (3.3a)$$
$$\frac{\partial u_p}{\partial t} \bigg|_0(t) = -U_p \omega \sin(\omega t).$$  \hspace{1cm} (3.3b)$$

The excitation of the perilymph by the stapes can then be modeled by a time-dependent Neumann pressure boundary condition, i.e.

$$\frac{\partial p_p}{\partial x} \bigg|_0 = g(t),$$  \hspace{1cm} (3.4)$$

with $g(t)$ being a function of the perilymph velocity (3.3). It will be derived by a momentum analysis in Section 3.2.1.

At the location of the dehiscence ($x = L_1$), the perilymph pressure $p_p$ is equal to the pressure in the cranial cavity across the dura mater, a supple structure of low impedance. We idealize this behaviour by a Dirichlet boundary condition and set the reference pressure level at the dehiscence to zero at all times, i.e.

$$p_p|_{L_1} \equiv 0.$$  \hspace{1cm} (3.5)$$

As the perilymph motion does not stop abruptly at the dehiscence, we also consider the remaining (‘passive’) arm of the SC which goes past the SCD. Like the ‘active’ arm, it features a zero-pressure boundary condition at the dehiscence. At the vestibule ($x = L_2$), we set an outflow boundary condition, i.e.

$$\frac{\partial p_p}{\partial x} \bigg|_{L_2} = 0$$  \hspace{1cm} (3.6)$$

From a modeling point of view, the passive arm of the SC can be understood as a ‘better boundary condition’ on the active pathway. Both partitions are modeled as one-dimensional in $x$.

The canal’s major radius of curvature $R$ is an order of magnitude greater than the minor radius $r_0$, i.e. $R \gg r_0$. Hence we neglect the influence of curvature and straighten the semicircular toroid to a perfect cylinder instead, as displayed in the upper right panel of Fig. 3.1.

The axisymmetric endolymph (density $\rho_f$, kinematic viscosity $\nu_f$) consumes a concentric subspace of the SC. In contrast to the partitioned
perilymph, the endolymph consists of only one computational domain with boundaries at the ampulla at $x=0$ and at the utricle at $x=L_2$. It features an interface to the membranous labyrinth (ML).

Beyond its model boundaries, the endolymph is contained within the larger chambers of the ampulla ($x<0$) and utricle ($x>L_2$) which could be regarded as reservoirs into which it can be drawn or fed from. We reflect this reservoir character by an outflow boundary condition with vanishing velocity gradient in centerline direction ($n_x$) and set the reservoir pressure to zero at all times, i.e.

$$
(n_x \cdot \nabla u_e)_x|_0 = (n_x \cdot \nabla u_e)_x|_{L_2} = 0,
$$

$$
p_e|_0 = p_e|_{L_2} = 0.
$$

We employ a linear-elastic (Young’s modulus $E$, Poisson ratio $\nu_s$), one-dimensional (in $x$) and infinitely thin (virtual thickness $h$, density $\rho_s$) shell model for the ML along its interface between endolymph and perilymph. Since nonlinearities in the structure are neither expected to have a significant effect on the endolymph motion nor computationally justifiable, they do not contribute to our model. The membrane displacements $\eta$ are treated as purely radial deflections from their resting state at radius $r_0$ due to transmural pressure differences $\Delta p_s$. The panel at the center right of Fig. 3.1 displays the described setup and introduces the nomenclature for this radial displacement as

$$
r(x,t) = r_0 + \eta(x,t).
$$

As the total cross-section in the SC does not change in time due to the rigid walls of the temporal bone, a local expansion of the perilymph area $A_p$ will yield a corresponding contraction of the circular endolymph area $A_e = r^2 \pi$. Hence we obtain the following expressions using an index notation for the differentiation with respect to space and time variables,

$$
\frac{\partial A_p}{\partial x} = -\frac{\partial A_e}{\partial x} = -2\pi r \eta_x,
$$

$$
\frac{\partial A_p}{\partial t} = -\frac{\partial A_e}{\partial t} = -2\pi r \eta_t,
$$

$$
\frac{\partial^2 A_p}{\partial t^2} = -2\pi r \eta_{tt} - 2\pi \eta_t^2.
$$

The ratio of the areas at the resting state (index ‘0’) will be referred to
as ‘perilymph dominance’ and is defined as
\[ \beta \equiv \frac{A_{p,0}}{r_0^2 \pi}. \]  
\( (3.11) \)

Fluid-structure interactions take place at the ML interface (index ‘\( \Gamma \)’). We follow a so-called ‘Dirichlet-Neumann’ approach (cf. Chapter 4.3) which sub-iterates for the membrane displacements (Dirichlet condition on the fluid velocity boundary) and the corresponding fluid pressure at the walls (Neumann condition on the structure boundary), i.e.
\[ \mathbf{n}_\Gamma \cdot \mathbf{u}_{e,\Gamma} = \eta_t, \]  
\( (3.12) \)
\[ \Delta p_s = p_e|_{\Gamma} - p_p. \]  
\( (3.13) \)

The cupula model is displayed in the lower left panel of Fig. 3.1 and is based on the commonly used volume approach (displacement volume \( V_c \)) which was explained in Chapter 1.2.2. The model requires a stiffness parameter \( K_c \) to relate volumetric deflections to transcupular pressures. Since the cupula motion due to the elastic restoring forces is three orders of magnitude slower than the high-frequency fluid oscillations, they will exclusively be applied on the mean streaming motion in a separate model (cf. Section 3.2.4).

A schematic overview of our modeling approach is given by the block diagram in Fig. 3.3.

### 3.2 Governing equations

As the physical model is now clearly defined along the boundaries of its subspaces, the governing equations are laid out in the order of their phenomenological appearance: we begin with a derivation of the perilymph equations in Section 3.2.1. In Section 3.2.2, we formulate the equation of motion for the elastic membrane and combine it monolithically with the fluid mechanics of the perilymph. Section 3.2.3 introduces the endolymph equations. Since the modeled cupula response builds up on the temporal mean of the endolymph flow (steady streaming), we establish a connection to the vestibulo-ocular reflex in Section 3.2.4.

#### 3.2.1 Perilymph

The one-dimensionally modeled perilymph varies along its centerline in area \( A_p \), pressure \( p_p \), and velocity \( u_p \). We can postulate the principle of
3.2 Governing equations

Figure 3.3: Block diagram displaying the transfer function circuit to model sound-induced vertigo in patients with SCD. Input/output variables (encircled): sound frequency \( f \), sound pressure level (SPL), cochlear fluid fraction \( \phi \), stapes motility \( U_s/p_s \), perilymph velocity amplitude \( U_p \), membrane Young’s modulus \( E \), steady endolymph streaming \( \bar{U}_e \), cupula displacement volume \( \bar{V}_c \), eye velocity \( \alpha_t \). All other symbols denote system constants corresponding to the (patho-)anatomy of an individual patient. Center box: an analytical fit of \( \bar{U}_e \) becomes available after numerical sensitivity studies in the patho-physiological range of interest \( (E, f, U_p) \).
mass conservation, i.e.
\[
\frac{\partial A_p}{\partial t} + \frac{\partial (A_p u_p)}{\partial x} = 0,
\]
which may be expanded and rewritten to arrive at an expression for the spatial velocity gradient,
\[
\frac{\partial u_p}{\partial x} = -\frac{1}{A_p} \frac{\partial A_p}{\partial t} - \frac{u_p}{A_p} \frac{\partial A_p}{\partial x}.
\]
By differentiating (3.15) with respect to time, we obtain
\[
u_{p,xt} = -\frac{1}{A_p} \frac{\partial^2 A_p}{\partial t^2} + \frac{1}{A_p^2} \left( \frac{\partial A_p}{\partial t} \right)^2 - \frac{\partial}{\partial t} \left( \frac{u_p}{A_p} \frac{\partial A_p}{\partial x} \right).
\]
We can further postulate conservation of momentum in the perilymph, i.e.
\[
\frac{\partial (A_p u_p)}{\partial t} + \epsilon_p (A_p u_p) = -\frac{A_p}{\rho_f} \frac{\partial p_p}{\partial x},
\]
with a pseudo-viscous damping $\epsilon_p$ to account for the radial effects of the viscous term which a one-dimensional model cannot resolve. It can be shown (Appendix A) that in flows of high Womersley numbers, i.e. strongly pulsating flows where transient forces dominate over viscous forces, the pseudo-viscous damping becomes
\[
\epsilon_p \approx \omega = 2\pi f.
\]
We expand and rewrite (3.17) such that we obtain
\[
\frac{\partial u_p}{\partial t} = -\frac{1}{\rho_f} \frac{\partial p_p}{\partial x} - \epsilon_p u_p - \frac{u_p}{A_p} \frac{\partial A_p}{\partial t}.
\]
This expression can further be differentiated with respect to the axial coordinate, i.e.
\[
u_{p,tx} = -\frac{1}{\rho_f} \frac{\partial^2 p_p}{\partial x^2} - \epsilon_p \frac{\partial u_p}{\partial x} - \frac{\partial}{\partial x} \left( \frac{u_p}{A_p} \frac{\partial A_p}{\partial t} \right),
\]
and by substituting the spatial velocity gradient on the right-hand side with (3.15), we get
\[
u_{p,tx} = -\frac{1}{\rho_f} \frac{\partial^2 p_p}{\partial x^2} + \epsilon_p \frac{\partial A_p}{\partial t} + \epsilon_p \left( \frac{u_p}{A_p} \frac{\partial A_p}{\partial x} \right) - \frac{\partial}{\partial x} \left( \frac{u_p}{A_p} \frac{\partial A_p}{\partial t} \right).
\]
When we subtract (3.21) from (3.16), the velocity derivatives drop out and the pressure gradient of second order can be obtained by rearranging the expression as follows,

\[
\frac{1}{\rho \beta} \frac{\partial^2 p}{\partial x^2} = \frac{1}{A_p} \frac{\partial^2 A_p}{\partial t^2} + \frac{\epsilon_p}{A_p} \frac{\partial A_p}{\partial t} - \frac{1}{A_p^2} \left( \frac{\partial A_p}{\partial t} \right)^2 + \left( \frac{\partial}{\partial t} + \epsilon_p \right) \cdot \left( \frac{u_p \partial A_p}{A_p \partial x} \right) - \frac{\partial}{\partial x} \left( \frac{u_p \partial A_p}{A_p \partial t} \right) \tag{3.22}
\]

Appendix B demonstrates that (3.22) can further be simplified using (3.10), (3.15) and (3.19). The result comprises three terms of increasing nonlinearity on the right-hand side, and reads

\[
\frac{\partial^2 p}{\partial x^2} = -2\pi \rho \beta \frac{r}{A_p} (\eta_{tt} + \epsilon_p \eta_t) - 2\pi \rho \frac{1}{A_p} \eta_t^2 + 2\pi \frac{1}{A_p} \frac{\partial p}{\partial x} \eta_x . \tag{3.23}
\]

As it is a priori unclear whether the higher order terms in (3.23) play a significant role in the generation of the hypothesized endolymph streaming, we kept them in our set of equations. However, it may be remarked here that an a posteriori assessment of (3.23) by means of numerical simulations revealed that the last two terms are smaller than the others by at least two orders of magnitude. We found also that the geometrical ratio \(r/A_p\) could safely be approximated by its value at the resting state, \(r_0/A_{p,0}\). Substituting (3.11), a purely linear relationship follows which yields equivalent results as (3.23), i.e.

\[
\frac{\partial^2 p}{\partial x^2} \approx - \frac{2\rho \beta}{\beta r_0} (\eta_{tt} + \epsilon_p \eta_t) . \tag{3.24}
\]

We will continue the description of our modeling with the linearized expression in (3.24). For the sake of completeness, however, the neglected nonlinear terms will reappear in dimensionless form (3.55) at the end of the present chapter. The effect of these terms will be quantified in Chapter 5.2.3.

Since two boundary conditions are specified for each partition of the perilymph in (3.4)-(3.6), the pressure can be calculated by integrating (3.21) twice with respect to the axial coordinate \(x\). The active and passive arms are denoted by ‘I’ and ‘II’, respectively, and one obtains

\[
\text{I: } p_p(x, t) = - \int_{x}^{L_1} \int_{0}^{\tilde{x}} \frac{\partial^2 p_p}{\partial x^2} \, d\tilde{x} \, d\tilde{x} - (L_1 - x) \cdot g(t) , \tag{3.25a}
\]
\[ \Pi: \quad p_p(x, t) = -\int_{L_1}^{L_2} \int_{\bar{x}}^{x} \frac{\partial^2 p_p}{\partial x^2} \, d\bar{x} \, dx. \] (3.25b)

In the following, we derive the \textbf{stapes boundary condition} \( g(t) \) on the perilymph. Using the momentum balance in (3.17), we solve for the spatial gradient of the perilymph pressure, i.e.

\[ \frac{\partial p_p}{\partial x} = -\rho_t \frac{\partial u_p}{\partial t} - \epsilon_p \rho_t u_p - \frac{\rho_t u_p \partial A_p}{A_p} \frac{\partial A_p}{\partial t}. \] (3.26)

By substituting \( A_{p,t} \) with (3.10b), the last term on the right-hand side of (3.26) can be rewritten as

\[ -\frac{\rho_t u_p \partial A_p}{A_p} \frac{\partial A_p}{\partial t} = \frac{2\pi r}{A_p} \eta_t \rho_t u_p. \] (3.27)

The radial membrane velocity \( \eta_t \) has an approximate amplitude of \( |\eta_t| \approx \eta_{\text{max}} \cdot \omega \), with a maximal membrane displacement of \( \eta_{\text{max}} \ll r \). Expanding the fraction in (3.27) with the tube radius \( r \) and substituting the angular frequency \( \omega \) with (3.18), we obtain

\[ \left| \frac{2\pi r}{A_p} \eta_t \rho_t u_p \right| \leq \left( \frac{2A_e}{A_p} \frac{\eta_{\text{max}}}{r} \right) \cdot \omega \rho_t |u_p| \ll \epsilon_p \rho_t |u_p|, \] (3.28)

which shows that the third term on the right-hand side of (3.26) is significantly smaller than the second term and may be safely neglected.

With the perilymph velocity from (3.3a) and its temporal derivative from (3.3b), we arrive at a definition for the Neumann boundary condition \( g(t) \), i.e.

\[ g(t) \approx -\rho_t \frac{\partial u_p}{\partial t} \bigg|_0 - \epsilon_p \rho_t u_p \bigg|_0 = \rho_t U_p (\omega \sin(\omega t) - \epsilon_p \cos(\omega t)). \] (3.29)

### 3.2.2 Membranous labyrinth

\[ \text{Gautier et al. (2007)} \] derived the governing equations for an axisymmetric, elastic membrane which is subject to transmural pressure differences caused by its surrounding fluids. Their equations consider axial and radial membrane motions, and account for possible axial preloads as well.
as variations in the Poisson ratio $\nu_s$. According to Gautier et al., there exist five different types of waves which arise in such a coupled system of fluid-structure interaction (FSI). Four of them ‘mainly propagate within the structure: the torsional shell wave $[...]$, a quasi-longitudinal wave $[...]$, (and) longitudinal/flexural waves’. The only wave which is strongly coupled to the fluid motion is found to be the ‘quasi-plane acoustic wave’ related to the theory of Korteweg (1878).

As we are mainly interested in the fluid response, we consider only these quasi-plane acoustic waves which do not require an axial coupling within the structure. Therefore we may simplify the governing equations from Gautier et al. (2007) by omitting axial motions and axial preloads, and arrive at a linear, one-dimensional model of masses and radial springs,

$$\rho_s h \ddot{\eta} + \frac{E h}{r_0^2 (1 - \nu_s^2)} \eta = \Delta p_s = p_{e,\Gamma} - p_p . \quad (3.30)$$

With the perilymph pressure $p_p$ given by (3.25) and its second derivative $p_{p,tt}$ from (3.24), the membrane model in (3.30) can be partitioned (I, II) and rewritten as

$$I: \rho_s h \ddot{\eta} + \frac{E h}{r_0^2 (1 - \nu_s^2)} \eta = p_{e,\Gamma} + (L_1 - x)g(t) , \quad (3.31a)$$

$$II: \rho_s h \ddot{\eta} + \frac{E h_s}{r_0^2 (1 - \nu_s^2)} \eta = p_{e,\Gamma} . \quad (3.31b)$$

Evidently, in (3.31) the perilymph adds damping characteristics to the membrane motion, as well as it increases the effective mass of the oscillatory system. Introducing mass ($\mathcal{C}_m$), damper ($\mathcal{C}_d$) and spring coefficients ($\mathcal{C}_s$) as the respective bracketed expressions in (3.31), and denoting the right-hand side with $d$, the membrane dynamics may be reformulated as
a system of first-order differential equations, i.e.

\[
\begin{bmatrix}
C_m & C_d \\
0 & 1
\end{bmatrix}
\begin{bmatrix}
\eta_t \\
\eta
\end{bmatrix}
= \begin{bmatrix}
0 & -C_s \\
1 & 0
\end{bmatrix}
\begin{bmatrix}
\eta_t \\
\eta
\end{bmatrix}
+ \begin{bmatrix}
d(x, t) \\
0
\end{bmatrix}.
\] (3.32)

3.2.3 Endolymph

The endolymph motion is governed by the Navier-Stokes equations in an axisymmetric coordinate system. These comprise the conservation of mass and momentum,

\[
\nabla \cdot \mathbf{u}_e = 0,
\] (3.33a)

\[
\frac{\partial \mathbf{u}_e}{\partial t} + \nabla \cdot (\mathbf{u}_e \mathbf{u}_e) = -\frac{1}{\rho_f} \nabla p_e + \nu_f \nabla \cdot (\nabla \mathbf{u}_e).
\] (3.33b)

Bold symbols denote vectorial quantities of axial (\(x\)) and radial (\(r\)) components. The momentum equation (3.33b) will not be simplified, as we want to capture the nonlinear effects which lead to steady endolymph streaming. Like the perilymph, the endolymph is a viscous fluid that has a similar density (\(\rho_f\)) and kinematic viscosity (\(\nu_f\)) as water.

3.2.4 Cupula and vestibulo-ocular reflex

In the present section we are only concerned with the mean flow (steady streaming \(\bar{U}_e\)) of the oscillating endolymph.

Before the onset of a net cupula deflection, at time \(t = t_0\), a transcupular pressure \(\Delta p_c\) builds up as nonlinear mechanisms force the endolymph in a certain direction (cf. Section 2.3). Drag forces counteract the fluid motion, and we may obtain the pressure gradient along the canal by assuming Poiseuille flow. It leads to the so-called Hagen-Poiseuille equation,

\[
\Delta p_c(t_0) = -\int_0^{L_2} \frac{\partial p_e}{\partial x} \left|_{SC} \right. \, d\tilde{x} \approx \frac{8\rho_f \nu_f}{\bar{U}_e} \int_0^{L_2} \frac{d\tilde{x}}{r_0^2} \bar{U}_e = \frac{8\rho_f \nu_f L_2}{r_0^2} \bar{U}_e. \] (3.34)

With increasing net cupula displacement volume \(\bar{V}_c\), the transcupular pressure yields restoring forces until a balance is obtained in which no additional streaming can be generated from the stimulus. At that time \((t = t_\infty)\), the cupula has reached a constant deflection volume,

\[
\bar{V}_c(t_\infty) = \frac{1}{K_c} \Delta p_c(t_0) = \frac{8\rho_f \nu_f L_2}{K_c r_0^2} \bar{U}_e(t_0).
\] (3.35)
Grieser et al. (2014c) studied the endolymph and cupula dynamics in a three-dimensional morphology of the horizontal canal (HC) in response to patient-specific video head impulse tests (vHIT). Numerical simulations of the cupula displacement volume $V_c$ were compared with measured eye velocities $\alpha_t$. Their model featured a cupula stiffness of $K_c = 600 \text{ GPa/m}^3$ which was calibrated such that the simulated exponential decay as $\exp(-t/\tau_c)$ was in accordance with the literature, i.e. $\tau_c \approx 4.4 \text{ s}$.

Revisiting these results in the context of the present work, we identified a proportionality constant $K_\alpha = 122^\circ \text{s}^{-1}/0.003 \text{mm}^3$, relating $\alpha_t$ and $V_c$. Assuming that the superior canal (SC) has similar characteristics, we may incorporate these findings into our model and obtain

$$\alpha_t|_{V/T}(t_\infty) \approx K_\alpha V_c(t_\infty) = \frac{8K_\alpha \rho_f \nu_f L_2}{K_c r_0^2} \bar{U}_e(t_0) \equiv K_s \bar{U}_e(t_0),$$

for vertical (V) and torsional (T) velocity components of the eye motion.

By linear superposition principles, we could hence show that the sound-induced eye velocity is proportional to a steady endolymph streaming at the onset of the sound stimulus. With typical values for the geometrical and material properties, i.e.

$$\rho_f = 10^3 \text{kg/m}^3, \nu_f = 10^{-6} \text{m/s}, L_2 = 9 \text{mm}, \text{ and } r_0 = 160 \mu\text{m},$$

this proportionality constant becomes approximately $K_s \approx 191^\circ/\text{mm}$. A vertical/torsional eye velocity of $19^\circ/\text{s}$ would thus correspond to a steady endolymph streaming of around $0.1 \text{mm/s}$.

### 3.3 Dimensional analysis

The governing equations for the coupled motion of perilymph and membrane in (3.31) as well as for the endolymph in (3.33) were derived in dimensional form. In the following, we carry out a dimensional analysis to determine the reduced set of dimensionless parameters which completely define our system.

Substituting the pseudo-viscous damping $\epsilon_p$ with the angular frequency $\omega$ due to (3.18), we identify eleven dimensional quantities of combinations of three different SI units, listed in Tab. 3.1. According to the Buckingham $\Pi$ theorem (cf. Hornung (2006)), there exist exactly
Table 3.1: Eleven dimensional quantities which completely define the physical model of perilymph (PL), membranous labyrinth (ML) and endolymph (EL).

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
<th>System</th>
<th>SI Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\omega$</td>
<td>Sound frequency</td>
<td>PL</td>
<td>s$^{-1}$</td>
</tr>
<tr>
<td>$U_p$</td>
<td>Velocity amplitude</td>
<td>PL</td>
<td>m s$^{-1}$</td>
</tr>
<tr>
<td>$A_{p,0}$</td>
<td>Cross-sectional area</td>
<td>PL</td>
<td>m$^2$</td>
</tr>
<tr>
<td>$L_1$</td>
<td>Dehiscence location</td>
<td>PL</td>
<td>m</td>
</tr>
<tr>
<td>$L_2$</td>
<td>SCC length</td>
<td>PL/ML/EL</td>
<td>m</td>
</tr>
<tr>
<td>$r_0$</td>
<td>Radius</td>
<td>PL/ML/EL</td>
<td>m</td>
</tr>
<tr>
<td>$h$</td>
<td>Thickness</td>
<td>ML</td>
<td>m</td>
</tr>
<tr>
<td>$E$</td>
<td>Young’s modulus</td>
<td>ML</td>
<td>kg m$^{-1}$ s$^{-2}$</td>
</tr>
<tr>
<td>$\rho_s$</td>
<td>Density</td>
<td>ML</td>
<td>kg m$^{-3}$</td>
</tr>
<tr>
<td>$\rho_f$</td>
<td>Density</td>
<td>PL/EL</td>
<td>kg m$^{-3}$</td>
</tr>
<tr>
<td>$\nu_f$</td>
<td>Viscosity</td>
<td>EL</td>
<td>m$^2$ s$^{-1}$</td>
</tr>
</tbody>
</table>

It may be noted that the ‘perilymph dominance’ $\beta$ was already intro-
duced in (3.11) and has been reproduced by the dimensional analysis. The so-called ‘fluid loading’ \( \mathcal{L} \) was mentioned by Gautier et al. (2007) in their study of elastic tubes undergoing FSI. We further assign a ‘Korteweg number’ \( K_0 \) which may be understood as a Reynolds number whose characteristic velocity is given by the Korteweg wave speed \( c_K \),

\[
c_K = \sqrt{\frac{E h}{\rho_2 r_0}}, \quad \text{Korteweg (1878)}.
\] (3.45)

In the following, we eliminate the dimensional variables from our set of equations. Dimensionless coordinates and flow variables are distinguished from their dimensional counterpart by a prime (‘), as given below.

\[
x' \equiv \frac{1}{r_0} \cdot x
\] (3.46)

\[
t' \equiv \omega \cdot t
\] (3.47)

\[
\eta' \equiv \frac{2}{r_0} \cdot \eta
\] (3.48)

\[
p_e' \equiv \frac{1}{\rho_f (r_0 \omega)^2} \cdot p_e
\] (3.49)

\[
p_p' \equiv \frac{1}{\rho_f (r_0 \omega)^2} \cdot p_p
\] (3.50)

\[
u_e' \equiv \frac{1}{r_0 \omega} \cdot u_e
\] (3.51)

From these definitions, we can also derive the differentiated quantities which appear in the governing equations, i.e.

\[
\nabla' = r_0 \cdot \nabla,
\] \nabla'^2 = r_0^2 \cdot \nabla^2,

\[
\eta'_t = \frac{2}{r_0 \omega} \cdot \eta_t,
\] \neta'_tt = \frac{2}{r_0 \omega^2} \cdot \eta_{tt},

\[
\eta'_x = 2 \cdot \eta_x,
\]

\[
p_{p,x}' = \frac{1}{\rho_f r_0 \omega^2} \cdot p_{p,x},
\]

\[
u_{e,t}' = \frac{1}{r_0 \omega^2} \cdot u_{e,t}.
\]

Based on the dimensionless parameters defined by (3.37)-(3.44) and the
non-dimensionalized variables from (3.46)-(3.51), we can recast our set of equations. The active arm of the coupled perilymph-membrane system is defined between \(0 \leq x' \leq \kappa_1\) and then becomes

\[
I: \frac{1}{L} \eta_{tt}' + \frac{Ko^2}{Wo^4 (1 - \nu_s^2)} \eta' + \frac{1}{\beta} \int_{x'0}^{\kappa_1} \int (\eta_{tt}' + \eta_t') \, d\tilde{x}'d\tilde{x}' = p'_e + \frac{Re}{Wo^2} (\kappa_1 - x') (\sin t' - \cos t') . \tag{3.52}
\]

Likewise, the passive arm is defined between \(\kappa_1 \leq x' \leq \kappa_2\) and can be reformulated as

\[
II: \frac{1}{L} \eta_{tt}' + \frac{Ko^2}{Wo^4 (1 - \nu_s^2)} \eta' + \frac{1}{\beta} \int_{\kappa_1}^{x'\kappa_2} \int (\eta_{tt}' + \eta_t') \, d\tilde{x}'d\tilde{x}' = p'_e . \tag{3.53}
\]

Finally, the dimensionless Navier-Stokes equations may be obtained. They describe the motion of the endolymph between \(0 \leq x' \leq \kappa_2\), i.e.

\[
\nabla' \cdot u'_e = 0 \tag{3.54a}
\]

\[
u'_{e,t} + \nabla' \cdot (u'_e u'_e) - \frac{1}{Wo^2} \nabla'^2 u'_e = -\nabla' p'_e . \tag{3.54b}
\]

For the sake of completeness, we may also derive the non-dimensionalized equation for the active arm which includes all higher order terms, given by

\[
I: \frac{1}{L} \eta_{tt}' + \frac{Ko^2}{Wo^4 (1 - \nu_s^2)} \eta' + \frac{1}{\beta} \int_{x'0}^{\kappa_1} \int (\eta_{tt}' + \eta_t') \, d\tilde{x}'d\tilde{x}' = p'_e + \frac{Re}{Wo^2} (\kappa_1 - x') (\sin t' - \cos t') . \tag{3.55}
\]
The influence of the higher order terms in (3.55) on the steady endolymph streaming will be assessed and quantified in Chapter 5.2.3.
Numerical Modeling

The present chapter lays out the numerical discretization approach which we apply to our physical model. The computational domain is discretized in Section 4.1. We identify two sub-systems: the one-dimensional system of the perilymph, coupled to the membranous labyrinth, and the three-dimensional axisymmetric endolymph domain. These are highlighted separately in Section 4.2 with regard to the spatial and temporal discretization of their governing equations. The densities of the fluids are similar to the density of the membranous labyrinth, such that we deal with a strongly coupled problem (Causin et al., 2005). A coupling algorithm is devised in Section 4.3 to synchronize the solution variables along the common interface. The performance of the fluid-structure coupling is analysed in Chapter 4.4.

Parts of this chapter were documented in conference proceedings (Grieser et al., 2014b). The numerical model was implemented in a tailor-made solver called tullioFoam, written in C++ language. The commented program code is electronically available (Grieser et al., 2015).

4.1 Discretization of the computational domain

Figure 4.1 illustrates the spatial discretization of our model. We split the coupled, one-dimensional system of the perilymph and the membranous labyrinth at the location of the dehiscence into the directly stimulated, ‘active’ arm (I) and the indirectly excited, ‘passive’ arm (II). The domains are discretized along the axial coordinate $x$ into $N_I$ and $N_{II}$ equidistant vertices with mesh widths $\Delta x_I$ and $\Delta x_{II}$, respectively. Each vertex represents a computational node at which we solve for the radial membrane displacements $\eta$ and perilymph pressures $p_p$. The node at $x = L_1$ belongs to both domains. The length of the SCC, $L_2$, corresponds to

$$L_2 = (N_I - 1) \cdot \Delta x_I + (N_{II} - 1) \cdot \Delta x_{II}.$$  (4.1)

In the computational domain of the endolymph, we do not distinguish between the active and the passive arm. The whole canal is discretized
by an array of $N_x \times N_r$ hexahedral volumes, representing an axisymmetric wedge which revolves around the line of symmetry at $r=0$. The vertices at the corner are locked axially to the corresponding vertices of the combined membrane-perilymph system. Endolymph pressures $p_e$ and velocities $u_e$ are stored in the respective centers of the finite volumes. Initially, these are equally spaced in the radial direction with a mesh width $\Delta r$. In the course of the simulation, the corner vertices at the membrane interface $\Gamma$ will follow the radial displacements $\eta$.

We evaluate and store the pressure and velocity boundary conditions in the face centers of the membrane interface, $p_{e, \Gamma}$ and $u_{e, \Gamma}$. As the combined membrane-perilymph domain needs to access the endolymph pressure at the corner vertices, values of $p_{e, \Gamma}$ are interpolated from the face centers by bilinear schemes.

Figure 4.1: Numerical discretization of the computational domains of perilymph (PL), membranous labyrinth (ML) and endolymph (EL). At the dehiscence ($x = L_1$), the coupled, one-dimensional system of ML and PL is split into the ‘active’ (I) and ‘passive’ (II) arms. Each arm is discretized by $N$ equidistant ($\Delta x$) vertices (○) which represent the computational nodes for ML displacements $\eta$ and PL pressures $p_p$. The axisymmetric domain of the EL is discretized by an array of $N_x \times N_r$ finite volumes in the axial and radial direction, respectively. Center points (●) store velocities $u_e$ and pressures $p_e$. Face centers (■) at the ML interface ($\Gamma$) store pressures at the boundary, $p_{e, \Gamma}$. Vertices of the EL (○) move in the radial direction, yet maintain their axial location (coupled to the ML).
4.2 Discretization of the governing equations

In the following sections, the governing equations are discretized in the coupled membrane-perilymph domain and the endolymph.

4.2.1 Coupled system of membrane and perilymph

We restate the system of first-order differential equations,

\[
\begin{bmatrix}
C_m & C_d \\
0 & 1
\end{bmatrix}
\begin{bmatrix}
\eta_t \\
\eta
\end{bmatrix}
= 
\begin{bmatrix}
0 & -C_s \\
1 & 0
\end{bmatrix}
\begin{bmatrix}
\eta_t \\
\eta
\end{bmatrix}
+ 
\begin{bmatrix}
d(x, t) \\
0
\end{bmatrix}.
\]

(3.32)

In our spatial discretization approach, the membrane motion is evaluated at \(N\) computational nodes. We obtain the solution vector,

\[
\varepsilon \equiv 
\begin{bmatrix}
\eta_t \\
\eta
\end{bmatrix}
\in \mathbb{R}^{(2N \times 1)}.
\]

(4.2)

Likewise, the right-hand side vector of (3.32) is discretized as

\[
q \equiv 
\begin{bmatrix}
d \\
0
\end{bmatrix}
\in \mathbb{R}^{(2N \times 1)},
\]

(I):

\[
d = p_e, \Gamma + g(t) (L_1 1 - x),
\]

(4.3b)

(II):

\[
d = p_e, \Gamma.
\]

(4.3c)

Here, \(0\) and \(1\) denote a column vector of \(N\) zeros and ones, respectively. The mass, damper and spring coefficient matrices are given by

\[
C_m = \rho_s h \mathcal{I} + \frac{2\rho_f}{\beta r_0} \mathcal{A} \mathcal{B},
\]

(4.4a)

\[
C_d = \epsilon_p \frac{2\rho_f}{\beta r_0} \mathcal{A} \mathcal{B},
\]

(4.4b)

\[
C_s = \frac{E h}{r_0^2 (1 - \nu_s^2)} \mathcal{I},
\]

(4.4c)

with an identity matrix \(\mathcal{I}\) and two spatial-integral matrices, \(\mathcal{A}\) and \(\mathcal{B}\),

(I):

\[
\mathcal{A} = \int_x^{L_1} (\cdot) d\tilde{x}, \quad \mathcal{B} = \int_0^x (\cdot) d\tilde{x},
\]

(4.5a)

(II):

\[
\mathcal{A} = \int_x^{L_2} (\cdot) d\tilde{x}, \quad \mathcal{B} = \int_x^{L_2} (\cdot) d\tilde{x}.
\]

(4.5b)
The numerical integration in axial direction uses a trapezoidal scheme,

\[ \int_{x_a}^{x_b} (\cdot) \, dx \approx \Delta x \sum_{i=a}^{b-1} [(\cdot)_i + (\cdot)_{i+1}], \quad (4.6) \]

such that the integral operators become triangular matrices,

\[ A = \frac{L_1}{2(N_1-1)} \begin{bmatrix} 1 & 2 & 2 & \ldots & 2 & 2 & 1 \\ 0 & 1 & 2 & \ldots & 2 & 2 & 1 \\ 0 & 0 & 1 & \ddots & 2 & 2 & 1 \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & \ddots & 1 & 2 & 1 \\ 0 & 0 & 0 & \ldots & 0 & 1 & 1 \\ 0 & 0 & 0 & \ldots & 0 & 0 & 0 \end{bmatrix} \in \mathbb{R}^{(N_1 \times N_1)}_+, \quad (4.7a) \]

\[ B = \frac{L_1}{2(N_1-1)} \begin{bmatrix} 0 & 0 & 0 & \ldots & 0 & 0 & 0 \\ 1 & 1 & 0 & \ldots & 0 & 0 & 0 \\ 1 & 2 & 1 & \ddots & 0 & 0 & 0 \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots & \vdots \\ 1 & 2 & 2 & \ddots & 1 & 0 & 0 \\ 1 & 2 & 2 & \ldots & 2 & 1 & 0 \\ 1 & 2 & 2 & \ldots & 2 & 2 & 1 \end{bmatrix} \in \mathbb{R}^{(N_1 \times N_1)}_+, \quad (4.7b) \]

\[ \mathbb{I}: \quad A = \frac{L_2 - L_1}{2(N_2-1)} \begin{bmatrix} 1 & 2 & 2 & \ldots & 2 & 2 & 1 \\ 0 & 1 & 2 & \ldots & 2 & 2 & 1 \\ 0 & 0 & 1 & \ddots & 2 & 2 & 1 \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots & \vdots \\ 0 & 0 & 0 & \ddots & 1 & 2 & 1 \\ 0 & 0 & 0 & \ldots & 0 & 1 & 1 \\ 0 & 0 & 0 & \ldots & 0 & 0 & 0 \end{bmatrix} \in \mathbb{R}^{(N_2 \times N_2)}_+, \quad (4.7c) \]

\[ B = \frac{L_2 - L_1}{2(N_2-1)} \begin{bmatrix} 0 & 0 & 0 & \ldots & 0 & 0 & 0 \\ 1 & 1 & 0 & \ldots & 0 & 0 & 0 \\ 1 & 2 & 1 & \ddots & 0 & 0 & 0 \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots & \vdots \\ 1 & 2 & 2 & \ddots & 1 & 0 & 0 \\ 1 & 2 & 2 & \ldots & 2 & 1 & 0 \\ 1 & 2 & 2 & \ldots & 2 & 2 & 1 \end{bmatrix} \in \mathbb{R}^{(N_2 \times N_2)}_+, \quad (4.7d) \]
We denote the (invertible) coefficient matrices by

\[ \mathcal{M} \equiv \begin{bmatrix} C_m & C_d \\ 0 & T \end{bmatrix} \in \mathbb{R}^{(2N \times 2N)}, \tag{4.8a} \]

\[ \mathcal{N} \equiv \begin{bmatrix} 0 & -C_s \\ T & 0 \end{bmatrix} \in \mathbb{R}^{(2N \times 2N)}, \tag{4.8b} \]

such that we can write the discretized time derivative of the solution vector in the simple form

\[ e_t = \mathcal{M}^{-1} \left( \mathcal{N} e + q \right). \tag{4.9} \]

The temporal discretization is performed at discrete times \( t^{n+1} = t^n + \Delta t \) with a constant timestep \( \Delta t \). We apply the generalized Crank-Nicolson method,

\[ \frac{e^{n+1} - e^n}{\Delta t} = \vartheta e_t^{n+1} + (1-\vartheta)e_t^n, \tag{4.10} \]

with a coefficient \( \vartheta \in [0, 1] \). The scheme is implicit unless \( \vartheta = 0 \) (Euler forward scheme). The convergence tests in Section 4.4 are carried out in the range between \( \vartheta = 0.5 \) (Crank-Nicolson scheme) and \( \vartheta = 1 \) (Euler backward scheme). Applying (4.10) to (4.9) leads to

\[ \frac{e^{n+1} - e^n}{\Delta t} = \vartheta \mathcal{M}^{-1} \left( \mathcal{N} e^{n+1} + q^{n+1} \right) + (1-\vartheta)\mathcal{M}^{-1} \left( \mathcal{N} e^n + q^n \right). \tag{4.11} \]

Defining the (invertible) matrix \( \mathcal{G} \) by

\[ \mathcal{G} \equiv T - \Delta t \vartheta \mathcal{M}^{-1} \mathcal{N}, \tag{4.12} \]

and introducing a coefficient matrix \( \mathcal{K} \) with

\[ \mathcal{K} \equiv \Delta t \mathcal{G}^{-1} \mathcal{M}^{-1}, \tag{4.13} \]

it can be shown that the discretized equation in (4.11) may be rewritten with respect to the solution increment \( \Delta e \),

\[ \Delta e^{n+1} \equiv e^{n+1} - e^n, \tag{4.14} \]
as
\[ \Delta e^{n+1} = K \left( N e^n + \vartheta q^{n+1} + (1-\vartheta)q^n \right). \] (4.15)

Hence we are able to numerically determine the membrane motion at time \( t^{n+1} \) directly from the old state at time \( t^n \), given that we know the right-hand side \( q \) at time \( t^{n+1} \). Since the right-hand side depends on the endolymph pressure at the interface, \( p_{p,r} \), the coupling of the governing equations requires an implicit strategy (cf. Section 4.3).

The coupled membrane-perilymph equation (4.15) is implemented in our solver \texttt{tullioFoam}. All matrix and vector operations as well as corresponding data input/output are carried out using the Armadillo library (Sanderson, 2010). Further details on \texttt{tullioFoam} will follow in the subsequent sections of the present chapter.

### 4.2.2 Endolymph

In Section 4.1 we discretized our computational domain for the endolymph into finite volumes. Without internal re-meshing, a moving boundary such as the membrane interface would lead to significant variations of the cell volume in its vicinity. Such inhomogeneities may give rise to numerical errors and a smooth cell distribution is desirable at all times.

Therefore we apply a computationally inexpensive, algebraic approach to re-mesh the grid: we restrict the vertex positions to radial relocations, \( r_v(x_i) \), and we move them proportionally to the local boundary displacement \( \eta(x_i) \) and their relative tube location \( r_v/r_\Gamma \) by
\[ r_v^{n+1}(x_i) = r_v^n + \frac{r_v^n}{r_\Gamma^n} \left( \eta^{n+1} - \eta^n \right), \quad i = 1, \ldots, N_x + 1. \] (4.16)

An internal re-meshing has an arbitrary character, as it does not reflect physical motions inside the domain. However, any change of the cell volume results in numerical fluxes \( \varphi_g \) across the cell surface \( S \) which must be accounted for when we solve for physical fluxes \( \varphi_e \). The Arbitrary Lagrangian Eulerian (ALE) approach considers these by substituting advective fluxes with relative ones, \( \varphi_{rel} \), defined by
\[ \varphi_{rel} \equiv \varphi_e - \varphi_g = \int_S \mathbf{n} \cdot (\mathbf{u}_e - \mathbf{u}_g) \, dS, \] (4.17)
4.2 Discretization of the governing equations

where \( \mathbf{u}_g \) and \( \mathbf{n} \) denote grid velocities and surface normals, respectively.

Furthermore, the space conservation law (Demirdžić & Perić, 1988) ensures that a change in cell volume \( V \) is equal to the integral of the grid fluxes \( \varphi_g \) across the cell surface \( S \),

\[
\frac{d}{dt} \int_V dV = \int_S \mathbf{n} \cdot \mathbf{u}_g \, dS . \tag{4.18}
\]

Using (4.17)-(4.18), the Navier-Stokes equations (3.33) are integrated over the cell volume \( V \) according to the Finite-Volume Method (FVM). We obtain the well-known discretization in the ALE formulation,

\[
\int_S \mathbf{n} \cdot \mathbf{u}_e \, dS = 0, \tag{4.19a}
\]

\[
\frac{d}{dt} \int_V \mathbf{u}_e \, dV + \int_S \mathbf{n} \cdot (\mathbf{u}_e - \mathbf{u}_g) \, \mathbf{u}_e \, dS =
\]

\[
- \frac{1}{\rho_f} \int_S \mathbf{n} \rho_e \, dS + \nu_t \int_S \mathbf{n} \cdot (\nabla \mathbf{u}_e) \, dS , \tag{4.19b}
\]

where volume integrals over vector or scalar fields \( \circ / \bullet \), (4.20) were substituted by surface integrals according to Gauss’ theorem or the Green-Gauss theorem, respectively, given by

\[
\int_V \nabla \cdot (\circ) \, dV = \int_S \mathbf{n} \cdot (\circ) \, dS , \tag{4.20a}
\]

\[
\int_V \nabla (\bullet) \, dV = \int_S \mathbf{n} (\bullet) \, dS . \tag{4.20b}
\]

Details on the ALE approach can be found e.g. in Donea et al. (2004) and Duarte et al. (2004).

Our code \texttt{tullioFoam} solves (4.19) using the open source library of OpenFOAM in the extended version, 1.6-ext. The fluid solver as well as the treatment of the moving domain boundary \( \Gamma \) has been adapted from a solver\footnote{icoPsiElasticNonLinULSolidFoam, available in OpenFOAM ‘foam-extend-3.1’} by Ž. Tuković and P. Cardiff.
The solver follows the PISO algorithm [Issa et al., 1986], sketched in Fig. 4.2. It consists of a momentum predictor step and a pressure correction loop. The momentum is predicted by solving (4.19b) for the velocity $\mathbf{u}_e$ based on old values for the pressure $p_p$ and the flux $\varphi_e$. When the divergence operator is applied on (4.19b), the transient and viscous terms vanish due to mass conservation. Splitting the operators of the nonlinear term, a Poisson equation can be obtained which is solved iteratively for the pressure.

For PISO, we use a preconditioned bi-conjugate gradient solver (PBiCG) with a diagonal incomplete-LU preconditioner (DILU) for the iterative solution of the endolymph velocity during the momentum predictor step. The pressure correction loop employs a generalized geometric-algebraic multi-grid solver (GAMG) with a Gauss-Seidel smoother, and is repeated twice. In order to compensate for non-orthogonalities inside the mesh, the Poisson equation is successively repeated $N_\perp$ times before the momentum flux is corrected. Each
4.3 Fluid-structure coupling

<table>
<thead>
<tr>
<th>Type</th>
<th>Selected Scheme</th>
<th>Accuracy</th>
<th>OpenFOAM Keyword</th>
</tr>
</thead>
<tbody>
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<td>Time deriv.</td>
<td>Euler backward</td>
<td>2nd order</td>
<td>backward</td>
</tr>
<tr>
<td>Divergence</td>
<td>Central scheme</td>
<td>2nd order</td>
<td>Gauss linear</td>
</tr>
<tr>
<td>Gradient</td>
<td>Central scheme</td>
<td>2nd order</td>
<td>Gauss linear</td>
</tr>
<tr>
<td>Laplacian</td>
<td>Central scheme</td>
<td>2nd order</td>
<td>Gauss linear corrected</td>
</tr>
</tbody>
</table>

Table 4.1: Selected spatial and temporal discretization schemes from the library of OpenFOAM.

distribution of OpenFOAM already ships with our selected solution schemes.

Tab. 4.1 lists the numerical schemes we selected for the discretization of the respective operators in (4.19). A documentation of these schemes as well as of the software library in general can be retrieved online from the [OpenFOAM Foundation (2015)](http://www.openfoam.com). Information on the FVM in moving meshes can be obtained from [Jasak & Tukovic (2004)](http://www.cfd-foam.org). Examples of FSI with OpenFOAM are given in [Jasak & Tukovic (2010)](http://www.cfd-foam.org).

4.3 Fluid-structure coupling

Three media are interacting with each other. Two of them, the perilymph and the membranous labyrinth, were combined and solved monolithically, as described in Section 4.2.1. Their solution variable $\eta$, however, depends on the endolymph pressure at the membrane interface, $p_e$, and vice versa. This interdependence is even enhanced by the condition that the involved media feature similar densities, rendering the need for a strongly iterative procedure to couple both subsystems (Causin et al., 2005).

Therefore, we apply the Dirichlet-Neumann approach (cf. Fig. 4.3) which has been suggested in similar studies on liquid-filled flexible tubes, e.g. by Causin et al. (2005), Küttler & Wall (2008), Nobile (2009), Astorino & Grandmont (2010), Fernández et al. (2010) and Degroote & Vierendeels (2011). Structural displacements ($\eta$) and velocities ($\eta_t$) are imposed on the boundaries of the fluid which returns loads ($p_{e,r}$) on the structure interface.

In order to obtain convergence during a timestep, the displacements are typically relaxed towards the previous solution. Various relaxation schemes exist in the literature, ranging from simple relaxation constants, analysed in Causin et al. (2005), to dynamically adapting parameters
Figure 4.3: Partitioned Dirichlet-Neumann approach to couple the membrane motion $\eta$ iteratively with the endolymph pressure $p_e$ at a given instant in time. $F$ denotes the fluid solver, supplied with Dirichlet boundary conditions from the structure (imposed displacements and velocities). $S$ denotes the solid solver, supplied with a Neumann boundary condition from the fluid (imposed loads).

such as in the IQN-ILS (interface-quasi-Newton with inverse Jacobian from a least-squares model) method by Degroote et al. (2009).

We apply the so-called Aitken relaxation according to a recursive procedure described in Küttler & Wall (2008). During each iteration step $i$, the Aitken parameter $\theta$ is dynamically recalculated from the history of the solution residual $\text{res}_i$. The Aitken method is embedded into our solver framework as described in the following.

**Fluid-structure coupling algorithm**

Figure 4.4 displays a schematic overview of the fluid-structure coupling algorithm as it is implemented in our numerical solver tullioFoam.

The stapes boundary condition $g(t^{n+1})$ is evaluated at the beginning of each time step $t^{n+1}$. For numerical reasons, the amplitude of $g(t)$ is gradually increased from zero at the onset of the simulation ($t^n=t^0$) to its stationary maximum, given in (3.29), over a time span of at least five wave periods. Details on the applied ramp function $R$ can be found in Appendix C.

Before entering the Aitken loop, we provide initial guesses ($i=0$) for the solution variables by means of an extrapolation from previous times, i.e.

$$\Delta \xi_0^{n+1} = \Delta \xi^n,$$  \hspace{1cm} (4.21)
4.3 Fluid-structure coupling

Figure 4.4: Solution algorithm for numerical simulations of the Tullio phenomenon, as implemented in our code tullioFoam. Fluid-structure coupling is performed using the Aitken relaxation.
\[(p_{e, \Gamma})^n + 1 = 2(p_{e, \Gamma})^n - (p_{e, \Gamma})^{n-1}. \] 

(4.22)

Within the Aitken loop, we calculate the right-hand side of the coupled membrane-perilymph equation using (4.3),

\[ q_{i-1}^{n+1} = f \left( (p_{e, \Gamma})_{i-1}^{n+1}, g(t^{n+1}) \right). \] 

(4.23)

A preliminary solution of the membrane motion is denoted by a tilde and may now be obtained by

\[ \Delta e_i^{n+1} = K \left( \mathcal{N} \phi^n + \partial_q q_{i-1}^{n+1} + (1-\vartheta)q^n \right). \] 

(4.24)

With the displacement residual given by

\[ \text{res}_i \equiv \Delta \tilde{\eta}_i - \Delta \eta_{i-1}, \] 

(4.25)

the Aitken relaxation parameter \( \theta \) is calculated by the recursive definition from Eq. 44 in Küttler & Wall (2008), i.e.

\[ \theta_{i-1} = \theta_0 \in [0, 1], \quad i = 1, \] 

(4.26a)

\[ \theta_{i-1} = -\theta_{i-2} \frac{\text{res}_{i-1} \cdot (\text{res}_i - \text{res}_{i-1})}{(\text{res}_i - \text{res}_{i-1}) \cdot (\text{res}_i - \text{res}_{i-1})}, \quad i > 1. \] 

(4.26b)

Additionally, we limit the relaxation to values of

\[ |\theta| \leq 1. \] 

(4.27)

The preliminary solution is now relaxed by

\[ \Delta e_i^{n+1} = \theta_{i-1} \Delta \tilde{e}_i^{n+1} + (1 - \theta_{i-1}) \Delta e_{i-1}^{n+1}, \] 

(4.28)

and the membrane motion \( \varepsilon = [\eta_t, \eta]^T \) can be obtained from

\[ \varepsilon_i^{n+1} = \varepsilon^n + \Delta e_i^{n+1}. \] 

(4.29)

The mesh vertices at the boundary are re-positioned according to the new membrane displacement \( \eta \), and the internal vertices of the endolymph domain are redistributed according to the algebraic definition in (4.16). Additionally, we impose Dirichlet constraints on the velocity boundary.
4.4 Convergence analysis

of the endolymph by assuming no-slip conditions along the membrane interface $\Gamma$, i.e.

$$\mathbf{n}_\Gamma \cdot \mathbf{u}_{e,\Gamma} = \eta_t.$$ \hfill (3.12*)

With the updated mesh at hand, we solve the Navier-Stokes equations for the endolymph, as described by the PISO algorithm in the previous section. At the end of each Aitken cycle, a normalized residual $\varepsilon_A$ is computed according to

$$\varepsilon_A \equiv \frac{1}{r_0} \sqrt{\frac{1}{N} \sum_{v=1}^{N} \text{res}^2(x_v)}, \quad N = N_I + N_{II} - 1.$$ \hfill (4.30)

If the normalized residual falls below a pre-defined tolerance limit,

$$\varepsilon_A < \varepsilon_A^0,$$ \hfill (4.31)

we exit the Aitken loop and advance in time by $\Delta t$.

As we aim for a periodic steady-state solution during the continuous stimulation by the stapes, we introduce a convergence criterion to end the time loop. As long as transient effects of either physical or numerical origin persist, errors relating to the lack of periodicity (‘beat volume errors’) are recorded by comparing the total domain volume to previous periods. Hence we define a normalized beat residual as

$$\varepsilon_V \equiv \frac{\sum_{j=1}^{N} V_j(t^{n+1}) - \sum_{j=1}^{N} V_j(t^{n+1} - T)}{r_0^2 \pi L_2}, \quad N = N_x N_{r},$$ \hfill (4.32)

where $V_j$ is the annular cell volume of a cell with index $j$, obtained by revolving the cell about the line of symmetry. A convergence limit was specified by

$$|\varepsilon_V| < \varepsilon_V^0.$$ \hfill (4.33)

Selected tolerance levels are given in Tab. 4.2.

4.4 Convergence analysis

The convergence of tullioFoam is analyzed based on a default set of parameters for our computational model, given in Tab. 4.3.
<table>
<thead>
<tr>
<th>Symbol</th>
<th>Value</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$r_0$</td>
<td>160 $\mu$m</td>
<td>initial radius of membranous labyrinth</td>
</tr>
<tr>
<td>$L_1$</td>
<td>5 mm</td>
<td>axial location of dehiscence</td>
</tr>
<tr>
<td>$L_2$</td>
<td>9 mm</td>
<td>length of SCC</td>
</tr>
<tr>
<td>$\beta$</td>
<td>10</td>
<td>perilymph dominance (3.41)</td>
</tr>
<tr>
<td>$h$</td>
<td>20 $\mu$m</td>
<td>membrane thickness</td>
</tr>
<tr>
<td>$\rho_s$</td>
<td>1200 kg/m(^3)</td>
<td>membrane density</td>
</tr>
<tr>
<td>$\nu_s$</td>
<td>0.5</td>
<td>membrane Poisson ratio</td>
</tr>
<tr>
<td>$\nu_f$</td>
<td>$10^{-6}$ m(^2)/s</td>
<td>fluid viscosity</td>
</tr>
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<td>$\rho_f$</td>
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<td>fluid density</td>
</tr>
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<tr>
<td>$\theta_0$</td>
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<td>initial Aitken relaxation</td>
</tr>
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<td>time steps per sound period</td>
</tr>
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<td>$N_1$</td>
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</tr>
<tr>
<td>$N_\Pi$</td>
<td>320</td>
<td>no. of vertices in passive arm</td>
</tr>
<tr>
<td>$N_r$</td>
<td>32</td>
<td>no. of cells in radial direction</td>
</tr>
</tbody>
</table>

Table 4.3: Default parameter values of the computational setup.

**Aitken loop**

Figure 4.5 displays the observed convergence behavior of the Aitken residual $\varepsilon_A$. With the onset of a growing residual history at the second iteration step, the Aitken relaxation dynamically re-calibrates itself. Initially the coupling errors increase for two iteration steps. However, after the fourth iteration we observe a continuous decrease which decays by a slope of

$$\frac{\partial \left(\log_{10} \varepsilon_A\right)}{\partial i} \approx -0.13, \quad i > 12.$$  

(4.34)
4.4 Convergence analysis

Approaching the limits of numerical accuracy, the Aitken loop terminates as soon as the residual drops below a value of $\varepsilon_A^0 = 10^{-10}$.

**Time loop**

Advancing in time, we seek to determine the state of periodic steadiness of our results, characterized by vanishing beat volume errors $\varepsilon_V$ in the deforming computational domain of the endolymph. When the stimulation by the stapes is smoothly ramped up to reach full load at $t = 5T$, we observe initial oscillations of the residual which are locked to the stimulus frequency (Fig. 4.6). These errors recede with ongoing simulation time until they practically disappear at $t = 10T$. Furthermore we detect a second pulsation pattern of lower frequency $f_V$, clearly visible at $t > 10T$. It possibly relates to the time it takes to propagate membrane deflections through twice the length of the canal (forth and back). At this point, we may estimate these volume pulsations $f_V$ by the Korteweg wave speed (3.45), and obtain

$$f_V = \frac{c_K}{2L_2},$$  \hspace{1cm} (4.35)
Figure 4.6: Convergence of the beat volume residual $\varepsilon_V$ (4.32) during numerical simulation at default model configuration (Tab. 4.3). Stapes amplitudes are brought to full load (cf. Appendix C) during the first five sound periods $T = 1/f$, visualized by vertical, dotted lines. Oscillating residual values are displayed by magnitudes, with signs (+/-) indicated by different colors (black/grey). The dashed line corresponds to the slope of residual decay. A thick bar depicts the time which Korteweg waves need to reach the end of the canal, $x = L_2$, at speed $c_K$ (3.45). Simulations terminate if $|\varepsilon_V| < \varepsilon^0_V$ for at least three consecutive sound periods.

which is independent of the stapes vibration mode of frequency $f$. Similar to the Aitken loop, we observe that the beat volume residual decays by a slope of

$$\frac{\partial \left( \log_{10} \varepsilon_V \right)}{\partial (t/T)} \approx -0.12.$$  

(4.36)

When the residual reaches the limits of numerical accuracy, we terminate the simulation as soon as it falls below a value of $\varepsilon^0_V = 10^{-8}$ for at least three consecutive sound periods.

Total computation time

Fig. 4.7(a) shows how the choice of the generalized Crank-Nicolson scheme coefficient $\vartheta$ affects the total simulation time to reach a periodic solution. We observe that an increase in $\vartheta$ towards the first-order implicit Euler backward scheme at $\vartheta = 1$ reduces the accuracy of a single Aitken iteration and thus requires more iteration steps to converge, cf. Fig. 4.7(b).
4.4 Convergence analysis

![Graph showing computational effort in numerical simulations with the solver tullioFoam to obtain a periodically steady state. Analysed for different values of the generalized Crank-Nicolson scheme coefficient, \( \vartheta \in [0.5, 1.0] \), visualized by circles. (a) Total computation time. (b) No. of simulated sound periods \( T \) and average no. of Aitken iterations per time step. Dashed lines correspond to polynomial fits through the respective data. Simulations were carried out in nonparallel mode on desktop computers with an Intel® Core™ i5 CPU 650 processor (3.2 GHz, 8 GB memory). Simulation setup corresponds to default parameters listed in Tab. 4.3.

However, as we approach the lower limit corresponding to the second-order accurate Crank-Nicolson scheme at \( \vartheta = 0.5 \), numerical damping lessens. Therefore physical oscillations which arise from the gradual application of loads below \( t \leq 5T \) decay slower over time, resulting in an increase in the overall run time.

These two counteracting mechanisms - numerical accuracy vs. numerical damping - lead to an optimum in computational efficiency which is roughly located at \( \vartheta \approx 0.6 \), as the polynomial fit in Fig. 4.7(a) indicates.

\(^1\) Data acquisition by Benner (2015)
A numerical solution algorithm for fluid-structure interactions inside the inner ear was devised and tested for convergence in Chapter 4. Using the computational infrastructure at the Institute of Fluid Dynamics (IFD) at ETH Zurich, a series of numerical simulations have been carried out with the tailor-made solver \texttt{tullioFoam}. The results of these simulations will be presented in Sections 5.1-5.3 and subsequently discussed in Section 5.4.

The findings are grouped in three parts: we begin with first-order flow phenomena, i.e. the wave propagation of physical quantities between the oscillating stapes and the dehiscence (Section 5.1). This is followed by an evaluation of the second-order mean flow, as we seek to confirm the hypothesized steady endolymph streaming (Section 5.2). Using the principles of dimensional analysis, the underlying mechanisms may be identified and formulated at that point. Section 5.3 puts the results of the mean flow analysis into the context of clinical relevance. We will translate the calculated steady endolymph streaming via cupula mechanics and the VOR into slow-phase eye velocities of a virtual Tullio patient, and compare these velocities to clinical data available in the literature.

Preliminary results were documented in conference proceedings (Grieser et al., 2014\textsuperscript{a,b}). Data acquisition and post-processing was in part\textsuperscript{1,2} accomplished by Benner (2015) in the course of a Master thesis under supervision of the author.

### 5.1 First-order flow: wave propagation

The wave propagation along the canal is successively analyzed by isolating three major effects which determine the speed, the amplitude and the attenuation of the waves. In Section 5.1.1 we investigate how the...
wave speed depends on the membrane stiffness. Section 5.1.2 focuses on the effect of the stapes motility on wave amplitudes. In Section 5.1.3, we vary the sound frequency within the audible spectrum to detect changes in the wave attenuation.

5.1.1 Wave speed

In a first approximation, the wave speed in the coupled system of perilymph, membranous labyrinth and endolymph is solely determined by material constants (membrane stiffness \( E \), fluid density \( \rho_f \)) and cross-sectional dimensions (endolymph radius \( r_0 \), membrane thickness \( h \), perilymph lumen \( A_p \)). Most of these parameters are well documented in the literature such that we can rely on values from anatomical measurements, e.g. by Curthoys & Oman (1987).

However, measurements of Young’s modulus \( E \) are extremely rare, and were performed exclusively \textit{ex vivo} or in animals, e.g. by Yamamoto & Ishii (1991) and Rabbitt et al. (1999). Since the membrane properties may change significantly after death, we cannot resort to statistically firm values for the human membrane stiffness in our modeling. In theory, the whole range between the cupula stiffness of approximately \( 21 \text{ Pa} \) (McHenry & van Netten, 2007) to the stiffness of bone of approximately \( 18 \text{ GPa} \) (Fung, 1981) is possible, spanning across nine orders of magnitude. Practically, we may limit our sensitivity study to a reasonable range between \( E > 1 \text{ kPa} \) and \( E < 100 \text{ kPa} \), which coincides with stiffness values of the adjacent cochlear membranes, as measured by Gueta et al. (2011), for instance.

According to the theory by Korteweg (1878), we expect that the wave speed is proportional to the square root of the membrane stiffness, as

\[
     c_K = \sqrt{\frac{E h}{\rho_f 2r_0}}. \tag{3.45*}
\]

Therefore, as we ‘scan’ through \textit{two} orders of magnitude for the membrane stiffness, we can expect the realistic wave speed to differ from our results at most by \textit{one} order of magnitude. Subsequently, we carried out numerical simulations for different Young’s moduli \( E \) of the membrane. Figs. 5.1-5.2 show the periodic steady-state propagation of radial membrane deflections \( \eta \) and velocities \( \eta_t \) which we obtained for different Young’s moduli at otherwise standard parameters (Tab. 4.3).
We observe that the wavelength $\lambda$ increases as the membrane stiffens, which corresponds to an increase of the wave speed $c$ since
\[ c = \lambda \cdot f. \] (5.1)
Additionally, we note that the oscillation amplitudes are two orders of magnitude smaller than the tube radius, i.e. $\eta/r_0 \ll 1$, and that they decay towards the dehiscence. As the membrane stiffens, the amplitudes decrease as expected.

The interface pressures of the perilymph, $p_p$, and the endolymph, $p_{e, \Gamma}$, are displayed in Figs. 5.3 and 5.4 respectively. We find that the pressure amplitudes increase with Young’s modulus. In contrast to the endolymph, the pressure in the perilymph is dominated by the stapes forcing and only slightly oscillates along the membrane, maintaining an almost constant spatial pressure gradient throughout the active pathway between the stapes and the dehiscence. The endolymph pressure, on the other hand, shows a strongly oscillatory behavior with predominantly traveling waves which decay towards the end of the canal.

Fig. 5.5 completes the picture with plots of the instantaneous endolymph velocities in the axial and radial directions. We identify equally spaced spots of alternating flow reversals which correspond to the wavelength $\lambda$, consistent with the wavelengths of the membrane displacement $\eta$ in Fig. 5.4. According to the no-slip constraint, the radial endolymph velocities at the membrane interface are equal to the membrane velocities from Fig. 5.2. We observe a phase shift of $90^\circ$ with respect to the axial velocity pattern.

The Korteweg theory can be extended to a more general description of the acoustic wave propagation in elastic tubes, using a dispersion equation (Gautier et al., 2007, Eq. 4, p. 335). The restriction to high fluid loadings $\mathcal{L} \gg 1$ was lifted, and variations in the Poisson ratio $\nu_s \neq 0$ were accounted for. Furthermore, Gautier et al. captured interdependencies from longitudinal waves within the structure, $c_L$, and from the speed of sound within the fluid, $c_f \approx 1520 \text{ m/s}$, such that the limitation
\[ r_0^2 \omega^2 \ll c_L^2 \ll c_f^2 \] (5.2)
of the Korteweg theory could be overcome. An exact solution of the dispersion equation for ‘unstretched’ membranes was given (Gautier et al., 2007, Eqs. 8-10, p. 337), which may be rewritten as
\[ c_1 = \left(\frac{2}{a + \sqrt{b}}\right)^{\frac{1}{2}}, \text{ (Acoustic wave, radial oscillation)} \] (5.3a)
Results and Discussion

Figure 5.1: Wave propagation along the membranous labyrinth for different Young’s moduli $E$ at sound frequency $f = 400$ Hz, visualized by consecutive snapshots of radial membrane displacements (gray shades) and the corresponding envelope (red).
5.1 First-order flow: wave propagation

Figure 5.2: Wave propagation along the membranous labyrinth for different Young’s moduli $E$ at sound frequency $f = 400$ Hz, visualized by consecutive snapshots of radial membrane velocities (gray shades) and the corresponding envelope (red).
Figure 5.3: Wave propagation along the membranous labyrinth for different Young’s moduli $E$ at sound frequency $f = 400$ Hz, visualized by consecutive snapshots of the perilymph pressure (gray shades) and the corresponding envelope (red).
5.1 First-order flow: wave propagation

Figure 5.4: Wave propagation along the membranous labyrinth for different Young’s moduli $E$ at sound frequency $f = 400$ Hz, visualized by consecutive snapshots of the endolymp pressure (gray shades) and the corresponding envelope (red).
Results and Discussion

Figure 5.5: Wave propagation within the endolymph for different Young’s moduli $E$ at sound frequency $f = 400$ Hz, visualized by snapshots of axial ($r > 0$) and radial endolymph velocities ($r < 0$).
5.1 First-order flow: wave propagation

\[ c_2 = \sqrt[+]{{\frac{2 c_L^2}{a - \sqrt{b}}}}, \text{ (Longitudinal wave, axial oscillation)} \quad (5.3b) \]

\[ c_L = \sqrt{\frac{E}{\rho_s (1 - \nu_s^2)}}, \quad (5.3c) \]

\[ a = \frac{L - \left(1 - \frac{r_0^2 \omega^2}{c_L^2}\right) \cdot \left(1 - \frac{c_L^2}{c_f^2}\right) - \nu_s^2 \cdot \frac{c_L^2}{c_f^2}}{(1 - \nu_s^2) - \frac{r_0^2 \omega^2}{c_L^2}}, \quad (5.3d) \]

\[ b = a^2 - 4 \frac{L - \left(1 - \frac{r_0^2 \omega^2}{c_L^2}\right) \cdot \frac{c_L^2}{c_f^2}}{(1 - \nu_s^2) - \frac{r_0^2 \omega^2}{c_L^2}}. \quad (5.3e) \]

Fig. 5.6 shows our simulation results for the combined wave speed \( c \) for different Young’s moduli \( E \) and sound frequencies \( f \), and compares them to the analytical predictions by Korteweg and Gautier et al. It may be remarked that our results additionally contain compliance effects due to the presence of the perilymph, which generally leads to reductions in the combined wave speed \( c \).

In the lower audible range, i.e. for \( f < 500 \text{ Hz} \), the simulation results align well with the predictions by Korteweg (1878) within the investigated spectrum of Young’s moduli. As the sound frequency approaches the ring frequency \( f_\pi \) (Gautier et al., 2007), i.e. associated with the time that longitudinal waves need to travel around the circumference of the membrane,

\[ f_\pi = \frac{c_L}{2\pi r_0}, \quad (5.4) \]

such that \( r_0^2 \omega^2 \ll c_L^2 \), our numerical model deviates from the Korteweg theory towards lower wave speeds, recovering the analytical reference by Gautier et al. The latter asymptotically decays to zero speed at \( f = f_\pi \), corresponding to the onset of a ‘stop band’ for acoustic waves (Gautier et al., 2007), up to ultrasound in the inaudible MHz range. Since the Tullio phenomenon has been reported at sound frequencies as high as 3 kHz (Cremer et al., 2000b), we are led to the conclusion that the stop band must begin at higher frequencies. Hence Young’s modulus of the membranous labyrinth must be at least \( E > 10 \text{ kPa} \) (Fig. 5.6(b)), as there would occur no wave propagation otherwise.
5.1.2 Wave amplitude

During stimulation by sound, the stapes vibrates sinusoidally with frequency $f$ at amplitudes $U_s$. As mentioned earlier, studies by Huber et al. (2001) and Chien et al. (2009) found that the amplitudes are not only patient-specific, but also vary greatly across the audible spectrum (cf. Chapter 1.3.3), and depend on the sound pressure level (SPL). It is our intention to separate these anatomical boundary conditions from the mechanism which generates wave propagations along the membranous labyrinth. Apart from the sound frequency, our numerical model thus requires only the specification of a perilymph velocity amplitude $U_p$ at $x = 0$. This amplitude $U_p$ may be obtained separately and prior to the numerical simulation, as sketched in Fig. 3.3.

In the present section, we investigate the effects of $U_p$ on the wave propagation along the canal. For this purpose, we keep the sound frequency and the membrane stiffness constant and vary $U_p$ within a realistic range of values. The results for $\eta$, $\eta_t$, $p_p$, $p_e$, $\Gamma$ and $u_e$ are shown in Figs. 5.7-5.11, respectively.

We find that all first-order flow quantities scale strictly linearly with the perilymph velocity amplitude, i.e.

$$\{\eta(x), \eta_t(x), p_p(x), p_e(x, r), u_e(x, r)\} \propto U_p .$$

(5.5)

5.1.3 Wave attenuation

As the primary waves propagate along the vestibular pathway towards the dehiscence, they undergo attenuation, resulting in lower amplitudes downstream. Since the membranous labyrinth is modeled without damper elements, viscous effects within the fluid are solely responsible for the attenuation mechanism. We approximated the damping characteristics of the one-dimensional perilymph model by a pseudo-viscous damping $\epsilon_p$ which we could relate directly to the stimulus frequency, i.e. $\epsilon_p \approx \omega$ for large Womersley numbers. Within the endolymph, we solve the Navier-Stokes equations (3.33) in two dimensions, therefore no model is required to account for viscous effects. The effective wave attenuation thus results from the presence of both fluids.

Figs. 5.12-5.16 show the main flow quantities of perilymph, membranous labyrinth and endolymph for different stimulus frequencies $f$.
5.1 First-order flow: wave propagation

Figure 5.6: Speed $c$ of propagating waves along the membranous labyrinth for different Young’s moduli $E$ and frequencies $f$. Continuous lines correspond to the exact solution \([5.3a]\) of the dispersion equation by Gautier et al. (2007) for unstretched membranes. They decay asymptotically to zero speed at the respective ring frequency, $f = f_\pi(E) \quad [5.4]$. Dotted lines correspond to the Korteweg wave speed $c_K$ given in \([3.45]\). The theoretical data do not account for the presence of an external fluid like the perilymph (PL). Results of present numerical simulations are indicated by circles. Error bars denote standard deviations from the mean by post-processing $N_t = T/\Delta t$ snapshots of the simulated membrane shape during one sound period $T$ at periodical steady-state conditions. The computational setup is based on the default settings from Tab. 4.3.
Figure 5.7: Wave propagation along the membranous labyrinth for different perilymph velocity amplitudes $U_p$ at Young’s modulus $E = 40$ kPa and sound frequency $f = 800$ Hz, visualized by consecutive snapshots of radial membrane displacements (gray shades) and the corresponding envelope (red).
5.1 First-order flow: wave propagation

Figure 5.8: Wave propagation along the membranous labyrinth for different perilymph velocity amplitudes $U_p$ at Young’s modulus $E = 40$ kPa and sound frequency $f = 800$ Hz, visualized by consecutive snapshots of radial membrane velocities (gray shades) and the corresponding envelope (red).
Figure 5.9: Wave propagation along the membranous labyrinth for different perilymph velocity amplitudes $U_p$ at Young's modulus $E = 40$ kPa and sound frequency $f = 800$ Hz, visualized by consecutive snapshots of the perilymph pressure (gray shades) and the corresponding envelope (red).
5.1 First-order flow: wave propagation

Figure 5.10: Wave propagation along the membranous labyrinth for different perilymph velocity amplitudes $U_p$ at Young’s modulus $E = 40$ kPa and sound frequency $f = 800$ Hz, visualized by consecutive snapshots of the endolymph pressure (gray shades) and the corresponding envelope (red).

(a) $U_p = \frac{1}{8} U_{p,0}$
(b) $U_p = \frac{1}{4} U_{p,0}$
(c) $U_p = \frac{1}{2} U_{p,0}$
(d) $U_p = U_{p,0} = 2.6$ mm/s
Figure 5.11: Wave propagation within the endolymph for different perilymph velocity amplitudes $U_p$ at Young’s modulus $E = 40\, \text{kPa}$ and sound frequency $f = 800\, \text{Hz}$, visualized by snapshots of axial ($r > 0$) and radial endolymph velocities ($r < 0$).
at constant Young’s modulus $E$ and perilymph velocity amplitude $U_p$. We find an exponential increase of the effective damping with respect to the sound frequency (Fig. 5.12). For $f = 4\, \text{kHz}$, the displacements are strongly attenuated such that traveling waves occur only in the first 10% of the vestibular pathway to the location of the dehiscence, $L_1$, before they practically vanish. In contrast, the attenuation at $f = 500\, \text{Hz}$ is by far weaker, as the whole canal pulsates with displacements at the same order of magnitude.

The perilymph pressure, however, barely changes with the sound frequency. The oscillation amplitudes remain relatively constant, as it can be seen in Fig. 5.14. Within the endolymph, the stationary pressure envelope forms patterns of nodes and antinodes. We observe that the amount of nodes increases linearly with the sound frequency.

In order to quantify the strength of the wave attenuation, we define a characteristic length scale associated with the rate of decay. This so-called attenuation length $\sigma$ is shown in Fig. 5.17(a) which assumes that the displacement envelope $\tilde{\eta}$ decays exponentially towards the dehiscence according to

$$\tilde{\eta}(x) = \tilde{\eta}(x_0) \cdot \exp\left(-\frac{x-x_0}{\sigma}\right), \quad x, x_0 \in [0, L_1[. \quad (5.6)$$

Here, $\sigma$ is independent of $x$, and can hence be determined locally by the ratio of the displacement envelope to its spatial derivative,

$$\sigma = \frac{\tilde{\eta}(x)}{\tilde{\eta}_x(x)}. \quad (5.7)$$

The attenuation length $\sigma$ may also be regarded as a measure for the intensity of fluid-structure interactions (FSI). As $\sigma$ increases, a larger part of the canal actively provokes flow responses on both sides of the membrane. Hence we may introduce further dimensionless parameters by relating the attenuation length $\sigma$ to the length of the canal, $L_2$,

$$\Psi_L \equiv \frac{\sigma}{L_2}, \quad (5.8)$$

and to the wavelength $\lambda$,

$$\Psi_\lambda \equiv \frac{\sigma}{\lambda} = \frac{\tilde{\eta}}{\tilde{\eta}_x} \cdot \frac{f}{c}. \quad (5.9)$$

Figs. 5.17(b) and 5.17(c) visualize the simulation results for the attenuation length at various frequencies $f$ and Young’s moduli $E$. We observe
Figure 5.12: Wave propagation along the membranous labyrinth for different sound frequencies $f$ at Young’s modulus $E = 40$ kPa and perilymph velocity amplitude $U_p = 2.6 \text{ mm/s}$, visualized by consecutive snapshots of radial membrane displacements (gray shades) and the corresponding envelope (red).
5.1 First-order flow: wave propagation

![Wave propagation along the membranous labyrinth for different sound frequencies](image)

(a) $f = 500$ Hz

(b) $f = 1$ kHz

(c) $f = 2$ kHz

(d) $f = 4$ kHz

Figure 5.13: Wave propagation along the membranous labyrinth for different sound frequencies $f$ at Young’s modulus $E = 40$ kPa and perilymph velocity amplitude $U_p = 2.6$ mm/s, visualized by consecutive snapshots of radial membrane velocities (gray shades) and the corresponding envelope (red).
Figure 5.14: Wave propagation along the membranous labyrinth for different sound frequencies $f$ at Young’s modulus $E = 40\, \text{kPa}$ and perilymph velocity amplitude $U_p = 2.6\, \text{mm/s}$, visualized by consecutive snapshots of the perilymph pressure (gray shades) and the corresponding envelope (red).
5.1 First-order flow: wave propagation

![Graphs showing wave propagation along the membranous labyrinth for different sound frequencies.](image)

**Figure 5.15:** Wave propagation along the membranous labyrinth for different sound frequencies $f$ at Young’s modulus $E = 40$ kPa and perilymph velocity amplitude $U_p = 2.6$ mm/s, visualized by consecutive snapshots of the endolymph pressure (gray shades) and the corresponding envelope (red).
Figure 5.16: Wave propagation within the endolymph for different sound frequencies $f$ at Young’s modulus $E = 40 \text{kPa}$ and perilymph velocity amplitude $U_p = 2.6 \text{mm/s}$, visualized by snapshots of axial ($r > 0$) and radial endolymph velocities ($r < 0$).
that the effective FSI length $\Psi_L$ scales inversely with the frequency, as it was noted previously in Fig. 5.12. Stiffer membranes lead to higher wave speeds, and hence result in larger FSI lengths.

In all investigated combinations of $f$ and $E$, the attenuation length $\Psi_\lambda$ varied between roughly one and two wavelengths, as can be seen in Fig. 5.17(c). This may indicate that the wave attenuation is strongly coupled to the stimulus frequency.

Keeping all other dimensionless parameters constant, the perilymph dominance $\beta = A_p/A_e$ was varied between $\beta = 5$ and $\beta = 20$, covering the anatomical variance in human SCCs. The results of this study are shown in Fig. 5.18. One can see that the wave attenuation gradually weakens as the perilymph increasingly dominates in size, suggesting that the endolymph generally contributes more strongly to the effective wave attenuation than the perilymph.

This aspect is further investigated in Fig. 5.19. The perilymph damping was set to zero, i.e. $\epsilon_p = 0$, and resulting membrane displacements $\eta$ are compared to the standard case where pseudo-viscous damping applies, i.e. $\epsilon_p = \omega$. As there is no flow resistance in the perilymph for $\epsilon_p = 0$, the passive arm of the SCC exhibits unnaturally strong reflections at the utricle boundary. These oscillations propagate upstream and create interference patterns on the membrane envelope. However, regardless of the unphysical side effects, the overall decay of the wave amplitudes is only slightly affected by the change in perilymph damping.

5.2 Second-order flow: steady streaming in the endolymph

The present section analyses the temporal average of the endolymph motion at the periodic steady-state. First, we observe the local steady streaming $\bar{u}_e(x, r)$ from both the Eulerian and the Lagrangian perspective, and comment on the resulting Stokes drift (Section 5.2.1). The bulk streaming $\bar{U}_e(x)$ denotes the cross-sectional average of the local mean and represents the key parameter which connects the inner-ear dynamics to the eye response of Tullio patients. With regard to $\bar{U}_e$, a sensitivity study is carried out in Section 5.2.2 making use of the dimensionless parameters which we obtained from the dimensional analysis in Chapter 3.3. In Section 5.2.3 we assess different sources of nonlinearity and quantify their effect on the steady streaming in the endolymph.
Results and Discussion

(a) Definition of the attenuation length $\sigma$

$$\text{Displacement } \eta \text{[\mu m]}$$

- displacement envelope $\tilde{\eta}(x)$
- wavelength $\lambda = c/f$
- attenuation length $\sigma = \frac{\tilde{\eta}}{\tilde{\eta}_x}$

$$(a) \text{Definition of the attenuation length } \sigma$$

(b) Attenuation length $\sigma$, normalized by the length of the canal $L_2$

- $E = 80\, \text{kPa}$
- $E = 40\, \text{kPa}$
- $E = 20\, \text{kPa}$
- $E = 10\, \text{kPa}$
- $E = 5\, \text{kPa}$
- $E = 2.5\, \text{kPa}$

slope: $-1$

(c) Attenuation length $\sigma$, normalized by the wavelength $\lambda$

- $E = 80\, \text{kPa}$
- $E = 40\, \text{kPa}$
- $E = 20\, \text{kPa}$
- $E = 10\, \text{kPa}$
- $E = 5\, \text{kPa}$
- $E = 2.5\, \text{kPa}$

slope: $1$

Figure 5.17: Wave attenuation along the membranous labyrinth: dimensionless attenuation lengths $\Psi$ for different sound frequencies $f$ and Young’s moduli $E$, obtained from numerical simulations. Error bars in (b) denote standard deviations from the mean by post-processing $N_t = T/\Delta t$ snapshots of the simulated membrane shape during one sound period $T$ at periodic steady-state conditions. The computational setup is based on the default settings from Tab. 4.3.
5.2 Second-order flow: steady streaming in the endolymph

Figure 5.18: Attenuation length $\Psi_\lambda = \sigma/\lambda$ for different area ratios between perilymph and endolymph, $\beta$, and Young’s moduli $E$ at sound frequency $f=1$ kHz.

Figure 5.19: Wave propagation along the membranous labyrinth (a) with and (b) without flow damping in the perilymph. Simulated at sound frequency $f=1$ kHz, Young’s modulus $E=40$ kPa and perilymph velocity amplitude $U_p=2.6$ mm/s. Visualized by consecutive snapshots of radial membrane displacements (gray shades) and the corresponding envelope (red).
5.2.1 Eulerian and Lagrangian mean flow

Instantaneous values of the endolymph velocity are acquired for a discrete number of \( N_t \) time steps of length \( \Delta t \) within one sound period \( N_t \Delta t = T = 1/f \). In the following, we will apply different methods to calculate the temporal mean of the flow field.

The *Eulerian* mean \( \bar{u}_{e,E} \) is defined at a fixed point in space \((x, r)\) and corresponds to the average over \( N_t \) successive values of the instantaneous endolymph velocity,

\[
\bar{u}_{e,E}(x, r) \equiv \frac{1}{T} \int_{t_0}^{t_0 + T} u_e(x, r, \tilde{t}) d\tilde{t} \approx \frac{\Delta t}{T} \sum_{n=1}^{N_t} u_e(x, r, t^n).
\]  

(5.10)

As the numerical grid moves during simulation, we used the OpenFOAM library to interpolate the flow field at each time step with the so-called cellPointFace scheme applying mixed linear weights, documented in OpenFOAM Foundation (2015).

The *Lagrangian* mean \( \bar{u}_{e,L} \) refers to the mean distance a fluid particle travels during one sound period \( T \),

\[
\bar{u}_{e,L}(x, r) \equiv \frac{x_P(t_0 + T) - x_P(t_0)}{T}, \quad x_P(t_0) = \begin{bmatrix} x \\ r \end{bmatrix},
\]  

(5.11)

following a particle trajectory \( x_P(t) \),

\[
x_P(t) \equiv x_P(t_0) + \int_{t_0}^{t} u_e(x_P(\tilde{t}), \tilde{t}) d\tilde{t}.
\]  

(5.12)

We calculate the trajectory (5.12) with the explicit method of Heun. At time \( t_0 \), passive tracer particles are inserted into the endolymph and distributed homogeneously across the whole domain. Advancing in time, we calculate a preliminary guess \( \tilde{x}_P(t^{n+1}) \) of the new particle positions by

\[
\tilde{x}_P(t^{n+1}) = x_P(t^n) + u_e(x_P(t^n), t^n) \Delta t.
\]  

(5.13)

We interpolate the endolymph velocity at the estimated location \( \tilde{x}_P \) and obtain the new particle position according to Heun’s method by

\[
x_P(t^{n+1}) = x_P(t^n) + \frac{u_e(x_P(t^n), t^n) + u_e(\tilde{x}_P(t^{n+1}), t^n)}{2} \Delta t.
\]  

(5.14)
5.2 Second-order flow: steady streaming in the endolymph

The Stokes drift \( \bar{u}_{e,S} \) is the difference between the Eulerian and the Lagrangian mean,

\[
\bar{u}_{e,S}(x, r) \equiv \bar{u}_{e,E}(x, r) - \bar{u}_{e,L}(x, r).
\] (5.15)

Fig. 5.20 shows results on the mean flow field of the endolymph. We observe that the Eulerian mean streams at largest amplitudes in the vicinity of the ampulla, featuring a positive flow adjacent to the wall and a backflow at the centerline. In this entry region, propagating membrane displacements feature stronger amplitudes, as described in the previous section. Similar to the attenuation of first-order waves along the canal, the steady endolymph streaming relaxes its cross-sectional profile towards a Poiseuille shape within the passive arm, cf. Fig. 5.20(d). The flow resistance to steady streaming may hence be approximated as a steady Poiseuille flow, in accordance with our model assumptions in Chapter 3.2.4.

We note that the Eulerian and Lagrangian means coincide behind the dehiscence, such that the Stokes drift on fluid particles becomes zero in that area.

By principles of mass conservation, the overall volume flux stays constant along the canal. Hence a uni-directional, non-zero steady streaming is identified, revealing an effective transport of fluid in ampullofugal direction, i.e. from the stapes towards the dehiscence.

5.2.2 Bulk flow analysis

The bulk streaming of the endolymph, \( \bar{U}_e \), is defined by the cross-sectional average over the local, axial mean flow \( \bar{u}_e \big|_x \equiv \bar{u}_e \), regardless of the computational perspective (Eulerian or Lagrangian),

\[
\bar{U}_e(x) \equiv \frac{1}{r_0^2 \pi} \int_{A_e} \bar{u}_e(x, r) dA_e,
\] (5.16)

and is constant along the canal due to conservation of mass, i.e.

\[
\frac{\partial}{\partial x} (\bar{U}_e) = 0.
\] (5.17)

Thus, for any tripel of the sound frequency \( f \), Young’s modulus \( E \) and the perilymph velocity amplitude \( U_p \) we obtain from the numerical simulation one scalar value for the bulk endolymph streaming \( \bar{U}_e \). We may de-
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\[ x = 0 \]

(a) Eulerian mean \( \bar{u}_{e,E} \)

\[ r = r_0 \]

(b) Lagrangian mean \( \bar{u}_{e,L} \)

\[ r = r_0 \]

(c) Stokes drift \( \bar{u}_{e,S} = \bar{u}_{e,E} - \bar{u}_{e,L} \)

\[ x = 0 \]

(d) Mean axial velocity profiles

Figure 5.20: Axial mean of the endolymph velocity, \( \bar{u}_e \), viewed from an (a) Eulerian and (b) Lagrangian perspective, along with the resulting (c) Stokes drift. (d) Cross-sectional profiles of the axial mean velocities. Simulated for sound frequency \( f = 1 \) kHz, Young’s modulus \( E = 40 \) kPa and perilymph velocity amplitude \( U_p = 2.6 \) mm/s. Data acquisition and post-processing by Benner (2015).
5.2 Second-order flow: steady streaming in the endolymph

fine a dimensionless variable for the bulk flow by introducing the streaming Reynolds number $\bar{Re}$,

$$\bar{Re} \equiv \frac{\bar{U}_e 2r_0}{\nu_f}.$$  \hfill (5.18)

In the following, we intend to derive an analytical fit for $\bar{Re}$, based on a sensitivity study with respect to variations of the Womersley number $W_o$ (3.40), the Korteweg number $K_o$ (3.37) and the Reynolds number $Re$ (3.39), since

$$W_o^2 \propto f ,$$  \hfill (5.19a)
$$K_o^2 \propto E ,$$  \hfill (5.19b)
$$Re \propto U_p .$$  \hfill (5.19c)

We intend to confine the analytical fit to a physically reasonable parameter space which corresponds to realistic variances in the human inner ear morphology and material properties. Once the fit has been obtained, we are able to circumvent expensive numerical simulations. It can then be chained directly into our lumped parameter approach displayed in Fig. 3.3.

We begin with the effects of the stapes velocity amplitude $U_p$ on the formation of a steady endolymph flow. The results of Section 5.1 revealed that the first-order flow scales linearly with $U_p$. However, in Fig. 5.21 we find that the second-order flow scales quadratically with $U_p$,

$$\bar{Re} \propto Re^2 .$$  \hfill (5.20)

Furthermore, we detect a frequency shift of the bulk streaming pattern when increasing Young’s modulus $E$ of the membrane from $E = 40 \text{kPa}$ to $E = 80 \text{kPa}$. It appears that this shift correlates with the shift in the ring frequency $f_\pi$ (5.4) which marks the onset of a stop band for wave propagation (Gautier et al., 2007). Hence we introduce a dimensionless parameter for the ring frequency, $\Pi$, which can be expressed by a subset of the other dimensionless parameters (3.37)-(3.44),

$$\Pi \equiv \frac{f}{f_\pi} = \frac{W_o^2}{K_o} \sqrt{1 - \frac{\nu_s^2}{L}} .$$  \hfill (5.21)

Taking into account the shape of the bulk streaming pattern from Fig. 5.21(a) we approximate the strength of the steady streaming by

$$\bar{Re} \propto \log_{10} \left( \frac{\Pi}{K_4} \right) \cdot \left( \frac{1 - \Pi}{1 + \Pi} \right)^{K_5} ,$$  \hfill (5.22)
Figure 5.21: Steady endolymph streaming $\bar{U}_e$ from numerical simulations for different sound frequencies $f$ and Young’s moduli $E$, setting the perilymph velocity amplitude $U_p$ to a constant value. $U_{p,0} = U_s^* (f_0, \text{SPL}_0) = 2.6 \text{ mm/s}$, with $f_0 = 1 \text{ kHz}$, $\text{SPL}_0 = 120 \text{ dB}$ and $U_s^*$ from Fig. 1.11 (data labeled ‘Human cadaveric’). The ring frequency $f_\pi$ (5.4) is indicated by a dotted vertical line, marking the onset of a stop band for acoustic wave propagation according to Gautier et al. (2007). Dashed lines in (a) and (b) correspond to horizontal cuts of the contour plot in Fig. 5.23(a). Data acquisition by Benner (2015).
where \( \Pi = K_4 \) corresponds to the low-frequency limit at zero bulk velocity, and the ring frequency at \( \Pi = 1 \) marks the upper limit at wave evanescence. The constant \( K_5 \) defines the sharpness of the upper limit.

In order to test the robustness of the derived proportionalities in (5.20) and (5.22), we apply the frequency-dependent, anatomical spread of stapes velocities \( U_s \) at the perilymph boundary. Setting the perilymph velocity amplitude \( U_p \) equal to the stapes velocity measurements by Kringlebotn & Gundersen (1985) at SPL = 120 dB (Fig. 1.11), we re-run our numerical simulation series for different \( f \) and \( E \). The results are displayed in Fig. 5.22.

Since the anatomical reference features a maximum of the stapes motility at \( f \approx 1 \) kHz, the steady streaming becomes strongest in a similar range. This observation is well in accordance with the quadratic dependence (5.20) of the bulk streaming \( \bar{U}_e \) on the stapes velocity amplitude \( U_p \).

As we divide the simulated bulk velocities by (5.20) and (5.22), we identify a further dependency related to the excitability of the membrane, which we approximate by

\[
\bar{R}e \propto \frac{\Pi K_2}{W_0} \log_{10} \left( \frac{K_3 W_0}{\Pi} \right). \tag{5.23}
\]

The membrane excitability (5.23) will be revisited in Fig. 5.29(b) of Section 5.4. The dimensionless fit for the Reynolds number of the steady endolymph streaming \( \bar{R}e \) thus becomes

\[
\bar{R}e = K_1 R^2 \frac{\Pi K_2}{W_0} \log_{10} \left( \frac{K_3 W_0}{\Pi} \right) \log_{10} \left( \frac{\Pi}{K_4} \right) \left( 1 - \Pi \frac{1}{1 + \Pi} \right)^{K_5}, \tag{5.24}
\]

valid for \( \Pi \in [K_4, 1] \), with constants \( K_i \) from Tab. 5.1. In the stop band for wave propagation, we assume that the bulk streaming vanishes, i.e.

\[
\bar{R}e = 0, \quad \Pi > 1. \tag{5.25}
\]

Fig. 5.23(a) shows a contour plot of the analytical fit in dimensional quantities for a constant perilymph velocity amplitude \( U_p \). Although a constant \( U_p \) does not reflect the reality, the graph enables us to visually isolate the streaming mechanisms from their dependence on the anatomical boundary conditions. We find that the steady streaming is generally strongest for frequencies \( f_\Delta \) at

\[
\Pi_\Delta = f_\Delta / f_\pi \approx \frac{1}{3}, \tag{5.26}
\]
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Young’s modulus $E$ [kPa]

Steady streaming $\bar{U}_e$ [mm/s]

(a)

Figure 5.22: Steady endolymph streaming $\bar{U}_e$ for different sound frequencies $f$ and Young’s moduli $E$, assuming that perilymph velocity amplitudes $U_p$ are a function of the stapes motility $U_s^*$ ($f$, SPL$_0$). $U_s^*$ from Fig. 1.11 (data labeled ‘Human cadaveric’), with SPL$_0$ = 120 dB. Symbols denote simulation results, dashed lines correspond to (a) vertical and (b) horizontal cuts of the contour plot in Fig. 5.23(b).

Table 5.1: Fitting constants in (5.24) for the steady streaming Reynolds number $\bar{Re}(\bar{Re}, W_0, \Pi)$ at perilymph dominance $\beta = 10$, fluid loading $\mathcal{L} = 13.3$, membrane weight $\gamma = 1.2$, location of the dehiscence $\kappa_1 = 31.25$ and canal length $\kappa_2 = 56.25$. Dimensionless parameters are defined in (3.37), (3.44), (5.18), (5.21).

<table>
<thead>
<tr>
<th>$K_1$</th>
<th>$K_2$</th>
<th>$K_3$</th>
<th>$K_4$</th>
<th>$K_5$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.26</td>
<td>0.3</td>
<td>6.76</td>
<td>0.018</td>
<td>0.1</td>
</tr>
</tbody>
</table>
and that it increases monotonically along isolines of $\Pi$ towards softer membranes and lower frequencies. It may be remarked that $\Pi$ scales inversely with the wavelength $\lambda$,

$$\lambda \approx \frac{ck}{f} \propto \frac{\sqrt{E}}{f} \propto \frac{f_o}{f} = \frac{1}{\Pi}, \quad \Pi \ll 1. \quad (5.27)$$

Therefore isolines of $\Pi$ also correspond to isolines of $\lambda$. At $\Pi_\Delta$, the wavelength $\lambda_\Delta$ fits approximately six times into the length of the active arm, $L_1$.

Fig. 5.23(b) already captures the anatomical frequency dependence of the stapes motility $U_s(f)$ according to data by Kringlebotn & Gundersen (1985) from Fig. 1.11. We note that the anatomical constraints restrict the streaming corridor from the previous figure to a spot around $f = 1$ kHz.

The most realistic scenario is displayed in Fig. 5.23(c). It accounts for both the anatomy and the SCD pathology, since it incorporates the lumped lever arm model (3.1) described in Chapter 3.1. According to the model, part of the stapes-induced perilymph motion diverts into the cochlear scalae, alleviating the bulk streaming from Fig. 5.23(b) at higher frequencies (cf. Fig. 1.12). Additionally, the lever arm between the stapes footplate and the smaller SCC lumen increases the perilymph velocity amplitudes $U_p$ linearly, and hence the bulk streaming $U_e$ quadratically.

The influence of the dehiscence location $L_1$ on the steady endolymph streaming $U_e$ was assessed by numerical simulations. Results are shown in Fig. 5.24(a). We find that a shift of the dehiscence further away from the ampulla results in an increase of streaming amplitudes. This may correlate with an increase in the active membrane surface. However, it may be remarked that longer perilymph pathways $L_1$ increase the flow resistance in the vestibular system, possibly lowering the velocity amplitudes $U_p$ which are diverted towards the dehiscence. Such an effect was not accounted for, and may result in a less pronounced increase in Fig. 5.24(a).

Fig. 5.24(b) shows a similar study varying the total length of the slender duct, $L_2$. As the flow resistance scales with $L_2$, the steady streaming behaves inversely proportional to the canal length.

### 5.2.3 Sources of nonlinearity

A system which features only linear mechanisms is unable to exhibit second-order phenomena. Although the involved fluids of the vestibular system behave practically linearly in everyday head maneuvers, the spe-
Figure 5.23: Contour plots of steady endolymph streaming $\bar{U}_e$ as function of sound frequency $f$, Young’s modulus $E$ and perilymph velocity amplitude $U_p$. Circles indicate results from numerical simulations, from which the analytical fit (5.24) has been derived. $\bar{U}_e$ reverses its direction in region $\circled{1}$. The dashed line (red) corresponds to the ring frequency $f_\pi$ (5.4), marking the onset of stop band region $\circled{2}$ for acoustic plane waves, according to Gautier et al. (2007). Thick isolines correspond to values printed in the color bar. $f_0 = 1\, \text{kHz}$, $\text{SPL}_0 = 120\, \text{dB}$, $U_p^*$ from (3.1), $U_s^*$ from Fig. 1.11, $\phi^*$ from Fig. 1.12. Further physical and numerical parameters are given in Tab. 4.3.
5.2 Second-order flow: steady streaming in the endolymph

Figure 5.24: Steady endolymph streaming $\bar{U}_e$ for variations in dehiscence location, $L_1$, and canal length, $L_2$, at Young’s modulus $E = 40$ kPa and perilymph velocity amplitude $U_p = 2.6$ mm/s. Stars (⋆) denote standard values according to Tab. 4.3. Data acquisition and post-processing by Benner (2015).

Special condition of SCD introduces high-frequency oscillations to the fluid dynamics when exposed to sound. These give rise to nonlinearities in the perilymph as well as in the endolymph, since both are coupled strongly across the membranous labyrinth.

Higher-order terms in the perilymph

First, we will analyze the effect of the higher-order terms (HOT) in the perilymph. These were neglected in (3.24). However, our numerical solver tullioFoam incorporates all nonlinear terms within the Aitken loop, such that we can directly obtain the error from the difference in numerical simulations by switching the terms ‘on’ or ‘off’, respectively. Fig. 5.25 visualizes the errors of first- and second-order flow quantities, i.e. of the maximum of the membrane displacement, $\eta_{\text{max}}$, the wave speed $c$ and the bulk streaming $\bar{U}_e$. We find that all these errors are well below
Results and Discussion

unity. Most importantly, the second-order flow in the endolymph is affected by less than three orders of magnitude. Hence we conclude that higher-order terms can safely be neglected, as they do not contribute significantly to the Tullio phenomenon.

$$\begin{align*}
10_0 & \quad 10^{-6} \\
10^{-5} & \quad 10^{-4} \\
10^{-3} & \quad 10^{-2} \\
10^{-1} & \quad 10_0
\end{align*}$$

Figure 5.25: Relative errors $\varepsilon$ due to neglects of higher order terms (HOT) in the perilymph, corresponding to numerical simulations (w/ and w/o HOT) at sound frequency $f = 1 \text{ kHz}$. Displayed are errors of first-order (max. membrane displacement $\eta_{\text{max}}$, wave speed $c$) and second-order flow quantities (steady endolymph streaming $\bar{U}_e$). Absolute errors are normalized by the magnitude of the respective flow quantity.

Nonlinearities in the endolymph

Within the endolymph, further mechanisms are able to create second-order flow components. First of all, as the Navier-Stokes equations comprise the nonlinear advection term, Reynolds stresses may drive the mean flow. We carried out numerical simulations to assess the influence of these stresses on the bulk streaming, comparing cases when advection was switched ‘on’ or ‘off’.

Fig. 5.26 visualizes the results for different Young’s moduli $E$ and sound frequencies $f$. Although all nonlinear terms in the governing equations are suppressed, we still obtain a non-zero mean flow. Obviously there is a second streaming mechanism at play which can only correspond to nonlinearities in the FSI coupling at the interface. A similar phenomenon has been observed for steady streaming in the cochlea (Edom et al., 2014). We denote this type of flow by ‘wall-induced’ streaming $\bar{U}_e|_w$. The difference in streaming, $\bar{U}_e|_\text{Re}$, thus relates to the Reynolds forcing,

$$\bar{U}_e|_\text{Re} \equiv \bar{U}_e - \bar{U}_e|_w.$$  \hspace{1cm} (5.28)
5.3 Eye response to sound stimuli

A patient presenting with the Tullio phenomenon may sense dizziness and experience vertigo when exposed to loud sound stimuli. As these are rather subjective symptoms which can hardly be quantified, one looks for secondary effects such as the eye response to vestibular stimulation. The cupula mechanics in combination with the vestibulo-ocular reflex...
(VOR) translates the induced endolymph flow into a motion of the eye, with a slow-phase velocity component $\alpha_t$. In Chapter 3.2.4 we demonstrated that these velocities scale directly proportionally to the steady endolymph streaming $\bar{U}_e$ by

$$\alpha_t \approx K_s \bar{U}_e,$$

(5.29)

with a proportionality constant $K_s \approx 191^\circ/mm$. In the course of a sensitivity study, we obtained an analytic expression (5.24) for the steady endolymph streaming $\bar{U}_e$ from numerical simulations, i.e.

$$\bar{U}_e = \frac{\nu \rho}{2r_0} \Re \left( \Re \propto U_p, \omega_0 \propto \sqrt{f}, K_0 \propto \sqrt{E} \right).$$

(5.30)

The perilymph velocity amplitude $U_p$ relates to the (patho-)anatomy of the specific Tullio patient. This may be estimated by our lumped lever arm model (3.1), being a function of the cochlear admittance $\phi$, the anatomical ratio of the stapes footplate area $A_s$ to the perilymph lumen $A_p$, the stapes motility $U_s/p_s$ and the sound pressure level SPL,

$$U_p(f, \text{SPL}, D) = \frac{A_s}{A_p} \cdot (1 - \phi(f, D)) \cdot \frac{U_s}{p_s}(f) \cdot p_{\text{ref}}^{10\frac{\text{SPL}}{20\text{dB}}},$$

(5.31)

where $D$ denotes the radius of the dehiscence $r_d$ relative to the canal radius $r_{SC}$, as in Kim et al. (2013). Once the patho-anatomical reference data are obtained, e.g. by

$$\phi = \phi(f, D) \quad \text{from Kim et al. (2013),}$$

(5.32)

$$\frac{U_s}{p_s} = \frac{U_s}{p_s}(f) \quad \text{from Kringlebotn & Gundersen (1985),}$$

(5.33)

the slow-phase eye velocities $\alpha_t$ can be directly calculated for any harmonic sound stimulus using (5.29)-(5.31).

Fig. 5.27 shows our model predictions on the slow-phase eye velocities $\alpha_t$ for a sound pressure level of $\text{SPL} = 110\,\text{dB}$, and compares them to reference data from different Tullio patients in the literature.

In order to account for anatomical variations among subjects, we additionally shifted the stapes characteristics $U_s(f)$ of the anatomical reference (2,5) by Kringlebotn & Gundersen (1985) to lower (1,4) and higher (3) frequencies. The effect of the size of the dehiscence $D$ was assessed by using predictions on the cochlear admittance $\phi(f, D)$ from
5.3 Eye response to sound stimuli

(a) Possible scenarios of (patho-)anatomical conditions in patients with SCD

(b) Corresponding predictions of the slow-phase eye velocities $\alpha_t$

Figure 5.27: Comparison of predicted slow-phase eye velocities $\alpha_t$ from five virtual patients (1-5) with measurement data from nine real patients at a sound pressure level of SPL = 110 dB. Physioanatomical conditions correspond to stapes motility measurements $U_s^*$ by Kringlebotn & Gundersen (1985), displayed in Fig. 1.11 labeled ‘Human cadaveric’. SCD pathoanatomy relates to numerical predictions of the cochlear admittance $\phi^*$ by Kim et al. (2013), displayed in Fig. 1.12 for different sizes of the dehiscence, $D \equiv r_d/T_{SC}$, where $D_1 = 1$, $D_{0.1} = 0.1$. Perilymph velocity amplitudes $U_p$ obtained from (3.1) with stapes footplate area $A_s = 3.21 \text{mm}^2$ (Aibara et al., 2001) and perilymph lumen $A_p = \beta r_0^2 \pi$. Further parameters are listed in Tab. 4.3.
Kim et al. (2013) for $D = 1$ (①,②,③) and $D = 0.1$ (④,⑤). The corresponding graphs are shown in Fig. 5.27(a).

The eye velocity predictions from our fluid-dynamical model correspond well with the typical range of patient responses from the literature, e.g. by Cremer et al. (2000b), Minor (2000), Minor et al. (2001), Minor (2005) and Kaski et al. (2012). Within the tested anatomical variance among the virtual patients ①-⑤, the symptoms of already five out of eight real patients can be explained by our model.

Generally we note that the peak sensitivity for the Tullio phenomenon coincides with the peaks in stapes motility $U_s(f)$. An anatomical variance in the membrane stiffness $E$ plays a role predominantly at sound frequencies around these sensitivity peaks and below, with stiffer membranes increasingly alleviating pathological symptoms.

We further observe that smaller dehiscence radii $r_d$ shift the peak response slightly towards lower frequencies and reduce the intensity of the Tullio phenomenon by roughly

$$\frac{\partial \log_{10}(\alpha_t/[\circ/s])}{\partial \log_{10}(r_d/r_{SC})} \approx 0.64, \quad r_d \leq r_{SC}. \quad (5.34)$$

The effect of the dehiscence location $L_1$ was shown in Fig. 5.24(a) for frequencies of $f = 0.4$ kHz and $f = 1$ kHz. Although we remarked that the true increase of $\bar{U}_e (\propto \alpha_t)$ with $L_1$ is probably slightly weaker than displayed, the effect of $L_1$ on the Tullio phenomenon is on a similar order as the effects from the dehiscence size, i.e.

$$\frac{\partial \log_{10}(\alpha_t/[\circ/s])}{\partial \log_{10}(L_1/r_0)} \lesssim 0.66, \quad L_1 \gg r_0. \quad (5.35)$$

The values for the slopes in (5.34)-(5.35) correspond to respective averages over the available data, and vary slightly with $E$ and $f$.

Fig. 5.28 shows additionally the effect of the sound pressure level on the Tullio phenomenon. If we assume that the subjective feeling of vertigo and dizziness grows equally in strength as the corresponding eye motions, we may suppose that there exists a measurable threshold $\alpha_{t,\text{min}}$ below which a Tullio patient does not experience vestibular responses to sound. With regard to physiological operating ranges of the vestibular system, rotational head motions with velocities on the order of $0.1\circ/s$ are located at the low-frequency end of the balance sense. Thus we may regard the corresponding isocontour of the eye response as a conceivable
5.3 Eye response to sound stimuli

Figure 5.28: Activity map of the Tullio phenomenon for the virtual patient of the previous figure. Contour plots of the predicted slow-phase eye velocity $\alpha_t$ correspond to our model (5.29)-(5.33), and are shown as a function of the sound frequency $f$ and the sound pressure level SPL. The dashed line (red) corresponds to the ring frequency $f_{\pi}$ (5.4), marking the onset of a stop band region for acoustic plane waves according to Gautier et al. (2007). Physical parameters are given in Tab. 4.3.
threshold for the Tullio phenomenon, i.e. at
\[ \alpha_{t,\text{min}} \approx 0.1^\circ/s. \] (5.36)

Showing only isocontours of \( \alpha_t \geq 0.1^\circ/s \), Fig. 5.28 basically reveals the activity map of the Tullio phenomenon. In the white space, the associated vestibular responses are too weak to produce perceivable symptoms. In the active region, a ‘sweet spot’ for vestibular reactions can be determined for each sound pressure level. We qualitatively observe that this spot narrows for stiffer membranous labyrinths. This coincides with the frequency shift of \( f_\pi \) which moves the peak of steady streaming sensitivity \( (\Pi_\triangle \approx 1/3 \text{ (5.26), Fig. 5.23(a))} \) away from the peak of the stapes sensitivity towards higher frequencies.

Tullio patients may as well feature patho-anatomical conditions slightly different from the ones which were suggested by Kim et al. (2013). If the cochlear admittance \( \phi \) was less pronounced at higher frequencies, the perilymph would oscillate by stronger amplitudes \( U_p \). As the steady streaming scales quadratically with \( U_p \), this could explain the three case reports from Fig. 5.27(b) which exhibited symptoms at \( f = 2 - 3 \text{ kHz} \).

However, it may also be possible that a further mechanism exists at values of approximately \( 0.5 \leq \Pi < 1 \) where our linear membrane model is not able to resolve the increasing influence from longitudinal motions on the plane-wave propagation.

### 5.4 Discussion of the results

The results of our numerical simulations enabled us to establish an analytical formulation (5.24) for the Tullio phenomenon in the parameter range of interest,

\[
\begin{align*}
f & \in [100 \text{ Hz}, 10 \text{ kHz}] \quad \Rightarrow \quad W_0 \in [4, 40] \text{ , } & (5.37a) \\
E & \in [1 \text{ kPa}, 100 \text{ kPa}] \quad \Rightarrow \quad K_0 \in [40, 400] \text{ , } & (5.37b) \\
U_p & \in [0 \text{ mm/s}, 10 \text{ mm/s}] \quad \Rightarrow \quad Re \in [0, 1.6] \text{ . } & (5.37c)
\end{align*}
\]

As we identified several mechanisms affecting the vestibular response to sound, we will highlight each of them separately in the following.

**Nonlinear mechanisms**

The governing equations of our computational model comprise two nonlinear mechanisms: Reynolds stresses arising from the advection term
5.4 Discussion of the results

Figure 5.29: Sketch of the mechanisms which are responsible for vestibular reactions to pure-tone sound stimuli. The signs (+/-) indicate the direction of the corresponding mean endolymph flow (ampullofugal/ampullopetal). (a) Nonlinear forces in the endolymph from Reynolds stresses, $\bar{F}_{Re}$, and from FSI in the vicinity of the vibrating walls, $\bar{F}_{w}$. (b) Optimal wave attenuation $\Psi_L$, and increasing membrane excitability $\eta_{\text{max}}$ for softer membranes at equal wavelengths ($\lambda \propto 1/\Pi$). (c) Patho-anatomical variance of the cochlear admittance $\phi$ and the stapes motility $U_s/p_s$.

in the Navier-Stokes equations (3.33b), and wall-induced nonlinearities due to the fluid-structure coupling at the vibrating membrane.

The Reynolds stresses are independent of the membrane stiffness and increase with the stimulus frequency (Fig. 5.26). In other words, it requires a certain amount of oscillation energy from the sound stimulus to generate sufficient drive on the mean endolymph flow.

The FSI across the vibrating membrane accounts only for a fraction of the nonlinear forces. We observe that the wall-induced forces reverse their direction above a certain membrane stiffness $E$ and thus weaken the overall response to sound (Fig. 5.26).

In combination, the nonlinear mechanisms are weakest at low frequencies and stiff membranes, and strongest vice versa. Fig. 5.29(a) visualizes this behaviour schematically.

Attenuation mechanisms

Results show that the attenuation length $\sigma$ is closely connected with the stimulus frequency, since $\sigma$ stays in a constant range of one to two wavelengths $\lambda$ for the majority of the cases (Fig. 5.17(c)). For frequencies just below the ring frequency $f_\pi$ ($\Pi = 1$), the corresponding wavelength
\( \lambda \) is very small, such that the effective attenuation length \( \Psi_L = \sigma / L^2 \) becomes very small as well. Therefore the membrane is hardly able to provoke nonlinear reactions since the propagating waves ‘die out’ too quickly.

We know from Gautier et al. (2007) that the wave propagation is evanescent (‘stop band’) in regions of \( \Pi \geq 1 \). Moving away from this stop band across isolines of \( \Pi \) in Fig. 5.29(b), the wave length \( \lambda \) increases. This yields a stronger vestibular response as the effective FSI area on the membrane surface increases with \( \lambda \).

However, above a certain wavelength \( \lambda_\Delta \approx L_1 / 6 \) the gain in steady streaming is alleviated by the growing intensity of wave reflections, as the weakly attenuated displacements reach the end of the canal. When we approach the line of \( \Pi = K_4 \), the wavelengths have reached the size of \( \lambda \approx L_2 \) and the steady streaming vanishes almost completely.

Along the ‘streaming corridor’ of optimal sensitivity, i.e. at \( \Pi_\Delta \), another mechanism is able to enhance vestibular responses. Walking towards lower frequencies \( f \) and softer membranes \( E \) at constant wavelength \( \lambda \), the membranous labyrinth becomes less resistive to transmural loads. This results in larger displacement maxima \( \eta_{\text{max}} \) which intensify the pulsatile character of the endolymph response, and ultimately the steady streaming.

**Influence of the (patho-)anatomy**

The superior canal dehiscence leads to a redistribution of the stapes-induced perilymph motion, away from the cochlear scalae. From predictions by Kim et al. (2013) we know how the cochlear admittance \( \phi \) drops in function of the sound frequency and the dehiscence size. At the low-frequency limit of hearing, practically all flow enters the vestibular system. The opposite happens at frequencies higher than \( f > 500 \text{ Hz} \), where the vestibular pathway features a larger impedance than the cochlea. Our computational model predicts that the Tullio phenomenon decreases with the square of the cochlear admittance, i.e.

\[
\alpha_t \propto \bar{U}_e \propto U_p^2 \propto (1 - \phi)^2.
\]

The stapes motility \( U_s / p_s \) exhibits an equally strong influence which peaks at approximately \( f = 1 \text{ kHz} \) according to measurements by Kringlebotn & Gundersen (1985), Huber et al. (2001) and Chien et al. (2009). Since \( U_s / p_s \) decays in the log-log scale by a slope of 1 towards
both lower and higher frequencies, the resulting decay in eye motion amounts to a slope of \(2\). This imposes a sharp peak on the Tullio activity map in Fig. 5.28, creating a ‘sweet spot’ for the phenomenon. Fig. 5.29(c) visualizes the corresponding mechanisms.

**Limitations of the computational model**

Outside the range of \(\Pi \in [0.05, 0.5]\), the computational model approaches its limits both numerically and phenomenologically. As already mentioned, at frequencies close to \(\Pi \lesssim 1\) occur interdependencies with longitudinal waves within the membrane. These cannot be captured, as our model accounts only for radial membrane oscillations. This could explain why our predictions do not match the patient data at \(f = 2 - 3\) kHz in Fig. 5.27(b).

Additionally, a numerical restriction appears when the wavelength \(\lambda\) becomes very small, i.e. \(\lambda \ll r_0\). In this case, the necessary spatial resolution along the centerline coordinate \(x\) increases so heavily that it renders a timely solution virtually impossible. Likewise, for very large wavelengths \(\lambda \gtrsim L_2\), the FSI coupling algorithm fails to converge.

It may also be remarked that our computational model neglects fluid motions along alternative pathways to the dehiscence. We believe that these do not contribute significantly to the observed phenomenon and such a scenario is not captured by our numerical simulations.
Concluding Remarks and Outlook

Vestibular responses to sound in presence of a superior canal dehis- cence (SCD) obey intricate mechanisms of fluid-structure interaction. In the course of the present work, we sought to identify the key parameters which generate, enhance or weaken such misperceptions of the balance sense.

Based on a computational modeling approach from a primarily fluid-dynamical perspective, we carried out numerical simulations of pure-tone stimulations across the whole bandwidth of the audible frequency spectrum. For that purpose, we specifically designed a tailor-made code which incorporates state-of-the-art methods to resolve interdependencies between the inner-ear fluids and the membranous labyrinth. The numerical solver was validated against similar studies on wave propagation in elastic tubes (Gautier et al., 2007), assuring that our results are sound and in good agreement with the literature.

An analysis of the primary flow response revealed that the staples vibrations induce traveling waves along the membranous labyrinth as the perilymph begins to oscillate forth and back in the SCC. Exposed to oscillating transmural pressures, the elastic walls of the membranous labyrinth displace and provoke further mechanical responses in the adjacent fluids (endolymph and perilymph). On their pathway through the semicircular canals towards the dehiscence, the waves are attenuated by viscous and inertial effects from both fluids. This attenuation was found to be closely coupled to the frequency of the sound stimulus.

Within the vibrating membranous duct, the endolymph responds by strong pulsations at sound frequency, thus continuously displacing the cupula forth and back. This behaviour might explain the observations from animal experiments by Carey et al. (2004) who reported a phase-locking of irregular cupula afferents to the sound stimulus.

When we calculated the temporal mean of the primary endolymph oscillations, we identified a secondary, steady flow component. This
so-called *steady streaming* corresponds to a continuous pumping of fluid uni-directionally through the slender membranous canal until the cupula reaches a balanced offset displacement. We suspect that this mechanism corresponds to the tonic behavior of regular cupula afferents (Carey et al., 2004).

Revisiting previous studies on the fluid dynamics of the balance sense during head rotation (Grieser et al., 2014), we were able to link the cupula displacement to the resulting eye velocities. Furthermore, an analytical formulation for the steady streaming was deduced from the simulation results by means of a dimensional analysis.

Our predictions for the eye motion compare well with clinical data from patients with a superior canal dehiscence. It appears that the Tullio phenomenon is restricted to a ‘sweet spot’ in the sound spectrum. This spot is expected to shift due to the individual anatomy of the patient, as the stapes oscillates strongest at certain sound frequencies (Chien et al., 2009). Additionally, this spot is confined by the degree of nonlinearity in the system response. Higher frequencies, if not overdamped, generally yield stronger vestibular reactions.

However, when the sound frequency falls into the ‘stop band’ for wave propagation (Gautier et al., 2007) at roughly 4–6 kHz, the acoustic waves become evanescent and thus the phenomenon disappears. Moreover, the impedance of the vestibular system outweighs the impedance of the cochlear pathway at high frequencies (Kim et al., 2013), such that the balance sense becomes less responsive to sound.

We further noted that both the location and the size of the dehiscence play a role in the Tullio phenomenon, although this is subordinate to the other effects: larger dehiscences further away from the stapes yield stronger responses.

The present thesis has identified mechanisms which may explain the Tullio phenomenon. In order to confirm the validity of our predictions, follow-up studies may be carried out *in vitro* on upscaled models of the membranous labyrinth. Since we are providing an analytical correlation in dimensionless form, the corresponding experimental setup can be designed relatively easily by maintaining the dimensionless numbers.

Making minor modifications, our computational model could also be used to investigate more complex sound stimuli. If the reported sweet spot resonance can be canceled out by means of destructive interference
with other frequencies, one could actively suppress vestibular responses ('intelligent earphones'). In such a scenario, the patient would have to trade sensations of vertigo with the exposure to interference noise. Alternatively, using the modern technology of hearing aids, it is conceivable that the intensity of the incoming sound spectrum were to be selectively attenuated. That way, vestibular reactions could be alleviated by lowering the sound pressure level of trigger frequencies for the Tullio phenomenon.

We may conclude this thesis by emphasizing and confirming the statement by Rabbitt et al. (1999): ‘increasing the membrane stiffness would make canal plugging more effective in attenuating cupular responses, whereas reducing the stiffness would cause the opposite’. If ways can be found to artificially stiffen the membranous labyrinth on the vestibular side, it should be possible to alleviate the symptoms for patients with SCD, maybe even without the need for surgery.
Appendix A

Pseudo-viscous damping

In the following we derive the pseudo-viscous parameter $\epsilon_p$ which models the damping characteristics of the perilymph in strongly pulsating flows such as they occur in the Tullio phenomenon. $\epsilon_p$ was introduced in the momentum equation (3.17) and attempts to capture radial contributions from viscous stresses and inertial effects which cannot be resolved by the one-dimensional model given by

$$\frac{1}{A_p} \frac{\partial (A_p u_p)}{\partial t} + \epsilon_p u_p = - \frac{1}{\rho_f} \frac{\partial p_p}{\partial x}. \qquad (A.1)$$

Obrist (2014) obtained an exact solution for unsteady pipe flows with harmonic forcing,

$$u(r, t) = \frac{i}{\omega} \cdot \left( 1 - \frac{J_0(i^{3/2} W_0 \ r/a)}{J_0(i^{3/2} W_0)} \right) \cdot \frac{1}{\rho_f} \frac{\partial p}{\partial x}(t), \qquad (A.2)$$

where $J_0$ denotes the Bessel function of first kind and zeroth order, $i$ the imaginary unit, $a$ the pipe radius and $W_0$ the Womersley number,

$$W_0 \equiv \frac{a}{\delta_\nu} = a \sqrt{\frac{\omega}{\nu_f}}, \quad (A.3)$$

related to the Stokes boundary layer thickness $\delta_\nu$. Integration of (A.2) in the radial direction $r' \equiv r/a$ leads to the bulk velocity $U$,

$$U(t) = \frac{a^2}{a^2 \pi} \int_0^1 \left[ u(r', t) \cdot 2\pi r' \right] \, dr' = \frac{W}{\omega} \cdot \frac{1}{\rho_f} \frac{\partial p}{\partial x}(t), \quad (A.4)$$

where $W$ is a dimensionless and complex-valued function of the Womersley number,

$$W = 2i \int_0^1 \left( 1 - \frac{J_0(i^{3/2} W_0 \ r')}{J_0(i^{3/2} W_0)} \right) \, r' \, dr'. \quad (A.5)$$

A steady ($W_0 \ll 1$), laminar ($R_e \ll 1$) pipe flow results from the balance of viscous stresses with the pressure gradient, i.e.

$$-\nu_f \left( \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial}{\partial r} u(r) \right) \right) = - \frac{1}{\rho_f} \frac{\partial p}{\partial x}. \quad (A.6)$$
The solution of (A.6) can be directly obtained from the Hagen-Poiseuille law, stating that the bulk velocity $U_{HP}$ is identical to

$$U_{HP} = -\frac{a^2}{8\nu_f} \cdot \frac{1}{\rho_f} \frac{\partial p}{\partial x}.$$  \hspace{1cm} (A.7)

In other words, the viscous stresses on the left-hand-side of (A.6) scale linearly with the bulk velocity $U_{HP}$ as

$$-\nu_f \left( \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial}{\partial r} u(r) \right) \right) = \epsilon_{HP} U_{HP},$$  \hspace{1cm} (A.8)

with a proportionality constant $\epsilon_{HP}$ given by

$$\epsilon_{HP} = \frac{8\nu_f}{a^2} = \frac{8\omega}{W_0^2}. \hspace{1cm} (A.9)$$

Since the right-hand side of (A.8) resembles our pseudo-viscous model term in the momentum balance (A.1), we may postulate that

$$\epsilon_{HP} U_{HP} = \epsilon_{HP} \frac{U_{HP}}{U(t)} U(t) = \epsilon_{HP} \left| \frac{U_{HP}}{U(t)} \right| u_p = \epsilon_p u_p,$$  \hspace{1cm} (A.10)

such that the pseudo-viscous damping becomes

$$\epsilon_p = \omega \frac{1}{|W|} = \epsilon_{HP} \frac{W_0^2/8}{|W|}. \hspace{1cm} (A.11)$$

![Figure A.1: Correction factor $|W|$ for the pseudo-viscous damping $\epsilon_p$, obtained from numerical evaluations of the complex-valued integral in (A.5) in function of the Womersley number $W_0$ (A.3) with a radial resolution of 1000 computational nodes.](image_url)
Fig. A.1 shows how the pseudo-viscous damping $\epsilon_p$ recovers the Hagen-Poiseuille limit $\epsilon_{HP}$ at flows of low Womersley numbers where $\mathcal{W}$ becomes $Wo^2/8$ and thus

$$\epsilon_p \approx \epsilon_{HP}, \quad Wo \ll 1.$$  \hfill (A.12)

Towards the upper limit at high Womersley numbers, the damping $\epsilon_p$ becomes identical to the angular frequency $\omega$ of the sound stimulus,

$$\epsilon_p \approx \omega, \quad Wo \gg 1.$$  \hfill (A.13)

Since the stapes-induced flows within the perilymph are characterized by high Womersley numbers significantly larger than unity, expression (A.13) represents a valid approximation in the frequency spectrum in which the Tullio phenomenon is observed.

A ‘sanity check’ may be performed by approximating the second derivative in the viscous stress with the bulk velocity $u_p$ divided by the square of the Stokes boundary layer thickness, $\delta_\nu$, i.e.

$$-\nu_t \left( \frac{1}{r} \frac{\partial}{\partial r} \left( r \frac{\partial}{\partial r} u(r) \right) \right) \approx \nu_t \frac{u_p}{\delta_\nu^2} = \omega u_p,$$  \hfill (A.14)

confirming our findings in (A.13).
Appendix B

Derivation of the perilymph pressure gradient

In Chapter 3.2.1 we claim that

$$\frac{1}{\rho} \frac{\partial^2 p}{\partial x^2} = \frac{1}{A_p} \frac{\partial^2 A_p}{\partial t^2} + \frac{\epsilon_p}{A_p} \frac{\partial A_p}{\partial t} - \frac{1}{A_p^2} \left( \frac{\partial A_p}{\partial t} \right)^2 +$$

$$+ \left( \frac{\partial}{\partial t} + \epsilon_p \right) \cdot \left( \frac{u_p}{A_p} \frac{\partial A_p}{\partial x} \right) - \frac{\partial}{\partial x} \left( \frac{u_p}{A_p} \frac{\partial A_p}{\partial t} \right) \quad (3.22)$$

may be rewritten as

$$\frac{\partial^2 p}{\partial x^2} = -2\pi \rho_r \frac{r}{A_p} (\eta_{tt} + \epsilon_p \eta_t) - 2\pi \rho_r \frac{1}{A_p} \eta_t^2 + 2\pi \frac{1}{A_p} \frac{\partial p}{\partial x} \eta_x. \quad (3.23)$$

In a first step, we expand the expression in the second line of (3.22) as follows:

$$\left( \frac{\partial}{\partial t} + \epsilon_p \right) \cdot \left( \frac{u_p}{A_p} \frac{\partial A_p}{\partial x} \right) - \frac{\partial}{\partial x} \left( \frac{u_p}{A_p} \frac{\partial A_p}{\partial t} \right) =$$

$$= \frac{u_p}{A_p} \frac{\partial^2 A_p}{\partial x \partial t} + u_p \frac{\partial A_p}{\partial x} \frac{\partial}{\partial t} \left( \frac{1}{A_p} \right) + \frac{1}{A_p} \frac{\partial A_p}{\partial x} \frac{\partial u_p}{\partial t} + \epsilon_p \frac{u_p}{A_p} \frac{\partial A_p}{\partial x}$$

$$- u_p \frac{\partial^2 A_p}{A_p \partial x \partial t} - u_p \frac{\partial A_p}{\partial t} \frac{\partial}{\partial x} \left( \frac{1}{A_p} \right) - \frac{1}{A_p} \frac{\partial A_p}{\partial t} \frac{\partial u_p}{\partial x}$$

$$= - \frac{u_p}{A_p^2} \frac{\partial A_p}{\partial t} \frac{\partial A_p}{\partial x} + \frac{1}{A_p} \frac{\partial A_p}{\partial x} \frac{\partial u_p}{\partial t} + \epsilon_p \frac{u_p}{A_p} \frac{\partial A_p}{\partial x}$$

$$+ \frac{u_p}{A_p^2} \frac{\partial A_p}{\partial x} \frac{\partial A_p}{\partial t} - \frac{1}{A_p} \frac{\partial A_p}{\partial t} \frac{\partial u_p}{\partial x}$$

$$= \frac{1}{A_p} \left( \left[ \frac{\partial u_p}{\partial t} + \epsilon_p u_p \right] \frac{\partial A_p}{\partial x} - \left[ \frac{\partial u_p}{\partial x} \right] \frac{\partial A_p}{\partial t} \right). \quad (B.1)
Applying (3.19) and (3.15) at the respective bracketed expressions, we rewrite (B.1) as

$$\frac{1}{A_p} \left( \left[ \frac{\partial u_p}{\partial t} + \epsilon_p u_p \right] \frac{\partial A_p}{\partial x} - \left[ \frac{\partial u_p}{\partial x} \right] \frac{\partial A_p}{\partial t} \right) =$$

$$= \frac{1}{A_p} \left( - \frac{1}{\rho_f} \frac{\partial p}{\partial x} - \frac{u_p}{A_p} \frac{\partial A_p}{\partial t} \right) \frac{\partial A_p}{\partial x} - \left[ - \frac{u_p}{A_p} \frac{\partial A_p}{\partial x} - \frac{1}{A_p} \frac{\partial A_p}{\partial t} \right] \frac{\partial A_p}{\partial t}$$

$$= \frac{1}{A_p} \left( - \frac{1}{\rho_f} \frac{\partial p}{\partial x} \frac{\partial A_p}{\partial x} + \frac{1}{A_p} \left( \frac{\partial A_p}{\partial t} \right)^2 \right). \tag{B.2}$$

Thus (3.22) becomes

$$\frac{1}{\rho_f} \frac{\partial^2 p}{\partial x^2} = \frac{1}{A_p} \frac{\partial^2 A}{\partial t^2} + \frac{\epsilon_p}{A_p} \frac{\partial A}{\partial t} - \frac{1}{A_p^2} \left( \frac{\partial A}{\partial t} \right)^2 +$$

$$+ \frac{1}{A_p} \left( - \frac{1}{\rho_f} \frac{\partial p}{\partial x} \frac{\partial A}{\partial x} + \frac{1}{A_p} \left( \frac{\partial A}{\partial t} \right)^2 \right)$$

$$= \frac{1}{A_p} \frac{\partial^2 A}{\partial t^2} + \frac{\epsilon_p}{A_p} \frac{\partial A}{\partial t} - \frac{1}{\rho_f} \frac{1}{A_p} \frac{\partial p}{\partial x} \frac{\partial A}{\partial x}. \tag{B.3}$$

Using geometrical relations (3.10), the expression in (B.3) can be finally rewritten as

$$\frac{\partial^2 p}{\partial x^2} = -2\pi \rho_f \frac{r}{A_p} (\eta_{tt} + \epsilon_p \eta_t) - 2\pi \rho_f \frac{1}{A_p} \eta_t^2 + 2\pi \frac{1}{A_p} \frac{\partial p}{\partial x} \eta_x, \tag{B.4}$$

q.e.d.
Appendix C

Ramp function for numerical loading

For numerical reasons, we apply a ramp function $\mathcal{R}$ to the stapes boundary condition $g(t)$ in (3.29) such that the stapes motion increases smoothly from zero to maximum amplitude within the first five sound periods $T$. The ramp function is defined as

$$\mathcal{R}(t) \equiv \frac{1 + \tanh \left( \tau_{99} \left[ t - \frac{t_{99}}{2} \right] \right)}{2}, \quad (C.1)$$

with

$$\tau_{99} = \frac{\ln(99)}{t_{99}}, \quad (C.2)$$
$$t_{99} = 5T, \quad (C.3)$$

such that the numerical window is 99% closed at $t=0$ and 99% open at $t=t_{99}$. Fig. C.1 shows the ramp function $\mathcal{R}(t/T)$.

Figure C.1: Ramp function $\mathcal{R}$ (C.1), applied to the stapes boundary condition $g$ (3.29). The amplitude of $g(t)$ is denoted by $g_0$, the sound period by $T$. 


Grieser, B., Obrist, D. & Kleiser, L. 2012 Validation of assumptions on the endolymph motion inside the semicircular canals of the inner ear. *ETH E-Collections* ETH Zürich, doi:10.3929/ethz-a-007588055.


Minor, L. B., Solomon, D., Zinreich, S. J. & Zee, D. S. 1998 Sound-and/or pressure-induced vertigo due to bone dehiscence of the


OBRIST, D. 2011 Fluid mechanics of the inner ear. Habilitation treatise, ETH Zürich, DOI:10.3929/ethz-a-007318979.


TULLIO, P. 1929 Das Ohr und die Entstehung der Sprache und Schrift. Berlin: Urban & Schwarzenberg.


WIT, H. P., SCHEURINK, A. J. W. & BLEEEKER, J. D. 1985 Hearing thresholds of normal and fenestrated deaf pigeons. A behavioural study


Publications


Grieser, B., Obrist, D. & Kleiser, L. 2012 Validation of assumptions on the endolymph motion inside the semicircular canals of the inner ear. *ETH E-Collections* ETH Zürich, doi:10.3929/ethz-a-007588055.


# Curriculum vitae

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Patients with a superior canal dehiscence (SCD) in the inner ear suffer from events of dizziness and vertigo in response to sound, also known as Tullio phenomenon (TP). To date, the mechanisms behind TP remain obscure. When risky surgical interventions appear to be the only means to cure the symptoms, it may be worthwhile to study the underlying mode of operation and possibly devise less invasive methods.

Approaching this medical condition from an engineering perspective, we are faced with an intertwined system of fluids (endolymph and perilymph), elastic structures (membranous labyrinth, cupula, dehiscence) and rigid bone (temporal bone, stapes). In accordance with the so-called ‘third window theory’, we assume that the vibrating stapes causes abnormal perilymph pulsations towards the pathologic ‘window’ in the superior canal of the balance sense. Based on this assumption, we developed a computational model in order to resolve fluid-structure interactions which we expect to arise from such a coupled system.

The simulation results confirm our hypothesis, revealing the occurrence of wave propagation phenomena along the deforming membranous canal. More specifically, we note that two substantially different flows are evolving. First, the deforming labyrinth causes pulsations of the endolymph which lead to rapid vibrations of the cupula in phase with the sound stimulus. Second, these primary pulsations feature a static component, the so-called steady streaming, such that endolymph is continuously driven through the canals in (mostly) ampullofugal direction. Reaching a quasi-steady balance with the opposing cupula, the latter maintains a constant deflection amplitude. Both findings are in agreement with clinical observations on the cupula response in patients with SCD.

Carrying out a sensitivity study, we were able to obtain an analytical fit to match our simulation results in a relevant range of parameters. We coupled the inner-ear dynamics to the corresponding eye response (vestibulo-ocular reflex). The results reveal a ‘sweet spot’ for TP within the audible spectrum which largely coincides with patient data. We found that the underlying mechanisms originate primarily from Reynolds stresses in the fluid, which are weakest in the lower sound spectrum. Additionally, natural variations in the membrane stiffness and the stapes motility are observed to shift the sweet spot. Waves become evanescent above 4-6kHz, such that we cannot expect vestibular responses in that range.