Fluid-Structure Interaction Model of Active Eustachian Tube Function in Healthy Adult Patients

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Abstract: The Eustachian Tube (ET) is a collapsible tube that connects the middle ear to the nasopharynx. Dysfunction in the ET leads to a disease called Otitis Media. Our goal is to develop and use fluid-structure interaction (FSI) models of ET opening phenomena to better understand what factors contribute to ET dysfunction in certain patients. We model the ET in COMSOL using geometric data obtained from histological specimens. Using histological data allows us to make anatomically accurate models which are then used to simulate the clinical tests performed on patients. This serves as an excellent validation tool for our COMSOL model. We have created a FSI model of a healthy ET which successfully simulated the large tissue deformations that occur during ET opening. We found that higher order basis functions were required in order to prevent element failure. We are currently extending this model to account for transient dynamics and two-way coupling.

Keywords: Eustachian Tube, Otitis Media, fluid dynamics, finite element analysis, fluid-structure interaction

1. Introduction

The Eustachian Tube is a collapsible tube that connects the Middle Ear (ME) to the nasopharynx and has three primary functions: 1) regulation of ME pressure 2) protection of the ME from foreign pathogens and 3) drainage of fluid from the ME. In healthy patients, the ET opens during swallowing because the surrounding tissue is deformed by muscle activity. If the ET fails to open, the ME develops painful sub-ambient pressure and fluid accumulates in the ME. ET dysfunction results in Otitis Media (OM), the most commonly diagnosed disorder in young children and costs the U.S. economy $4 billion dollars annually to treat. The overall goal of our lab is to identify the mechanisms responsible for ET dysfunction and to develop novel treatments for OM that seek to restore normal ET dysfunction.

The ET is a complex, 3D structure made of primarily cartilage and glandular soft tissue surrounded by bone and muscle.

Figure 1. Typical cross section of the Adult Eustachian Tube

There are two primary muscles that act on the ET, the tensor veli palatini muscle (TVPM), and the levator veli palatini muscle (LVP). The TVPM is a ribbon muscle that pulls on the superior faces of the cartilage and glandular tissue while the LVP is a muscle bundle that pushes up on the inferior faces of the cartilage and glandular tissue. When the two muscles contract, the soft tissue is deformed and a lumen opening is generated for air to flow or mucus to drain. The goal of this study is to develop a fully coupled fluid-structure interaction (FSI) model of ET opening and closing phenomena that can replicate the transient flow dynamics observed experimentally in OM patients. The long term goal is to use these FSI models to analyze patient specific data and develop patient-specific therapies for ET dysfunction.

2. Methods

Histological data of the ET’s soft tissue is obtained from the University of Pittsburgh’s Otopathology laboratory located within the
Children's Hospital of Pittsburgh. This data is then processed with standard protocols within our lab which uses Rhinoceros V3.0 CAD software to produce 3D solid models of cartilage, glandular tissue, and lumen opening. Rhinoceros then exports IGES files. These three solid bodies are then imported into COMSOL using the CAD Import Module.

Figure 2. Three domains in the COMSOL model

Pictured in Figure 2 are the three subdomains present in the COMSOL model. The green domain represents the cartilage, the red domain represents the glandular tissue, and the blue domain represents the lumen opening. To build the FSI model, multiple COMSOL application modes are used. First, the structural mechanics module is included in a fixed reference frame. Then the ALE moving mesh application mode is used to define a moving reference frame for the fluid domain. Then, the Fluid Mechanics module is used in the moving reference frame. Since multiple modules are used, subdomain settings and boundary conditions must be applied in all three modes.

The solid domain consists of both the cartilage and glandular tissue, both represented by a Mooney-Rivlin hyper-elastic material model. This material model is described by equation (1).

\[ W_S = C_{10} (I_1 - 3) + C_{01} (I_2 - 3) \]  

where \( W_S \) is the strain energy density function, \( I_1 \) and \( I_2 \) are the strain invariants, and \( C_{10} \) and \( C_{01} \) are material constants. There are two physical boundary conditions that need to be put into the numerical model. 1) Attachment of the cartilage to the cranial base of the skull and 2) Attachment the distal and proximal ends of the cartilage and glandular tissues to bony portions of the ET via tendons. To represent physical condition 1, a zero displacement boundary condition is assigned to the face of the cartilage where it touches the cranial base. To represent physical condition 2, the proximal and distal faces of the cartilage and glandular tissue have a zero normal displacement since the tendons only allow for in-plane motion. Also in the solid domain, the forces due to the TVPM and LVPM need to be represented. The LVPM is a muscle bundle that exerts a force normal to the surface that it acts on. Therefore, all the inferior surfaces of the cartilage and glandular tissue have a pressure with the correct magnitude prescribed. The TVPM is a ribbon-like muscle that descends inferiorly and wraps around the pytergoid hamulus bone. As a result, the TVPM produces force vectors which all point to one specific location in space, i.e. the hamulus location. The muscle pulls on the superior faces of the cartilage and glandular tissue and thus pulls to the hamulus location. A routine was created that calculates the three components of the TVPM force using equations in the Global Expressions list, given the hamulus location.

Figure 3. Load plot due to muscle forces

Figure 3 visualizes the loads that are on the soft tissue due to muscle forces, noting the directions of the forces vectors.

The ALE module is used to define a moving reference frame for the fluid domain. Both the cartilage and glandular tissue are specified to have 'Physics Induced Displacements' with displacement components u, v, and w while the fluid domain is allowed to displace freely. The ALE module uses a Winslow smoothing technique to solve for the
displacements of the fluid domain. The Winslow method is described by the following equations.

\[
\frac{\partial^2 \phi}{\partial x^2} + \frac{\partial^2 \phi}{\partial y^2} + \frac{\partial^2 \phi}{\partial z^2} = 0 \tag{2}
\]

\[
\frac{\partial^2 \psi}{\partial x^2} + \frac{\partial^2 \psi}{\partial y^2} + \frac{\partial^2 \psi}{\partial z^2} = 0 \tag{3}
\]

\[
\frac{\partial^2 \omega}{\partial x^2} + \frac{\partial^2 \omega}{\partial y^2} + \frac{\partial^2 \omega}{\partial z^2} = 0 \tag{4}
\]

where X,Y,Z are the fixed coordinate system and x,y,z are degrees of freedom solved for in the problem. The walls of the lumen are identified as FSI interfaces so they are assigned a known displacement of u, v, and w in their respective directions. The proximal and distal ends of the lumen opening have a zero normal displacement boundary condition, again due to the physical condition 2 as stated earlier.

In the fluid domain, the working fluid is air and the flow is governed by the incompressible continuity and Navier-Stokes equations.

\[
\frac{\rho}{\rho} \frac{\partial v_x}{\partial x} = 0 \tag{5}
\]

\[
\frac{\rho}{\rho} \frac{\partial v_x}{\partial t} + \rho v_x \frac{\partial v_x}{\partial x} = -\frac{\partial p}{\partial x} + \mu \frac{\partial^2 v_x}{\partial x^2} \tag{6}
\]

where \(v_x\) is the fluid velocity vector, \(p\) is the fluid pressure, and \(X_i\) represents the coordinates in the moving reference frame. For these models, we are trying to replicate clinical test data, so boundary conditions in the fluid domain are set by clinical test protocols. On the upstream end of the ET, or ME side, a normal stress of 200 mm H\(_2\)O, or 1961 Pa scals is applied. On the downstream end, or nasopharynx side, a normal stress of 0 mm H\(_2\)O, or 0 Pascals is applied. Due to the complex geometry and large aspect ratio of the lumen opening, it presents some very complicated meshing challenges. To solve this problem, a relatively coarse mesh is used but implemented with high order basis functions.

Figure 4 is a picture showing the coarse mesh used in the fluid domain. The lumen opening only has two elements across it where the fluid is flowing. The picture mesh is implemented with 4th order basis functions for the fluid velocities and third order basis functions for fluid pressure. The higher order basis functions allow the solver to accurately capture the flow field with so few elements. It is this feature that allows the mesh to undergo the large deformations seen during ET opening with having any elements collapse on themselves.

This method difference can easily be verified. In Ghadiali et al. [2], an analytical solution is derived for pressure driven flow through a spinal needle that is 0.3 mm in diameter and 8 cm in length. The analytical solution gives the following relationship between pressure difference and flowrate through the needle.

\[
\Delta P = \frac{\mu Q}{A^2} \Gamma \tag{7}
\]

where \(\Delta P\) is the pressure drop across the length of the needle, \(L\) is the length of the needle, \(A\) is the cross sectional area of the needle, \(\mu\) is the viscosity of the working fluid, and \(\Gamma\) is a hydraulic shape factor based only on the geometry. Using air as the working fluid, a 200 mm H\(_2\)O pressure drop through the needle, L=8 cm, A=\(\pi R^2\) for a circle, and \(\Gamma=8\pi\) for a circle, the flowrate \(Q\) can be found to be 2.703e-7 m\(^3\)/s. The spinal needle is then modeled in COMSOL to see if COMSOL will calculate the same flowrate. The mesh used in solving the flow field is shown in Figure 5.

![Figure 4. Mesh in the fluid domain](image)

![Figure 5. Mesh used in COMSOL verification model](image)
The mesh used in the verification model has 24 elements per cross section, then the mesh is extruded down the length of the needle with 35 divisions resulting in a total of 840 elements. After solving the flow field, COMSOL calculates the flowrate through the needle as $2.707 \times 10^{-7}$ m$^3$/s. This proves that using fewer, higher order elements is just as accurate as more, lower order elements.

3. Results

Using a novel set of higher order basis functions proved to be an effective way to accurately model the large displacements seen during ET function.

Note that the deformations in Figure 66 are 1:1 scale and that the maximum displacement in solid domain was $1.5 \times 10^{-3}$ mm. Another very important factor to note in these biological systems is the very high strains. In this model, the maximum effective strain is 65%. Effective strain is calculated according to the following equation.

$$
\varepsilon_{\text{eff}} = \sqrt[3]{\frac{2}{3} (\varepsilon_x + \varepsilon_y + \varepsilon_z) + 2 (\varepsilon_{xy} + \varepsilon_{yz} + \varepsilon_{xz})}
$$

Figure 7 shows a plot of the effective strain in the soft tissue surrounding the lumen opening. All of these displacements in the solid domain resulted in a 95% increase in lumen volume. This increase in lumen volume resulted in a 131% increase in flowrate through the lumen.

In the fluid domain, the maximum velocity was 18.1 m/s, giving a maximum Re of 375. So the initial presumption of laminar flow was correct.

This model demonstrates COMSOL’s capabilities to model the large deformations seen in biological systems and couple that deformation with a fluid domain that also undergoes very large deformations. However, at this point, the model is only stationary. More work needs to be done to convert this into a model that capture the transient dynamics observed in experimental data.

6. Other Eustachian Tube Analysis

We have also used another FEA package well known for its FSI capabilities, ADINA, to model the transient dynamics that occur during ET opening with limited success.
The results from these ADINA simulations are shown in Figure 9. For these simulations we varied the contraction sequence of the TVPM and LVPM in order to produce transient changes in flow rate. Although the data in Figure 9 qualitatively matches experimental data, i.e. it contains appropriate increases and decreases in flow rate; the main limitation in ADINA is the limited types of elements available. ADINA can only implement first order fluid elements. This means that many elements are needed to discretize the fluid domain. When the full physiological forces are put into the simulation, the simulation fails due to element collapse. As a result, Figure 9 was produced with muscle force magnitudes an order of magnitude lower than the physiological magnitudes. In the ADINA simulations, the flowrate only changes approximately 1% while in the experiments the flowrate changes 150%. We believe that the higher-order elements developed in our COMSOL model will prove useful in solving this problem and will therefore be able to eventual accurately match the experimental data.

5. Conclusions

Although the ADINA FE program can solve for fluid-structure interactions within the ET, this program was not able to simulate the large tissue and fluid domain deformations that occur in-vivo. Specifically, it was only able to solve the small deformation problem due to its inability to implement higher order basis functions. We have also shown that COMSOL is capable of solving the large deformation problem; however it is currently not a transient simulation. Future plans include making the current model transient so that it can replicate experimental data from real patients. Once a transient model is created, it will be used to conduct parameter variation studies to identify how various parameters impact ET function and to help guide/identify surgical/pharmacutical therapies that restore ET function.

6. Acknowledgements

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7. References