2.6

Biomechanical evaluation of eustachian tube function and its role in regulating middle ear pressures

SN. Ghadiali[1], ED. Bell[1], CM. Alper[2], JD. Swarts[2], CD. Bluestone[2], Bethlehem[1], Pittsburgh[2]; USA

Purpose: The Eustachian tube (ET) is a collapsible respiratory airway which connects the nasopharynx (NP) with the middle ear (ME). In addition to other physiological functions, the ET is responsible for maintaining ambient pressures within the ME. The ET normally regulates ME pressures by periodically opening during swallowing and transporting gas between the NP and ME. However, the inability to open the ET results in sub-ambient ME pressures, fluid transudation into the ME and the development of persistent Otitis Media (OM). The goal of this study is to develop a mathematical/computational model of the opening and pressure regulation functions of the ET. These models have been correlated with experimental ME pressure measurements obtained in both healthy adults and young children with OM in order to identify the biomechanical properties that may be responsible for ET dysfunction.

Material and Methods: Cross-sectional histological images from temporal bone specimens were used to create anatomically-accurate finite element (FE) models of the ET's soft tissue structures (i.e. cartilage and fat tissue). The mechanical properties of the various tissue elements were based on direct experimental measurements and ET opening phenomena during swallowing was simulated by specifying an asynchronous muscle contraction sequence. Finally, molecular dynamics simulations were used to quantify the adhesive properties of glycoproteins in the ET. Changes in the open lumen area, ME pressure and air flow rate during swallowing were calculated and these parameters were compared with experimental measurements.

Results: For healthy adults, our models indicate that ET opening is a three-staged event. First, contraction of the levator veli palatini muscle (LVPM) results in a small decrease in lumen area (i.e lumen constriction). Second, contraction of the tensor veli palatini muscle (TVPM) results in a large dilation or opening of the ET lumen. Finally, sustained contraction of the LVPM can result in a post-swallow constriction of the ET lumen. In addition, our mathematical models indicate that the expression of mucoadhesive glycoprotiens during inflammatory OM can significantly increase mucosal adhesion forces and may therefore result in an inability to open the ET.

Conclusions: By correlating sophisticated mathematical models with experimental data, this study has provided important insights into the biomechanical mechanisms responsible for ET dysfunction. This work was supported by a grant from the NIH DC005345. SNG is a Parker B. Francis Fellow in Pulmonary Research.