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Obstructive sleep apnea syndrome.

Part 2 : computer simulation of the fluid-structure interaction

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Introduction

Obstructive sleep apnea (OSA) is defined as the intermittent cessation of breathing during sleep, caused by airway obstruction (1). To study this phenomenon, a physical model of the airflow inside a constriction is described in (2), associated to experimental data from an in-vitro set-up. This paper introduces a numerical model of the fluid-walls interaction in the pharynx and then compares simulations with experimental data.

Material and Methods

In (2) is presented an experimental setup, that tries to reproduce the morphology of the upper airways. The « tongue » is a cylinder inserted into « the pharynx », a square rigid pipe. The cylinder can either be rigid or deformable, with latex full of water. On figure 1, the finite element model of the latex cylinder is plotted at its rest position in black. It reproduces latex mechanical properties (Young modulus = 1.5 MPa, Poisson's ration = 0.49). An internal pressure of 400 Pa over atmosphere is applied (water pressure). The geometry of the model and the boundary conditions have also been chosen as close as possible as those of the setup. The pressure due to the airflow is computed using the Bernoulli law and an assumption on the location of the flow separation point (3). Resulting force on surface of each element is obtained by integration of pressure. Because of small deformation hypothesis, a linear relation between constraints and deformations can be computed using a finite element solver (AnsysTM). Model deformation induces a new geometry, thus a new pressure distribution from which comes a new deformation. The algorithm stops when equilibrium is obtained (quasi-static hypothesis).

Results

First simulations are provided with a pressure drop of 800 Pa. The model is thus deformed due to the application of a negative pressure in the constriction area (Venturi effect) and a resulting decrease of the constriction height is observed (fig. 1, grey). This behaviour is in agreement with in-vitro experiments. Moreover, volume flow velocity limitation is observed (fig. 2), which is coherent with in-vivo measurement provided during hypopnea (4).

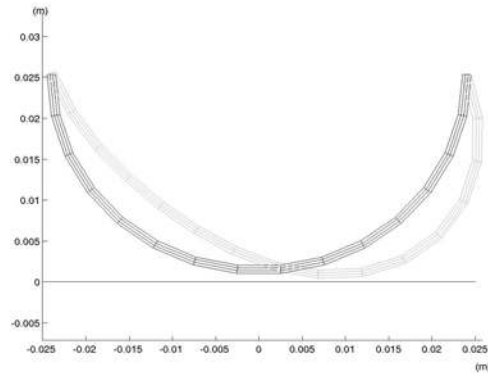


Fig. 1 : The model in rest position (black), and deformed after an inlet pressure of 800 Pa (grey). 64 finite elements are used for the cylinder wall.

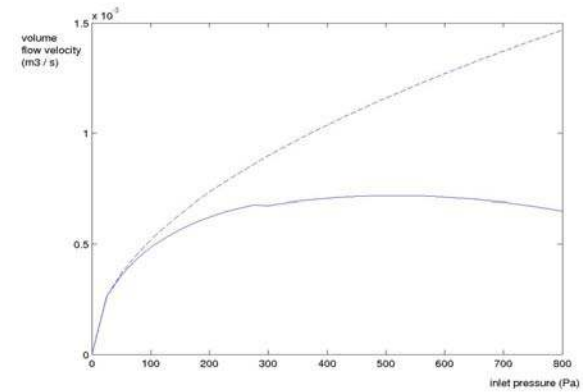


Fig. 2 : Curves volume flow rate / inlet pressure, for the deformable model (solid line), and a rigid model (dashed line).

Discussion and Conclusion

Using a simple but physically founded numerical model of the interaction between airflow and pharyngeal walls, encouraging preliminary results were obtained. The next step will be to quantitatively compare the model predictions with in-vitro measurements. The model will be improved, first, to get a behaviour as realistic as possible, next, to be in agreement with anatomical and physiological considerations. These conditions are fundamental if we want to use the model for sleep apnea syndrome prediction.

References

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