FINITE ELEMENT MODELS OF THE TONGUE AND VELUM FOR A PHYSICAL UNDERSTANDING OF SLEEP APNEA SYNDROME

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1. ABSTRACT

Sleep Apnea Syndrome (SAS) is defined as a partial or total closure (collapse) of the patient's upper airways during sleep. From a fluid mechanical point of view, this collapse can be understood as a spectacular example of fluid-walls interaction. Indeed, the upper airways are delimited by soft tissues having different geometrical and mechanical properties: velum, tongue and pharyngeal walls. Airway closure during SAS comes from the interaction between these soft tissues and the inspiratory flow.

The aim of this work is to understand the physical phenomena at the origin of the collapse and the metamorphosis occurring in the inspiratory flow pattern that has been reported during SAS. Indeed, a full comprehension of the physical conditions allowing this phenomenon is a prerequisite to provide computer-based assistance in the planning of the surgical acts that can be prescribed for the patients. Surgical techniques used for the treatment of the SAS can either reduce the volume of the tongue or stiffen the velum, or try to have a more global and progressive action on the entire upper airways.

The work presented here focuses on a simple but coherent model of fluid-walls interactions. The equations governing the airflow inside a constriction are coupled with a Finite Element (FE) biomechanical model of the velum and with a FE model of the tongue. The geometries of those two models were extracted from a single midsagittal radiography of a patient. The tongue model integrates the main extrinsic and intrinsic muscles that are responsible for tongue deformations and displacements inside the mouth (genioglossus, hyoglossus, styloglossus, verticalis, longitudinalis)

The lingual and velar deformations induced by muscle activations and by airflow interactions are computed, presented, discussed and compared to measurements.

Keywords: Sleep Apnea Syndrome, Finite Element Modelling, Fluid/wall interaction

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2. INTRODUCTION

Airway closure during sleep apnea syndrome (SAS) comes from the interaction between inspiratory flow and the walls of the upper airway that are formed by the succession of anatomical and physiological singularities with their own geometrical and mechanical properties [1]. The tongue, the pharynx and the velum are the main anatomical structures implicated in the collapse (figure 1).

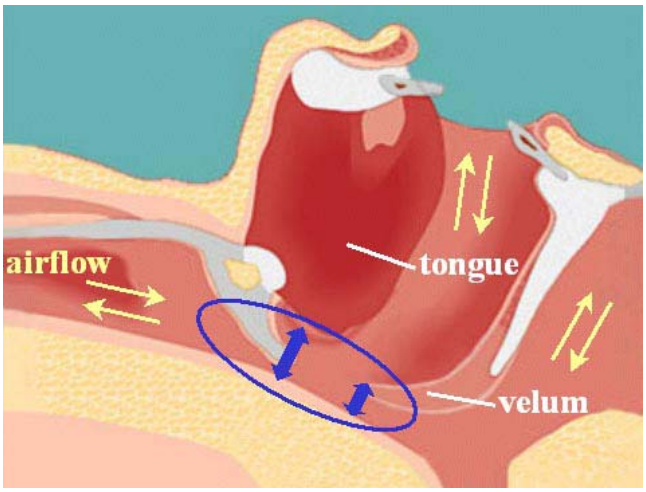

Fig. 1: Sleep Apnea Syndrome: fluid (airflaw) / wall interactions.

Surgical techniques used for the treatment of the SAS can either reduce the volume of the tongue or stiffen the velum, or try to have a more global and progressive action on the entire upper airways. In order to fully understand the metamorphosis occurring in the inspiratory flow pattern that has been reported during SAS, some mechanical models of the upper airway have been developed, assuming that upper airways can be represented by a single compliant segment ([2], [3]), or by series of individual segments representing singularities [4]. In this aim, a complete biomechanical model of the upper airways appears thus to be interesting, to describe and explain — at the physical point of view — the upper airway obstruction. This papers aims at introducing 2D Finite Element models of the tongue and velum. Then, a physical model of the airflow through the upper airways is presented and coupled to the biomechanical model of the velum. First simulations are presented and discussed.

3. TONGUE MODEL

Human tongue is a soft, continuous and quasi-incompressible structure, consisting of a complex interdigitating of muscular fibers, glands and mucous membranes. Its shape depends on the recruitment of ten muscles or so, some of them being internal to the structure. Our tongue model was originally developed in the framework of an articulatory speech synthetiser [5]. A first important stage in limiting the complexity of the model was to reduce the description of tongue structure to the midsagittal plane. The method chosen to describe tongue elasticity and incompressibility was the Finite Element Method (FEM). A set of 63 nodes, delimiting 48 isoparametric elements (with four nodes each) defines the FE structure. This structure was designed to fit the upper airways contours (measured from sagittal radiographic images) of a "reference" patient (figure 2a). Muscles whose actions have significant influences on tongue shaping in the sagittal plane (*styloglossus, genioglossus, hyoglossus, superior and inferior*

longitudinalis, verticalis) were modeled by "macro-fibers", acting on specific nodes of the FE structure. Elastic parameters of this FE structure were chosen to model tongue quasi-incompressibility (Poisson's ratio $\nu = 0.49$) and stiffness (Young modulus $E = 15$ kPa). Those parameters were validated by comparing deformations generated under realistic muscle forces (from 1 to 4 N), with lingual deformations measured on patients. Figure 2b plots tongue deformations under the activation of styloglossus muscle (recruited to pull tongue tissues towards velar region).

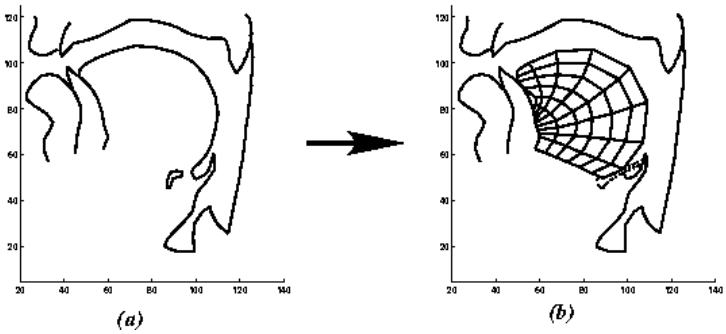

Fig. 2a: Definition of the 2D FE tongue mesh from a radiography

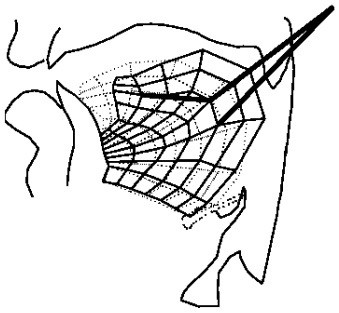

Fig. 2b: Styloglossus muscle action

4. VELUM MODEL

A continuous model of the velum was elaborated and defined with the Finite Element Method. The numerical model assumes no displacement in the transverse direction (*the plane strain hypothesis*) and small deformations. Again, for quasi-incompressibility reasons, a value close to 0.5 was chosen for the Poisson ratio. A 10 kPa value was taken for the Young modulus, which seems consistent with values reported for tongue [5] and vocal folds [6]. The geometry of the model was extracted from a single midsagittal radiography of a patient (figure 3).

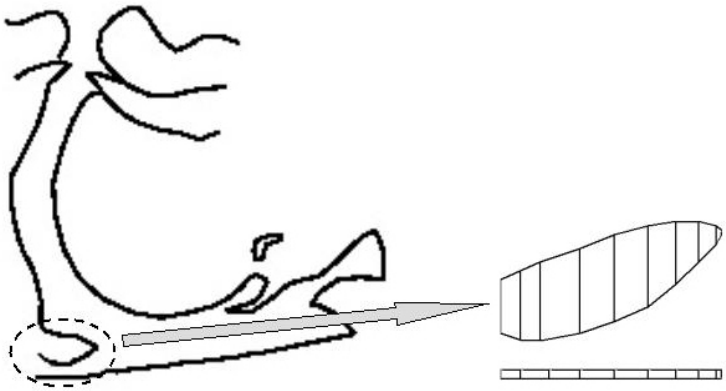

Fig. 3: Midsagittal view of the upper airways (left) and FE model of the velum (right).

The upper part of the model represents the velar tissues. Geometrical values were coherent with values reported in the literature [7] [8]: thickness of the velum varies from one extremity to the other (with a mean of 5-mm) while the total length is around 30-mm.

For the current simulations, only the velum deformations were taken into account, as the pharyngeal walls (the lower part of the model on figure 3) were supposed to be rigid during FE computations. The two points located onto the left part of the velum were

also considered as fixed in order to model the velar attachment to the hard palate. Last, simulations are limited to the midsagittal plane, but for the computation of pressure forces, a 30-mm value was taken for the velar thickness in the frontal plane.

5. FLUID MECHANICAL ASPECTS

From a fluid mechanical point of view, the partial or the total collapse of the upper airway, as observed during sleep hypo-apnea or apnea, can be understood as a spectacular example of fluid-walls interaction. While the most important parameters influencing this effect *in vivo* are well known, this phenomenon is still difficult to model and thus to predict. To illustrate our approach, figure 4 presents in a simple way a constriction inside the upper airways.

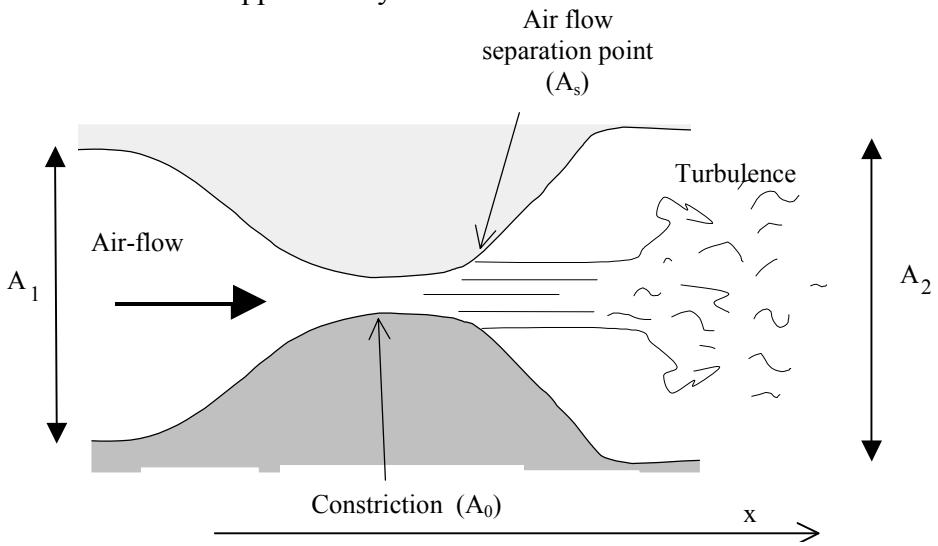

Fig. 4: Schematic Illustration for airflow inside the constriction.

An exact analytical solution for the flow through such a constriction is not available. Further, full numerical simulations of the unsteady three-dimensional flow through a deformable structure are still, at present time, impossible even using the recent numerical codes and using powerful computers [9]. For these reasons, and also because the aim of this paper is to provide a qualitative description of a sleep apnea, we use in the following a simplified flow theory based on the following assumptions:

- As the airflow velocity in the upper airways is, in general, much smaller than the speed of sound (low Mach number flow), it can be assumed that the flow is locally incompressible.
- It can be reasonably assumed that the time needed for the constriction to collapse (of order of a second) is large compared with typical flow convection times (the time needed for the flow to pass the constriction is of order of a few milliseconds). Therefore, it will be assumed that the flow is quasi-steady (low Strouhal number flow).

The principle of mass-conservation thus yields the following relationship:

$$\Phi = \text{constant} \qquad (1)$$

where $\Phi = v.A$ is the volume flow velocity, v and A are respectively the (local) flow velocity and upper-airways area. As a third and last assumption, we now consider that all viscous effects can be neglected. This assumption can be rationalized partially by considering that typical Reynolds numbers involved are of order of 1000 and thus that viscous forces are negligible compared with convective ones. This leads to the well-

known one-dimensional equation (Bernoulli law):

$$p + \frac{1}{2}\rho v^2 = \text{constant} \qquad (2)$$

where p is the local pressure and ρ the (constant) air density

Equations (1) and (2) must be corrected in order to take into account a spectacular viscous effect: flow separation. Indeed, it is expected that the strongest pressure losses are due to the phenomenon of flow separation at the outlet of the constriction. This phenomenon is due to the presence of a strong adverse pressure gradient that causes the flow to decelerate so rapidly that it separates from the walls to form a free jet. Very strong pressure losses, due to the appearance of turbulence downstream of the constriction, are associated with flow separation. As a matter of fact, the pressure recovery past the flow separation point is so small that it can in general be neglected. In the following, we consider that the flow separates from the walls of the constriction at the point where the area reaches 1.2 times the minimum area A_0 (see figure 4). This approximated value was empirically proposed and constitutes an acceptable approximation of the phenomena [10].
To summarize, for a given pressure drop (*p1-p2*), and for a given geometry of the constriction, the volume flow velocity Φ is:

$$\phi = A_s\sqrt{\frac{2(p_1 - p_2)}{\rho}} = 1.2A_0\sqrt{\frac{2(p_1 - p_2)}{\rho}} \qquad (3)$$

and the pressure distribution *p(x)* within the constriction is predicted by:

$$p(x) = p_1 + \frac{1}{2}\rho\phi^2\left(\frac{1}{A_1^2} - \frac{1}{A(x)^2}\right) \qquad (4)$$

where *A(x)* is the transversal area at the *x* abscissa (figure 4).

Thus, the force exerted by the airflow onto the walls of the constriction can be computed by integrating the pressure along the *x* axis up to the flow separation point. This force induces a deformation of the upper airways soft tissues, thus modifying the airways geometry, and therefore changing the pressure distribution along the airways. Next part describes the model that was developed to simulate this phenomenon.

6. COUPLING AIR PRESSURE FORCES AND THE VELUM MODEL

The soft tissues that constitute the tongue, the velum and the pharyngeal walls are partly responsible for the SAS as their deformations can lead to a partial (hypo-apnea) or total (apnea) closure of the upper airways. In a first step, we only focused on the velo-pharyngeal region, at the intersection between the nasal and the oral cavity. An iterative process governed the coupling between the airflow pressure forces computation and the deformations of the FE model of the velum. An adaptive Runge Kutta algorithm was used to solve the dynamical equations that govern the deformations of the model and its coupling with the airflow. In order to simulate a respiratory cycle, the pressure drop (*p1-p2*) was approximated by a sinusoid: $p_1\text{-}p_2 = p_{max}*\sin(4*\pi*t)$. Figure 5 shows simulations of the airflow / velum interactions for a half-period pressure drop command with an 800 Pa maximal value. A clear reduction of the constriction can be observed, thus simulating a hypo-apnea [11].

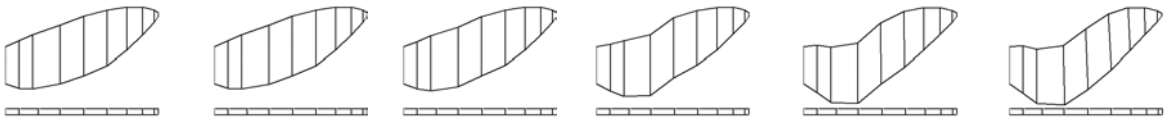

Figure 5: Deformations of the model coupled with airflow: from initial (left) to final (right) positions.

7. DISCUSSION

Despite the limitations of our modeling hypotheses, preliminary interesting simulations were carried out. Indeed, the airflow model coupled with a 2D Finite Element model of the velum provided a reduction of the constriction with the increase of the pressure drop. This result is well known by the clinicians and is described as the airflow limitation phenomenon (also called the hypo-apnea phenomenon). It is also interesting to note that an increase of the velum stiffness (modeled with an increase of the Young modulus value) or an increase in the size of the velo-pharyngeal constriction both tend to limit the hypo-apnea syndrome. Those results are consistent with some surgery techniques that try to modify mechanical properties of the velum (by burning tissues, thus increasing their stiffness) or try to have a more global and progressive action on the entire upper airways (in order to increase the size of the upper-airway constriction).

8. REFERENCES

1. Pépin J.L., Lévy P., Veale D. & Ferreti G., Evaluation of the upper airway in sleep apnea syndrome. Sleep, 1992, Vol.15, S50-S55.
2. Auregan Y. & Meslier N., Modélisation des apnées obstructives du sommeil. C.R. Acad. Sci. Paris, 1993, Vol. 316, 1529-1534.
3. Smith P.L., Wise R.A., Gold A.R., Schwarts A.R. & Permutt S., Upper airway pressure-flow relationship in obstructive slepp apnea. J. Appl. physiol., 1988, Vol. 64, 789, 795.
4. Fodil R., Ribreau C., Louis B., Lofaso F., Isabey D., Interaction between steady flow and individualised compliant segments: application to upper airways. Medical & Biological Engineering & Computing, 1997.
5. Payan Y. & Perrier P., Synthesis of V-V Sequences with a 2D Biomechanical Tongue Model Controlled by the Equilibrium Point Hypothesis. Speech Communication, 1997, Vol. 22 (2-3), 185-205.
6. Min Y., Titze I. & Alipour F., Stress-Strain Response of the Human Vocal Ligament, NCVS Status and Progress Report, 1994, Vol. 7, 131-137.
7. Pépin J.L, Ferretti G., Veale D., Romand P., Coulomb M., Brambilla C., Levy P.A., Somnofluoroscopy, computerised tomography and cephalometry in the assesment of the airway in obstructive sleep apnoea, Thorax, 1992, 47, 150-156.
8. Trudo F.J., Gefter W.B, Welch K.C., Gupta K.B., Maislin G., Schwab R.J., « State-related changes in upper airway caliber and surrounding soft-tissue structures in normal subjects », Am. J. Respir. Care Med., 1998, Vol. 158, 1259-1270.
9. Hirsch C., Numerical computation of internal and external flows, John Wiley & sons Eds, 1994.
10 Pelorson X., Hirschberg A., Wijnands A.P.J., Bailliet H., Description of the flow through the vocal cords during phonation, Acta Acustica, 1995, Vol. 3, 191-202.
11 Rapoport D.M., Methods to stabilize the Upper Airway using Positive Pressure, Sleep, 1996, Vol. 19, 123-130.