FSI Analysis of a Healthy and a Stenotic Human Trachea Under Impedance-Based Boundary Conditions

In this work, a fluid-solid interaction (FSI) analysis of a healthy and a stenotic human trachea was studied to evaluate flow patterns, wall stresses, and deformations under physiological and pathological conditions. The two analyzed tracheal geometries, which include the first bifurcation after the carina, were obtained from computed tomography images of healthy and diseased patients, respectively. A finite element-based commercial software code was used to perform the simulations. The tracheal wall was modeled as a fiber reinforced hyperelastic solid material in which the anisotropy due to the orientation of the fibers was introduced. Impedance-based pressure waveforms were computed using a method developed for the cardiovascular system, where the resistance of the respiratory system was calculated taking into account the entire bronchial tree, modeled as binary fractal network. Intratracheal flow patterns and tracheal wall deformation were analyzed under different scenarios. The simulations show the possibility of predicting, with FSI computations, flow and wall behavior for healthy and pathological tracheas. The computational modeling procedure presented herein can be a useful tool capable of evaluating quantities that cannot be assessed in vivo, such as wall stresses, pressure drop, and flow patterns, and to derive parameters that could help clinical decisions and improve surgical outcomes. [DOI: 10.1115/1.4003130]
1 Introduction

The trachea begins immediately below the larynx and runs down the center of the front part of the neck, ending behind the upper part of the sternum. Here, it bifurcates in the left and right main bronchi that enter the lung cavities. The trachea forms the trunk of an upside-down tree and is flexible, so that the head and neck may twist and bend during the process of breathing [1]. The main components that constitute the trachea are fibrous and elastic tissues and smooth muscle, which runs longitudinally and posteriorly to the trachea, with about 20 rings of cartilage, which helps the trachea to keep it open during extreme movement of the neck [2]. The main role of the tracheal cartilaginous structures is to keep the windpipe open despite the interthoracic pressure during breathing, sneezing, or coughing [3]. Under these conditions, among others, the smooth muscle collapses to regulate the air flow by dynamically changing the tracheal diameter. This contraction and the transmural pressure generate bending and tensile stresses in the cartilage.

Although a clear understanding of the respiration process has not yet been reached and the influence of the implantation of a prosthesis is very important from a medical point of view as well as challenging from the computational aspect, a few studies have analyzed the behavior of the tracheal walls under different ventilation conditions. Most numerical analyses of the respiratory system have been focused purely on computational fluid dynamics (CFD), neglecting the interaction with the wall airways. In particular, most works dealt with the airflow patterns using idealized [4,5] or approximated airways geometries [6–8] while only few studies were based on accurate airway geometries developed from computed tomography (CT) or magnetic resonance images (MRIs) [9–14]. While these studies, following the CFD approach, failed to incorporate constitutive material modeling of the tracheal wall [6–8,15–17], recent studies in lower airway geometries have successfully presented FSI results using simplified models/geometries for single bifurcations [18–21]. Wall and Rabczuk [22] analyzed the behavior of a CT-based respiratory system through a FSI analysis during breathing and mechanical ventilation even using a simplified model for the composing material. Moreover, Koombua and Pidaparti [23], following a FSI approach, studied the stress distributions in a two-bifurcation tracheal model and compared the relative differences between an isotropic and an orthotropic material model of the airways. Regarding the constitutive behavior of the tracheal walls, there is in fact a large dispersion of the mechanical properties of the different composing tissues and only a few studies have analyzed their mechanical behavior for humans [24–26]. Abnormal airways deformation and alteration of airway mechanical properties were previously reported as a potential cause of tissue damage and associated to chronic complications, as reported by Spitzer et al. [27]. In most of the works on this topic, the isolated tracheal cartilage was considered as a linearly elastic isotropic material [28]. In many of the previous studies, the human tracheal smooth muscle dealt with its plasticity, stiffness, and extensibility and the influence of the temperature on force-velocity relationships. Begis et al. [29] developed a structural finite element model of an adult human tracheal ring, accounting for its morphometric features and mechanical properties. More recently, Costantino et al. [28] analyzed the collapsibility of the trachea under different pressure conditions by comparing the numerical results with experimental tests. Regarding tracheal pathologies, Brousse et al. [30] provided a CFD numerical model of a healthy trachea without any bifurcation, in which different stages of artificial stenosis were imposed, with the aim to establish a relationship between local pressure drop and velocity depending on the degree of stenosis. Sera et al. [31] investigated the mechanism of wheeze generation with experimental tests conducted on a rigid and an extensible realistic CT-based tracheostomotic model. Cebal and Summers [32] showed how virtual bronchoscopy can be used to perform aerodynamic calculations in anatomically realistic models.

For both CFD and FSI simulations, the choice of the boundary conditions is critical. Most studies used velocity or flow conditions at the entrance of the trachea as inlet boundary conditions and usually utilize approximations such as time dependent resistance [8,22], zero-pressure [9–11], or simply outflow conditions [8,9,30] as outlet boundary conditions. Recently, Wall and coworkers proposed a structured tree for modeling the human lungs and analyzing the respiration and mechanical ventilation under interference conditions [33–35]. Finally, Elad et al. [36] proposed a nonlinear lumped-parameter model to study the dependency of airflow distribution in asymmetric bronchial bifurcations on structural and physiological parameters. In this study, we analyzed the tracheal wall stresses and the flow patterns through a study of healthy and stenotic human tracheas during inhalation using a FSI approach. In this analysis, we gave special emphasis to the solid domain in order to predict stresses and displacements under physiological conditions. The aim of the this work is to create a functional method to simulate and classify the tracheal wall stress and strain. Understanding the mechanical behavior of the central airways may allow for instance the investigation of their response to the mechanical ventilatio-associated pressure loads, which is important for clinical applications and for stenting technique. In this sense, our study can be considered as a step toward building a framework that could aide surgical decisions in future. Moreover, we tackle the challenging aspect of the physiological boundary conditions by modeling the resistance of the entire respiratory system through impedance-based boundary conditions. This approach allows the computation of physiological pressures that are not measurable in vivo starting from patient speciﬁc spirometry. Computed pressures waveforms are essential for the evaluation of stresses and strains during breathing and coughing, especially for diseased and stented tracheas. The method followed, developed by Oulsen [37] and later extended by Steele et al. [38], has been applied to the cardiovascular systems [39–43] and to the pulmonary system [33–35].

2 Materials and Methods

2.1 Fluid-Airflow. Thoracic CT scans were taken from a 70 year old healthy and a 56 year old diseased man and the images were then segmented and post-processed to create the fluid model. An initial graphics exchange specification (IGES) file of the computational fluid model of the internal tracheal cavity was created from these segmented images. The model was then meshed with unstructured tetrahedral-based elements using the commercial software FEMAP (Siemens PLM Software, Plano, TX) (see Fig. 1). The fluid was assumed as Newtonian \( \rho_f = 1.225 \text{kg/m}^3, \mu = 1.83 \times 10^{-5} \text{kg/m s} \) and incompressible under unsteady flow.
Flow was assumed turbulent for the analyzed cases since the Reynolds numbers, based on the median tracheal section, at peak velocity, were in the turbulent regime ($Re_{\text{healthy}} = 30,000$, $Re_{\text{stenosis}} = 10,000$). To ensure that the results were insensitive to the computational grid size, a sensitivity study was carried out. Four different meshes of about 100,000, 200,000, 250,000, and 300,000 elements were tested for the healthy and stenotic tracheas, studying the inspiratory peak flow to ensure that the results at the highest Reynolds number were grid independent.

Comparison between four grid sizes revealed that the velocity profiles for the grid of 200,000, 250,000, and 300,000 elements were found almost coincident (see Fig. 2, section 1). However, the velocity profile near the wall was found to be smoother for these three meshes with respect to the coarser mesh. This indicates that the higher number of grid cells within the wall region can capture the flow field in more detail than that for the coarser grid size. The difference between coarser and finer grids is also noticeable as the air flows downstream in the left bronchus (see Fig. 2, section 2).

As finer grid increases the computing time considerably, we selected the grids with a total number of tetrahedral elements of about 200,000 for further simulations. The velocity profiles obtained from the grids of about 200,000, 250,000, and 300,000 show a difference of only 2–3%. Due to the turbulent nature of the considered tracheal flow, which is basically transitional rather than fully turbulent, and taking into account the limitation of the use of the $K$-$\omega$ model for its modeling, as documented in Refs. [44,45] among others, the standard $K$-$\omega$ model was used to modify the air viscosity. However, it is not clear if Reynolds averaged Navier-Stokes (RANS)-based turbulence models are a good choice or not. Due to the recirculatory flow and turbulent eddies caused by the laryngeal jet, Gemci et al. [47], for instance, used a large eddy simulation (LES) turbulence model in their study. In particular, in the Navier–Stokes equations, the viscosity $\mu$ is substituted by $\mu = \mu_0 + \mu_t$, where $\mu_t$ is the turbulent viscosity, which is computed by Eq. (1),

$$\mu_t = \alpha \rho \frac{K}{\omega} \quad (1)$$

where $\alpha$ is a constant of the model, expressed as a function of the
Reynolds number [46]. The variable \( \omega \) is the specific dissipation rate of turbulence. This is related to the so-called kinetic energy and rate of dissipation of the turbulence (\( K \) and \( \varepsilon \), respectively) and is defined as follows:

\[
\omega \sim \frac{\varepsilon}{K}
\]

(2)

with

\[
K = \frac{1}{2} \mathbf{v}' \cdot \mathbf{v}', \quad \varepsilon = \frac{\mu}{\rho F} (\nabla \mathbf{v}') \otimes (\nabla \mathbf{v}')
\]

(3)

where \( \mathbf{v}' \) is the fluctuating velocity, \( K \) and \( \omega \) are governed by Eqs. (4) and (5),

\[
\frac{\partial (\rho \mathbf{v})}{\partial t} + \nabla \cdot (\rho \mathbf{v} K - \mathbf{q}_k) = \gamma^0 G_K
\]

(4)

\[
\frac{\partial (\rho \omega)}{\partial t} + \nabla \cdot (\rho \omega \mathbf{v} - \mathbf{q}_\omega) = \gamma^0 G_\omega
\]

(5)

where \( \alpha \) is 0 for two- and three-dimensional flows and 1 for axisymmetric flows. Other corresponding terms are defined as follows:

\[
\mathbf{q}_k = \left( \frac{\mu_0 + \mu_k}{\sigma_k} \right) \nabla K
\]

(6)

\[
\mathbf{q}_\omega = \left( \frac{\mu_0 + \mu_\omega}{\sigma_\omega} \right) \nabla \omega
\]

(7)

\[
G_K = 2\mu D^2 - \beta_K K \omega + B
\]

(8)

\[
G_\omega = \frac{\omega}{K} (2\alpha_\omega \mu D^2 - \beta_\omega \rho K \omega + \beta_\omega \beta_B)
\]

(9)

where \( \alpha, \sigma_\omega, \beta_K, \beta_\omega, \sigma_K, \sigma_\omega, \alpha_\omega, \) and \( \beta_\omega \) are model constants. These values, input to the finite element solver at the beginning of the simulation, are displayed in Table 1. A complete description of this turbulence model may be found in Ref. [46].

### 2.2 Solid-Tracheal Wall

In order to determine the real geometry of the trachea and to distinguish between the muscular membrane and the cartilage rings, we proceeded to a nonautomated segmentation of the CT scans using MIMICS\textsuperscript{®}. Each tracheal constitutive part could be detected through its different density. Finally, with the commercial software ABAQUS\textsuperscript{®}, a full hexahedral mesh of about 60,000 elements for the healthy trachea and of about 90,000 elements for the stenotic trachea was generated. In Fig. 1, these grids are shown. The stenosis, which is a rigid fibrous cap surrounding the internal tracheal wall, due to its irregular and asymmetric geometry was meshed with about 40,000 tetrahedral elements. The properties of the different tissues of the trachea were analyzed and numerically modeled through different experimental tests described in previous studies [24,48]. Here, we will provide only a short explanation. Human tracheas were obtained through the autopsy from two subjects (aged 79–82 years) and subjected to different tensile tests to obtain the material models. The cartilaginous rings were modeled as isotropic material [24,48]. The muscular membrane presented two orthogonal families of smooth muscle cells, one running mainly longitudinally and the other transversely. Therefore, for its behavior, the anisotropy coming from the fiber orientations was introduced in the material model. For cartilage, since there is no preferential orientation, a neo-Hookean model with strain density energy function (SEDF), \( \Psi = C_1 (I_1 - 3) \), was used to fit the experimental results. Regarding the smooth muscle, and taking into account that the histology showed two orthogonal fiber families, the Holzapfel SEDF [49] for two families of fibers was used,

\[
\Psi = C_1 (I_1 - 3) + \frac{K_1}{2K_2} [\exp(K_2(I_{41} - 1)^2) - 1] + \frac{K_1}{2K_4} [\exp(K_4(I_{42} - 1)^2) - 1] + \frac{1}{K} (J - 1)^2
\]

where \( C_1 \) is the material constant related to the ground substance, \( K_i > 0 \) are the parameters that identify the exponential behavior due to the presence of two families of fibers, and \( D \) is identified with the tissue incompressibility volumetric modulus. The invariants \( I_j \) are defined as

\[
I_1 = \text{tr} \bar{C}, \quad I_2 = \frac{1}{2} (\text{tr} \bar{C}^2 - \text{tr}(\bar{C})^2)
\]

\[
I_{41} = \bar{a}^0 \cdot \bar{C} a^0, \quad I_{42} = \bar{b}^0 \cdot \bar{C} b^0
\]

where \( a^0 \) is a unitary vector defining the orientation of the first family of fibers, \( b^0 \) is the direction of the second family both in the reference configurations, and \( \bar{C} \) is the modified right Green strain tensor defined as \( \bar{C} = J^{1/3} C \) being \( C = F F^T \). In Table 2, a summary of the material constants used for the different tissues is shown. For further explanations, see Refs. [24,48]. Not much information is available about the constitutive properties of the tracheal stenotic tissue. For this reason, this fibrous tissue was modeled as a hyperelastic material, in which we assumed an increased stiffness of about 2 times with respect to the muscular membrane (see Table 2). In Fig. 3, the different tracheal constitutive tissues are separately shown for the healthy (a) and stenotic (b) models.

### 2.3 Numerical Method and Boundary Conditions

To facilitate the fluid dynamic model, in order to apply uniform velocity field as inflow boundary condition, a five-inlet extension was added to each model. In this way, a fully developed airflow is assessed through the trachea. Moreover, to take into account the effect of the laryngeal jet on the tracheal flow [50–52], the tra-
Fig. 3 Finite element model of the (a) healthy and (b) stenotic tracheas with their respective constitutive parts separately plotted (A denotes anterior part and P denotes posterior part).
ated, two pressure waveforms are computed using the method developed by Olufsen et al. [37,53]. These pressures are later applied at the outlets of each FSI model.

The bronchial network was modeled as a binary asymmetric-structured tree in which each branch was approximated by a straight compliant segment. The airways network resulted in a series of bifurcations composed by a series of parent and daughter bronchi, as shown in Fig. 4(a). Each parent bronchus bifurcates in two daughter bronchi following a scaling guided by the asymmetry factors \( \alpha \) and \( \beta \) of the root parent \( r_{\text{root}} \) according to Eqs. (12) and (13),

\[
\begin{align*}
\alpha &= (1+\gamma^{2\xi}r_{\text{root}}) / r_{\text{root}} \quad 0 \leq i \leq j \\
\beta &= r_{\text{pa}} / r_{\text{pa}}_i = \beta(r_{\text{pa}})_i
\end{align*}
\]

The structured tree continues branching until the radius of any bronchial segment is less than given minimum values \( r_{\text{min}} \) as for instance the alveolar radius (\( \approx 10-100 \) \( \mu \)m) where we assumed zero impedance. Although we know that during inspiration and expiration, the alveolar pressure sinusoidally varies between \(-1 \) cm H\(_2\)O and \(+1 \) cm H\(_2\)O [1], the assumption of zero impedance at the alveolar radius is clearly more appropriate than the assumption of zero-pressure applied in other works at intermediate generations [7,9–12].

In this study, we followed the approach used by Steele et al. [38], dividing the entire bronchial network in three different levels, to mimic the respiratory system structure. For each level, the parameters describing the fractal tree were varied. The minimum radius was set at the alveolar level (10 \( \mu \)m) where, as discussed, we assumed zero impedance. In the Table 3, the starting radii of the considered trachea models used as the input of the fractal tree scaling are shown. The asymmetry and area ratios of the bronchial network [57] were defined as [37]

\[
\eta = \frac{(r_{\text{pa}})_i + (r_{\text{pa}})_i}{(r_{\text{pa}})_i} \quad \gamma = \frac{(r_{\text{pa}})_i}{(r_{\text{pa}})_i}^2
\]

where \( r_{\text{pa}} \) is the radius of the parent bronchi, while \( \eta \) is the area ratio, and \( \gamma \) is the asymmetry ratio, are related to each other through the expression [37]

\[
\eta = \frac{1+\xi}{\gamma^{2\xi}}
\]

\( \gamma \) is known also as asymmetry index and describes the relative relationship between the daughter bronchi. The exponent \( \xi \) is known in the cardiovascular literature as the power exponent and comes from the power law [59].

\[
(r_{\text{pa}})_i = (r_{\text{pa}})_i + (r_{\text{pa}})_i
\]

This relation is valid for a range of flows [37]. Several studies show that \( \xi \) varies between 2 and 3 [38,59,60]. The power exponent \( \xi=3 \) is optimal for laminar flows while \( \xi=2.33 \) is optimal for turbulent flows [37].

Assuming that \( r_{\text{pa}} \leq r_{\text{pa}}_i \) [57], \( \gamma \) is between 0 and 1. The value of \( \gamma \) widely varies from humans to dogs and rodents as documented by Latourelle et al. [57]. Based on the morphometry of the models described in Refs. [4,54,61], in this work, we chose to vary the value of asymmetry index with \( \gamma=0.85, 0.9, \) and 0.95 (see Table 4). The value of \( \gamma \) in Eq. (15) describes the branching relationship across bifurcations between the radius of the parent bronchi and the radii of the daughter bronchi \( r_{\text{pa}} \) for \( i=1,2 \). Using the mentioned values of \( \gamma \) and the power-exponent \( \xi=2.33 \), we calculated the values of \( \eta \) for each respective level (see Table 4).

Finally, the length of a given branch \( L \) can be related to the radius \( r_{\text{pa}} \) of each branch segment through the parameter \( l_{\gamma} = L / r_{\text{pa}} \) [38,53]. Morphometric and anatomic data for humans and rodents are detailed in Refs. [4,6,55,58,62]; these works showed that this parameter rapidly varies from the trachea to the bronchioli, until the alveolar level (from around 12 to 2 [1,4]). Based on these studies, we finally fixed a constant average value of \( l_{\gamma} = 6 \) [1,4,54,61].

### 4 Impedance Recursive Computation

Impedance was computed in a recursive manner starting from the terminal branch [38,53] of the structured tree and was used as the outlet boundary condition for the tracheal left and right bronchi. The details of the recursive calculation are given elsewhere [37,38,53]. Input impedance was evaluated at the beginning of each airway daughter \( z=0 \) as a function of the impedance at the end of the airway daughter \( z=L \):

\[
Z(0,\omega) = \frac{Z(0,\omega)}{\cos(\omega L/c) + ig(\omega L/c)}
\]

where \( L \) is the bronchial length, \( c = \sqrt{\gamma/1(1-F)}/(\gamma C) \) is the wave propagation velocity, \( g = \sqrt{\gamma A_0 K/p_f} \), and

\[
Z(0,\omega) = \lim Z(0,\omega) = \frac{8\mu_{0} F}{\pi r_{0}^2} + Z(0,0), \quad F_j = \frac{Z(0,0)}{Z(0,0)}
\]

\( F_j(x) \) and \( J_j(x) \) being the zeroth and first order Bessel functions with \( \omega = \pi r_{0}^2 \) \( r_{0}^2 = r_{0}^2 \) and \( r_{0}^2 \) the bronchus radius corresponding to section \( A_0 \), while \( E \) is the Young modulus, whose value is 3.33 MPa according to the experimental study of Trabesi et al. [48]. In Fig. 4, the spirometry (b) with the corresponding left and right bronchi pressure waveforms (c) used in the computations are sketched. It has to be noted that in this figure, inspiratory flows are assumed as positive while expiratory flows are assumed as negative.

### 5 Results and Discussion

The airflow inside the trachea is analyzed at peak inspiration flow through bidimensional streamlines projected on selected tracheal longitudinal sections. Velocity distributions are plotted for each section to give a complete description of the flow patterns in the trachea during inhalation. The air flow, which is oscillatory, is governed by the Womersley number. This characteristic number represents a nondimensional frequency in oscillatory flows defined by Eq. (20),

\[
w = \frac{D}{\sqrt{2\pi/v}}
\]

with the forced breathing frequency \( f \), the kinematic air viscosity \( \nu \), and the diameter \( D \) of the trachea. The Womersley number for the two patient-specific models is showed in Table 5. Due to the high flow rates obtained from the patient-specific spirometry (these are forced breathing rather than normal breathing), \( w \) is higher than what is reported in other studies [6,13,61]. Noteworthy is that although the two spirometries have different frequencies, \( w \) was nearly identical for both models. With regard to the airflow mechanics, wall stresses and displacements are analyzed at peak inspiration to show the function of the different tracheal tissues and the overall behavior of the trachea during inspiration.

#### 5.1 Flow Patterns in the Healthy Trachea

The flow inside the healthy trachea is shown during inhalation with its respective pressure distribution in Fig. 5. During the inspiration, the trachea shows a strong primary axial flow resulting in a flattened velocity profile (in the order of 10 m/s) due to the turbulence (see Fig.
Fig. 4 (a) Structured tree (adapted from Olufsen [37]), (b) airflow, (c) inlet velocity, and (d) computed pressure waveforms (healthy trachea up, stenotic trachea bottom)
Up to the bifurcation the flow is nearly parabolic, while after the flow divider, the stream has split as the fluid moves toward the daughter tubes. The high axial flow intensity (8 m/s) reduces the effect of the geometrical bending curvature so that a fully developed Dean-flow pattern is not present, as documented in Refs. [9–11]. Also, and as found in other studies [8,22,63,64], the right main bronchus shows a typical M-shaped velocity profile, which is not fully developed probably due to the limited length of bronchus and its particular geometry (Fig. 6(b), section 3); this typical distribution is normally associated to a high Reynolds number as documented in Ref. [22] among others. For the same reason, in the left main bronchus the air flow possesses a high axial component, whose velocity is in the order of 6 m/s. Also here the typical Dean-flow pattern, found by Refs. [11,22,65], cannot be observed (see Fig. 6(a), section 3). The reason of these differences on the secondary flows can be explained considering the higher inlet velocity we used in comparison with other works. For the present computations we used in fact a forced respiration extracted from a patient specific spirometry, while other studies [9–11] on CFD with rigid wall and Wall and Rabczuk [22] on the FSI approach used velocity data extracted from normal breathing. Finally, the velocity distribution in the left main bronchus is slightly skewed toward the outer wall (see Fig. 6(b), section 3).

5.2 Wall Stresses and Deformations in the Healthy Trachea. Results corresponding to the wall deformation during the different phases of the inspiration are shown in Figs. 7(a) and 7(b), corresponding to the maximum logarithmic strain in the circumferential direction. This illustrates the radial deformation of the tracheal walls as a function of their initial configuration. The highest deformation (10% at \( \tau = 2 \) s) appeared in the muscular membrane, which increases the section of the trachea along its longitudinal axis during inflation and decreases it during expiration contributing to the pressure-balance inside the lungs, as documented in Ref. [2]. Besides, the deformation of tracheal rings was smaller than that of the muscular membrane. In Fig. 7(c), the amplified deformed shape of the tracheal rings at inspiration is shown. The cartilage rings opening, as shown in the figure, allows muscular membrane dilatation. In particular, in the figure, an increase of the cartilage expansion during inspiration from 0 s to 2 s and a reduction of this from 2 s to 4 s can be seen, at the end of which the expiratory process begins. This expansion is basically due to the positive pulmonary pressure [1,2]. In Fig. 9, the principal stresses are shown. The maximum registered value is 3.3 KPa. At inspiration, the muscular membrane is in tension. The muscular deflection generates tensile stresses in the cartilage and modulates the airway diameter as reported also in Ref. [3]. If only the cartilaginous rings of the trachea are shown, it can be seen how they absorbed most of the load, as shown in Fig. 7.

5.3 Flow Patterns in the Stenotic Trachea. In the diseased trachea, strong modifications of the local airway flow patterns were found due to the elevated pressure drop caused by the stenosis, as shown in Fig. 5(b), where the pressure distribution of the two analyzed models is compared. The pressure drop between the trachea entrance and the stenosis is around 8 cm H2O, which is higher with respect to values found in literature. This is basically due to the stenosis degree of the considered model being more severe than that in other works [30]. Another reason could be the nature of the computation. In the present study, the tracheal wall are in fact extensible while other works used a CFD approach. These different approaches could lead to different fluid field, as demonstrated by Ref. [31]. The deformation of the trachea at the stenotic constriction caused in fact an increase of the longitudinal vortex intensity, which progressively blocks the air flowing in the trachea, generating an additional increase of the pressure drop before and after the constriction (see Fig. 5(a)). The pressure distribution of Fig. 5(b) shows almost the same pressure values on both bronchi. This is basically due to the computed pressure waveforms (see Fig. 4), which are very similar for the left and the right bronchi. This can be clearly seen also in the healthy trachea pressure distribution (see Fig. 5(a)). Furthermore, the considered models only show one bifurcation. The flow in the daughter tubes is influenced by the absence of more bronchial generations while the pressure distribution is surely affected by this fact. For these reasons, perhaps the impedance approach could have a stronger impact than what we found for a one-generation model in more complex models considering many bronchial generations with different radii.

During inhalation, the velocity profile before the stenosis is almost axial (around 10 m/s) while the highly reduced geometrical cross section creates a dead fluid zone before the bifurcation (see Fig. 5) with a longitudinal vortex that influences the flow distribution after the flow divider. This velocity field is in agreement with those found by Brouns et al. [30]. The asymmetry of the stenosis causes the already mentioned high velocity jet that crosses the constriction. The flow distribution on the right and left main bronchi is shown in Fig. 6(c) on sections 5 and 6. Different from that reported by Sera et al. [31], the secondary flow is not present probably because of the higher axial velocity entering both bronchi (see Fig. 6(d) on sections 5 and 6) with respect to their work.

5.4 Wall Stresses and Deformations in the Stenotic Trachea. The wall deformation of the different constitutive parts of the stenotic trachea during inspiration is shown in Figs. 8(a)

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### Table 3 Radii of the CT-based trachea model used as input of the fractal tree and impedance recursive computation

<table>
<thead>
<tr>
<th>Trachea</th>
<th>Tracheal diameter (m)</th>
<th>Right bronch. diameter (m)</th>
<th>Left bronch. diameter (m)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy</td>
<td>0.023</td>
<td>0.016</td>
<td>0.0125</td>
</tr>
<tr>
<td>Stenotic</td>
<td>0.018</td>
<td>0.015</td>
<td>0.0095</td>
</tr>
</tbody>
</table>

### Table 4 Parameters used to describe the structured tree. The three-tiered tree is divided into three levels as a function of the bronchial radius. Scaling parameters are varied along the tree.

<table>
<thead>
<tr>
<th>Level</th>
<th>Radius (( \mu )m)</th>
<th>( \alpha )</th>
<th>( \beta )</th>
<th>( \xi )</th>
<th>( \gamma )</th>
<th>( \eta )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trachea-bronchi</td>
<td>200 &lt; ( r &lt; 500 )</td>
<td>0.772</td>
<td>0.7116</td>
<td>2.33</td>
<td>0.85</td>
<td>1.10</td>
</tr>
<tr>
<td>Bronchi-bronchi</td>
<td>50 &lt; ( r &lt; 200 )</td>
<td>0.7619</td>
<td>0.7228</td>
<td>2.33</td>
<td>0.9</td>
<td>1.102</td>
</tr>
<tr>
<td>Bronchioli-alveoli</td>
<td>( r &lt; 50 )</td>
<td>0.7521</td>
<td>0.7331</td>
<td>2.33</td>
<td>0.95</td>
<td>1.1031</td>
</tr>
</tbody>
</table>

### Table 5 Forced breathing frequencies and corresponding Reynolds and Womersley numbers

<table>
<thead>
<tr>
<th>Trachea</th>
<th>Frequency (Hz)</th>
<th>Re</th>
<th>( w )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy</td>
<td>0.097</td>
<td>30,000</td>
<td>2.32</td>
</tr>
<tr>
<td>Stenotic</td>
<td>0.159</td>
<td>10,000</td>
<td>2.33</td>
</tr>
</tbody>
</table>
Fig. 5  Projected 2D streamlines (in m/s) on frontal and lateral sections of the (a) healthy and (b) stenotic tracheas with respective pressure distributions (in cm H₂O) at peak flow during inhalation.

Fig. 6  Projected 2D streamlines (in m/s) at selected longitudinal sections of the (a) healthy and (c) stenotic tracheas with (b) and (d) respective velocity distributions at peak flow during inhalation.

Fig. 7  Logarithmic circumferential strain of the (a) complete model and of the (b) cartilage rings of the healthy trachea at selected time points during inspiration. In (c), the deformed shape of the cartilage rings during inspiration is shown.

Fig. 8  Logarithmic circumferential strain (a) of the complete model and (b) of the cartilage rings of the stenotic trachea at selected time points during inspiration. In (c), the deformed shape of the cartilage rings during inspiration is shown.
and 8(b). As for the healthy case, the muscular membrane expands, increasing the section of the trachea (in this case the maximum circumferential strain was around 37%) during inhalation. At the bottom of Fig. 8(c), the amplified deformed shape of the stenotic tracheal rings at inspiration is shown. The trachea shows a relative small increase in volume to allow the air flowing into the lungs (from 0 s to 1.3 s). Comparing the deformation of the cartilage rings between healthy and stenotic tracheas, it can be seen that these open only partially with respect to the healthy trachea (Fig. 8(b)). In Fig. 9, the maximum principal stresses are shown and compared with those of the healthy trachea. At inspiration, the muscular membrane is in tension and the maximum stress is 3 KPa. Showing only the tracheal cartilaginous parts as in Fig. 9(a), it can be seen how they open during inspiration while the muscle expands (compare with Fig. 8). However, in this case, the deflections are smaller than those registered in the healthy trachea due to the presence of the stenotic fibrous cap, which absorbs most of the load and restrains the cartilage opening capacity. The maximum stress is not obtained at the cartilage rings but, as expected, in the stenotic region, which increases the rigidity of the tracheal tube upstream of the constriction during inspiration and the downstream of it during exhalation. In addition, the maximum stress is slightly smaller for the diseased trachea. This is due to the different pressures applied to the models (smaller for the diseased trachea), which are subjected to the individual spirometries of the two patients. The membrane muscle is not free to dilate in the same way as shown in the healthy trachea (see Fig. 7) where the maximum displacements were located along the tracheal axis, on the same muscle. On the contrary, the maximal displacement for the pathological trachea is located in a smaller area of the interface between muscle and cartilage. Finally, at the bottom of Fig. 9, the maximum principle stresses of the stenotic region are displayed. The maximum values on the interior surface of the constriction are around 2 kPa. The highest values are shown in the posterior side of the stenosis (point P in Fig. 9) where the fibrous stenotic cap is in contact with the cartilage rings. This confirms the overload of the stenosis, which discharges the cartilage rings, preventing their opening/closing function. The maximal stress region on the cartilage is in fact registered at the interface with the stenosis (Fig. 9 top-right and bottom), on the superior part of the trachea. On the contrary, the healthy trachea shows its maximal values longitudinally on the central-lower part of the cartilage, as shown in Fig. 9 top-left.

5.5 FSI Versus CFD Comparison. Both FSI and CFD models were computed using the same fluid flow boundary conditions. In Fig. 10, the results of rigid wall simulations of the healthy and stenotic tracheas are shown with those obtained with the FSI approach. For the healthy trachea, the peak velocity at the median section of the trachea is 12 m/s in the CFD solution, which is about 10% higher than that obtained with FSI (10 m/s). In addition, the velocity in the daughter airways is higher, even though the increment is less than 5% for both left and right bronchi. For the diseased trachea, the flow patterns obtained at the constriction yield a different longitudinal vortex at peak flow during inspiration (see Fig. 10(b)). The longitudinal vortex in the CFD solution is larger and appears shifted downstream with respect to the FSI solution. In addition, the stenotic velocity jet in the FSI model is about 10% higher than that in the CFD model. Air flowing through the constriction begins to roll up after the narrow section, generating a strong recirculation area, as shown in Fig. 11, where

![Fig. 9 Comparison of the maximum principal stresses (in Pa) (a) (left) of the complete model and (a) (right) of the cartilage rings between healthy and stenotic trachea respectively, at peak flow during inspiration. In (b) the stress distribution of the stenotic region is shown.](image1)

![Fig. 10 Comparison between FSI and CFD computations at peak flow during inspiration: (a) healthy trachea and (b) stenotic trachea](image2)

![Fig. 11 Comparison between FSI and CFD vorticity magnitude (in s⁻¹) at peak flow during inspiration (top: healthy trachea; bottom: stenotic trachea (frontal/lateral section))](image3)
the vorticity magnitude is illustrated for each model, indicating the severity of the flow conditions in the diseased trachea. While for the healthy case no particular vortex structures are identifiable, mainly due to the strong axial inflow velocity, a strong vortical structure is visible in the stenotic region for both (CFD and FSI) diseased tracheas. The vorticity is higher for the CFD solution than for FSI, which agrees with the velocity field discrepancy obtained in that region. Downstream of the constriction, the vorticity progressively loses its intensity and in the daughter airways there are no secondary structures visible. The depicted maximal vorticity values are higher than those reported in other studies [66,67] because of the normal breathing inflow conditions used in those studies compared with the forced respiration adopted in the present work. Differences in the flow field for CFD and FSI simulations are also previously reported by Wall et al. [22] for a healthy trachea.

5.6 Turbulence Statistics. To gain insights into the turbulent characteristics of the tracheal airflow, we analyzed the turbulent kinetic energy (TKE). Figure 12 shows the TKE contours corresponding to a median longitudinal section of both models. For the healthy trachea (Fig. 12, left), the kinetic energy exhibits homogeneity through the tracheal axis due to the aforementioned strong axial velocity component (slightly stronger for the CFD solution). On the contrary, for the stenotic trachea, a higher TKE is observed at the constriction. The CFD model exhibits a lower TKE as the flow field illustrated in Fig. 10 showed lower velocities in the stenotic region. Noteworthy is that while the scale in Fig. 12 is the same for the stenotic and healthy trachea, the TKE is proportionally higher for the diseased trachea, taking into account the smaller flow rate used for CFD and FSI models of this pathology.

5.7 Limitations. Although this work contributes to the understanding of the response of a human trachea under impedance-based conditions and represents an improvement with respect to studies done before, there are some limitations. In these patient-specific tracheas, due to the complexity of the complete geometry, we have modeled only the first bifurcation and neglected the upper tracheal geometry. It is well known that the laryngeal flow causes a jet that affects the tracheal airflow. Even if we count the effect of this jet through skewed tracheal extensions, a complete model including this part should be considered for the next studies. In the impedance computation, several geometrical approximations were taken. A study of the influence of geometrical parameters of the fractal network on the pressure waveforms (as for instance, α, β, and l₀) should be done. In addition, as already mentioned, we assumed zero impedance at the alveolar level even if we know that the alveolar pressure is not zero but varies from negative to positive pressures (with respect to the atmospheric pressure) during breathing. Moreover, the presented FSI functional model should be extended and tested for a big number of pathologies, considering also the inclusion of prosthesis. In this sense, this is the first step of a future classifying work.

6 Conclusion
The effect of the unsteady airflow on a healthy and a stenotic trachea was studied under impedance-based boundary conditions and turbulence modeling. While our models have a single bifurcation, we took into account the resistance of the respiratory system through the use of the impedance-based method obtaining physiologically realistic flow features that qualitatively agree with previous studies for both healthy and pathologic tracheas. The importance of this work can be seen in the quantification of flow patterns and wall stresses inside different airway geometries such as those with stenotic and stented tracheas. Such numerical analysis could be performed before a surgery in order to provide help in the surgical technique and in determining the choice of stent type. In this paper, we presented a computational approach with the aim of constructing a diagnostic tool that in the future could predict the tracheal stresses and deformations due to the influence of different airway geometries originating from different geometries, pathological situations, and endotracheal prosthesis implantations. In this way it could be possible to extract physical variables not assessable in vivo as local pressure drops, muscular deflections, and stresses. Finally, with the aim of showing the importance of FSI simulation in the study of the respiratory system, we compared the fluid fields computed through the FSI and CFD approaches. The fluid structures postprocessed from these solutions, as found in previous works, showed differences between results obtained with rigid and deformable tracheal walls. This confirms that FSI is not only necessary for calculating stresses and strains inside the trachea but it is also important to extract fluid features that are influenced by the deformable walls.

Acknowledgment
This study was supported by the CIBER-BBN, an initiative funded by the VI National R&D&i Plan 2008-2011, Iniciativa Ingenio 2010, Consolider Program, CIBER Actions and financed by the Instituto de Salud Carlos III with assistance from the European Regional Development Fund and by the Research Project No. PI07/90023. The technical support of Plataforma for Biological Tissue Characterization of the Centro de Investigación Biomédica en Red en Bioingeniería, Biomateriales y Nanomedicina (CIBER-BBN) is highly appreciated. We finally wish to thank Christine M. Scotti (W.L. Gore & Associates, Inc. Flagstaff, AZ).

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