Analysis of lung parenchyma as a parametric porous medium

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First published June 1, 2006; doi:10.1152/japplphysiol.01548.2005.—The dynamic behavior of the lung in health and disease depends on its viscoelastic properties. To better understand these properties, several mathematical models have been utilized by many investigators. In the present work, we present a new approach that characterizes the dynamics of gas flow into a viscoelastic porous medium that models the lung structure. This problem is considered in terms of the lung input impedance on a macro level and parenchymal tissue impedance on the level of an alveolar wall. We start from a basic theoretical analysis in which macroscopic tissue deformations are represented in accordance with the linearized Navier-Stokes equations. This approach has strong theoretical underpinnings in other situations but has not been applied to analyze the impedance of the inflated lung. Our analysis provides a theoretical basis for analyzing the interaction between flow into the lungs as a biophysical diffusion process and parenchymal viscoelasticity described phenomenologically, within the frameworks of standard viscoelasticity and structural damping. This lung impedance incorporates parameters of porosity, permeability, and viscoelasticity on micro and macro levels of parenchymal tissue. The analysis shows the theoretical basis of the transformation from the impedance of alveolar walls or isolated tissue strips to that of the intact parenchyma. We also show how the loading impedance at the lung boundary may have a significant impact on the dynamic behavior of whole lung viscoelasticity. Our analysis may be useful in directing specific tests of different models and for analyzing experimental measurements of viscoelastic parameters of lung material under normal and pathological conditions.

impedance; admittance; resistance; viscoelasticity; structural damping; tissue resistance

AN IMPORTANT MACROSCOPIC MANIFESTATION of the pathophysiological structural changes in pulmonary disease is the viscoelastic behavior of the lung. Classically, this experimentally derived behavior is studied as a time-dependent measure of pressure and flow (17). The most useful characteristic of these functions is impedance, which is simply the ratio of time-dependent pressure and flow expressed as a complex variable. Impedance then is a function of the frequency, whose magnitude and phase are dependent on the losses and stored energy of the physical substance. In a linear physical system, one can analyze specific models and theoretically calculate the different impedances. To have the most practical utility, the lung impedance should be describable from the intrinsic viscoelastic properties of the parenchymal tissue. The most commonly used model to characterize lung impedance is known as the constant-phase (CP) model (9, 12). This model takes into account the finding that the phase angle of the mechanical impedance is often constant over a wide range of frequencies (9, 12, 23). In addition to its use in lung physiology, the CP model has also been widely used in polymer physics, material mechanics, and geophysics, suggesting a broad utility to characterize material properties (11). The CP model for the lung was originally based on an empirical pressure response to step changes in gas volume as performed with rubber balloons and excised lung tissue (12). The empirical relationships thus derived involved an inverse power law of time with a power exponent less than one, and a Fourier transformation of this functional dependency results in the constant-phase impedance relationship.

The CP model, however, does not directly address the link between tissue properties and lung properties. Although isolated tissue strips and intact lungs both can be modeled with the CP model, the theoretical linkage between them has not been described. This would not be an issue if the lung really were like a rubber balloon, because then the value of the lung input impedance would be identical to the parenchymal tissue impedance (i.e., analogous to the balloon wall in this case, and ignoring airway resistance and gas compression). However, the fact that this is not the case is highlighted in a textbook scanning electron micrograph of a cut lung surface as shown in Fig. 1. The lung is thus far closer in appearance to a sponge than a child’s balloon, and this anatomic complexity of a lung structure with such microscopic cavities needs to be considered. A second factor that only exists in the intact lung is the fact that the lung has a finite boundary condition dictated by the visceral pleura. Although this may not be a large constraint at low lung volumes, it may play an increasingly important role at higher inflations.

In the following analysis, we have attempted to theoretically provide the link between the impedance of intact lung parenchyma distal to terminal bronchioles (referred to here as the macro level) and that of the lung tissue at the level of an alveolar wall (micro level). To do this, we need a model that can dynamically handle the spongelike nature of the lung. One such model has considered the lung by modeling it as a porous medium (16). With this model, we have achieved a generalized mathematical description of the link between micromechanical and intact parenchymal impedances. This impedance analysis incorporates the process of flow diffusion in a porous lung medium and the influence of the pleural boundary condition (22) or loading impedance on this process. As will be shown, this theoretical basis can be used to characterize this linkage between several different lung models, including classical models with independent values of elastance and viscosity (2, 7) and models with structural damping that tightly couple elasticity and viscosity, as in the CP model (9, 12).

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OLEW-LEWIS EQUATIONS OF LUNG MECHANICS

Owen and Lewis considered the physical system of the lung as a fluid-solid system described by the Navier-Stokes equation, in terms of fluid velocity, pressure, fluid viscosity, Young’s modulus \( (E) \), shear viscosity \( (\mu) \), and density \( (\rho) \) as characterized by the stress-strain relationships for alveolar tissue (16). Utilizing the homogenization method of analysis (4), they considered the microscopic three-dimensional system to derive macroscopic equations, assuming the lung to be composed of a matrix of collagen, elastin fibers, and water. The micro scale length they used was that of an alveolus, \( l \equiv 2 \cdot 10^{-4} \text{ m} \), and the macro scale was related to length of an acinus with \( L \equiv 10^{-2} \text{ m} \). Thus the macro scale includes all of the lung tissue distal to terminal bronchioles. Defining \( \epsilon = ll/L \), and noting that \( \epsilon \ll 1 \), they wrote functional equations for pressure, velocity of fluid, and displacement of tissue as a series of two scale presentations (16). Proceeding with further subsystem analysis and averaging the three-dimensional equations over the unit cell by volume of the alveolus, they were able to develop a fundamental system of equations for lung tissue dynamics. They proceeded to derive a one-dimensional system of equations in terms of the macroscopic physical variables of pressure, fluid velocity, and material displacement in any specific direction. It is worth emphasizing that these equations contain macroscopic viscoelastic parameters that derive from integration in a three-dimensional space. By analogy, this is not unlike the situation relating microscopic random Brownian motion of molecules in three dimensions to create a net pressure that can be measured with a pressure transducer oriented in one specific direction. Although the analysis given by Owen and Lewis was elegant, what was missing from it was an extension of the theory to develop practical equations for lung impedance.

To address this omission, we derive in the following analysis a solution for lung impedance in terms of the physical functions of pressure \( (p) \), fluid velocity \( (v) \), and solid displacement \( (u) \). The time dependency \( (t = -\infty) \) of all functions can be written as \( \exp (j\omega t) \), where \( \omega \) is angular frequency, with time derivatives in the form \( \partial F/\partial t = j\omega F \), where \( F \) represents any of functions \( p, v, \) or \( u \). In the original analysis, the lung parenchyma was analyzed with a zero boundary condition for the pressure function. Although this situation may sometimes be approximated in a real lung at low stress, in general there is a finite load imposed by the tissue of the boundary. Thus we have extended the analysis to the more generalized situation including parameters of the boundary loading.

The system of equations presented by Owen and Lewis for a one-dimensional dynamic system (16) can be rewritten (ignoring the compressibility and iner tance of air) in the following form:

\[
\begin{align*}
\nu - j\omega \phi u &= - K(\rho, \omega)^{-1} \cdot p' \\
v' - j\omega \phi u' &= j\omega \phi u + j\beta(\rho, \omega L^2)^{-1} \cdot p \\
(\bar{E} + j\bar{\mu}) \cdot u &= - \alpha \cdot (\rho, \omega L^2)^{-1} \cdot p
\end{align*}
\]

where \( j = (-1)^{1/2} \), the prime symbol indicates differentiation with respect to spatial coordinate \( x \).

The other parameters of these equations are taken from Ref. 16 and shown in Table 1. Although Owen and Lewis described \( \beta \) as the macroscopic manifestation of the composite microscopic elements (16), they considered it to be negligible for all conditions. However, this variable \( \beta \) can be considered as a tissue admittance, and our analysis shows that, whereas it can be negligible for \( p \), it cannot be ignored for \( v' \). For this reason, we have added a component proportional to \( \beta \) in our Eq. 2.

The system of Eqs. 1–3 has physical meaning with respect to any diffusion process in a porous medium. With specific reference to the lung, we note here that the original article (16) gave an estimation of the parameters on the basis of summaries of published data, with special emphasis on specific properties of the viscosity in two cases: standard viscoelasticity (SV) and structural damping (SD). Such characterizations were originally considered by Fredberg and Stamenovic (6), on the basis of the assumption that an integral biomechanical element is responsible for both the elastic and viscous stress in the tissue, and as a result the viscosity of the lung tissue is inversely proportional to the frequency. We have further developed and extended this concept in the framework of the parametric model of structural damping, incorporating the frequency dependence of the viscoelastic parameters of a porous lung material, which adds an additional degree of freedom (see APPENDIX).

INPUT IMPEDANCE OF THE LUNG PARENCHYMA

Our objective was to transform the system of Eqs. 1–3 to equations for pressure \( (p) \) and flow \( (f) \). In the particular case of a one-dimensional system, the value of flow is coupled with fluid velocity as

\[
f = S \cdot v
\]

where \( S \) is cross-sectional area penetrated by the flow. Next we substitute Eq. 3 in the right side of the Eq. 2 and then insert this result into a differentiated Eq. 1, to arrive at the equation for the pressure:

\[
p'' = D \cdot p
\]
This equation results from integration by time of the respective diffusion equations. The coefficient $D$ can be represented in the following form:

$$D = (KL^2)^{-1} \cdot \frac{\alpha_b^2}{\mu - jE}$$  \hspace{1cm} (6)

where $\alpha_b^2 = \alpha^2 - \beta (E + j\mu) = \alpha^2 - \sigma (\phi + \alpha)$, and with the quantitative values in Table 1 becomes $\alpha_b^2 \approx 0.35$.

Next we substitute the relationship $p = D^{-1} \cdot p''$ in Eq. 3 and use this result in place of $u'$ in the differentiated Eq. 1. After integrating this resultant equation (with a zero integration constant), we get an equation describing the functional dependency between $p'$ and $v$. We obtain the functional dependency between $v'$ and $p$ by simply substituting Eq. 3 into Eq. 2. Finally, using Eq. 4, we arrive at the following two equations to replace Eqs. 1–3:

$$p' = z_d \cdot f,$$

$$f' = y_d \cdot p,$$  \hspace{1cm} (7)

where

$$z_d = \frac{\rho_0 \omega}{KS} \cdot \frac{\alpha^2 - \beta (E + j\mu)}{\beta (E + j\mu) - \alpha (\phi + \alpha)},$$

$$y_d = \frac{S}{\rho_0 \omega L^2} \cdot \frac{\beta (E + j\mu) - \alpha (\phi + \alpha)}{\mu - jE}.$$  \hspace{1cm} (8)

Here it is important note that $z_d \cdot y_d = D$.

This system of Eq. 7 has a well known physical interpretation as equations of a long transmission line (Fig. 2), with specific longitudinal impedance density,

$$z_d = \lim_{\Delta x \to 0} \frac{\Delta z}{\Delta x},$$

and cross-sectional admittance density,

$$y_d = \lim_{\Delta x \to 0} \frac{\Delta y}{\Delta x}.$$  \hspace{1cm} (9)

These parameters give physical meaning to the propagation of the gas through lung parenchyma. This is somewhat analogous to the acoustical propagation of gas pressure perturbations, where the densities of the longitudinal impedance and cross-sectional admittance characterize the compressibility. However, in our situation, we are concerned only with the compressibility (deformation) of lung parenchymal material and not with a gas compressibility, because such effects are relatively minor over a frequency range of less than 20 Hz.

Figure 2 includes the load impedance, $Z_L$, characterizing the boundary condition with $Z_L = p_f f_L$, where $p_L$ and $f_L$ are pressure and flow at the boundary of the lung, respectively. Note that flow, $f_L$, is proportional to velocity, $v_L$, of the fluid, and that $v_L$ exactly equals the velocity of the displacement of

![Fig. 2. Equivalent schematic of the diffusion process in a porous lung medium.](http://jap.physiology.org/)
the membrane $du/dt$ at the periphery of the lung. The impedance, $Z_L$, thus characterizes the viscoelastic impedance of the pleural membrane surrounding the lung. The solution of the system of Eq. 7 can be represented as follows:

$$p = p_1 \cosh(\delta(L - x)) + f_2 Z_0 \sinh(\delta(L - x)),$$
$$f = f_0 \cosh(\delta(L - x)) + (p_1/Z_0) \sinh(\delta(L - x))$$

where

$$\delta = \sqrt{\varepsilon_d \cdot \gamma_d} = \sqrt{D} = \gamma + j\omega$$

is the propagation constant of the diffusion process and $\gamma$ and $\omega$ are the attenuation and phase constants of the diffusion process, respectively;

$$Z_0 = \frac{z_d}{\gamma_d} = \frac{\delta}{\gamma_d} \sqrt{D}$$

is the wave impedance of the diffusion process. The value of the wave impedance, $Z_0$, plays a very important role in definition of lung impedance; in accordance with Eq. 8, this parameter can be represented as:

$$Z_0 = B\rho_s \omega L \sinh^2(\bar{\mu} - j\bar{E})(\delta L)$$

where

$$B = [\beta(E + j\bar{\mu}) - \alpha(\phi + \alpha)]^{-1}$$

This formula for $B$ can be simplified after substitution of the parameters from Table 1 into Eq. 12 as following:

$$B = [(\phi + \alpha)(3\sigma - \alpha)]^{-1}$$

Using the values provided in Table 1, we have, in our particular case, $B \approx 2.9$.

The next step in this simplification is to separate the value of the transformed parenchymal tissue impedance in formula Eq. 12 as

$$Z_i = B\rho_s LS^{-1}\omega(\bar{\mu} - j\bar{E})$$

and hence we have

$$Z_0 = Z_i \cdot (\delta L)$$

Equation 16 together with the load impedance $Z_L$ can be used to define the impedance of the whole lung. Considering $Z$ as the ratio $p_0/f_0$ at $x = 0$, Eq. 9 yields the following formula for lung impedance,

$$Z = Z_0 \frac{Z_L + Z_0 \tanh(\delta L)}{Z_0 + Z_L \tanh(\delta L)}$$

This formula represents the general relationship between lung input impedance and the material parameters of the lung.

**ANALYSIS OF THE LUNG INPUT IMPEDANCE**

Equation 17 allows interpretation of the influence of the internal viscoelastic parameters of parenchymal tissue [through parameters $Z_0$ and $(\delta L)$] and loading impedance ($Z_L$) on the overall lung impedance. The role of these parameters is dependent on values of the porosity ($\delta$), Poisson ratio ($\nu$) of the lung tissue, and the ratio between macroscopic Young’s modulus of the lung tissue ($E$) and Young’s modulus of the alveolar wall ($\bar{E}$) (through parameters $\alpha$ and $\sigma$). The specific relations are shown in Eqs. 6, 8, 15, and 16 and Table 1.

We next estimate significance of the propagation parameter $(\delta L)$, which can be represented in accordance to Eqs. 6 and 10 as following:

$$(\delta L) = \alpha_3 (KE)^{1/2} \cdot \left(\frac{\mu}{E} - j\right)^{-1/2}$$

In general, this variable can have a significant effect on the impedance in both normal and pathological conditions. However, in the case when parameters are close to those represented in Table 1, we can use this to simplify the equation. In this case, we have $\alpha_3 (KE)^{1/2} \approx 0.018\omega^{1/2}$, and with the approximation of $\tanh(\delta L) \approx \delta L$ for frequencies less than 10 Hz, Eqs. 16 and 17 give us

$$Z = \frac{Z_0 Z_L + Z_0^2 (\delta L)^2}{Z_0 + Z_L}$$

In the limiting case when $Z_L \gg Z_0$ we have from Eq. 15:

$$Z = Z_i \cdot B\rho_s LS^{-1}\omega(\bar{\mu} - j\bar{E})$$

showing that the lung input impedance equals the transformed parenchymal tissue impedance. We still have to take into account the fact that the transformation coefficient $B$ depends on porosity and the ratio between Young’s moduli measured on the macro and micro level. With the quantitative variables in Table 1 taken from Ref. 16, Eq. 20 gives, under standard viscoelasticity (SV) conditions,

$$Z = 5 \cdot 10^4 \rho_s LS^{-1}(1 - j0.615/\omega)$$

and under structural damping (SD) conditions,

$$Z = 5 \cdot 10^4 \rho_s LS^{-1}(1 - j6.154/\omega^{-1})$$

Equation 21 allows description of the dynamical behavior of the lung analogous to a classical lumped-parameter (RC) model with normalized parameters of resistance ($R = 1$) and capacitance ($C = 1.6$). Although Eq. 22 looks like a characterization of the CP model, this is not strictly true, because if the CP model exponent has a value of $-1$, this would automatically make the real part of the impedance equal zero. We also note that most experimental measurements find this power exponent of lung impedance to be in the range of 0.69–0.98. It is worth reiterating that Eq. 22 is obtained by substituting the structural damping model of viscosity used in Ref. 16 into Eq. 20. Furthermore, the time or frequency dependence of viscosity ($\mu$) and elasticity ($E$) from the structural damping model underlies the frequency dependence of impedance (G and H) in the CP model. Thus, to avoid potential contradictions between Eq. 22 and the CP model with measurements in actual physiological conditions, we offer a phenomenological representation of viscoelastic tissue properties in a framework of one variant of the parametric structural damping model (see Appendix). In accordance to this model along with Eqs. A.9–A.13 we can rewrite Eq. 15 as follows:

$$Z_i = R_i \cdot \left[1 - j\tan(\pi\alpha/2)\right] \cdot \omega^{-\sigma_i}$$

where $R_i = B\rho_s LS^{-1}$ is transformed parenchyma tissue resistance.
Finally, to complete the solution, we still have to include the loading impedance $Z_L$. Initially, we described this parameter as the impedance of the membrane material surrounding the lung parenchyma. Viscoelastic properties of this membrane may depend on several factors but are likely dominated by the mechanical properties of the visceral pleura. There have been a number of studies that have attempted to quantify the dynamic properties of this pleural boundary (3, 12, 20, 22). Simultaneous measurement of lung and pleural impedances showed significant differences between their frequency characteristics (22). This fact supports the use of $Z_L$, in accordance with the parametric model of structural damping, and by analogy with Eq. 23 in the following form:

$$Z_L = R_L \left[ 1 - \tan(\pi \alpha_2/2) \right] \cdot \omega^{-\alpha_1} \quad (24)$$

where $R_L$ is the loading resistance.

We can further interpret Eq. 19 using the relationship between the tissue and loading impedances. This formula can be further simplified over the frequency range less than 10 Hz, where terms containing $(\delta L)^2$ can be neglected. Then, on the basis of Eqs. 19, 23, and 24, we have:

$$Z = \frac{Z_L \cdot Z_t}{Z_L + Z_t} = R_t \cdot \frac{1 - \tan(\pi \alpha/2)}{1 + \zeta \cdot \omega^{\alpha_1 - \alpha_0}}$$

where

$$\zeta = \frac{R_t}{R_L} \cdot \frac{1 - \tan(\pi \alpha/2)}{1 - \tan(\pi \alpha/2)} \quad (25)$$

Equation 25 demonstrates that, for a normal lung, input impedance can be described as the parallel connection of parenchymal and loading impedances. Certain caveats apply, however, to this consideration, one being that we have not included any discrete upstream airway resistance, and the other that we do not include any heterogeneity of tissue properties. To the extent that airways smaller than terminal bronchioles are included in the porous matrix, their effect will be included, but the more conventional frictional airway resistance of the larger airways is not. It would surely be possible to add a series resistance to an expanded model without much additional complexity, but including the effect of parenchymal heterogeneity would be considerably more cumbersome. Equation 25 also shows the important role of $\zeta$ on the frequency dependence of lung input impedance. For example, when $\zeta \ll 1$ (i.e., $\alpha_1 > \alpha_0$), the value of the impedance equals the transformed parenchymal tissue impedance with a frequency dependence proportional to $\omega^{\alpha_1}$, at least when $\omega$ is less than $\sim 20$ Hz (i.e., minimal gas compressibility). At the other extreme, when $\zeta \gg 1$ (i.e., $\alpha_1 < \alpha_0$), the value of the impedance equals the pleural impedance with a frequency dependence proportional to $\omega^{\alpha_1}$. This analysis provides a theoretical explanation for the experimental observations comparing the dynamic properties of lung parenchymal tissue in vitro with those in the whole intact lung (8). This work showed a greater power of inverse frequency dependence of lung tissue damping compared with that for the inverse frequency dependence of the whole lung.

To summarize, we can now present a formula for lung impedance over an arbitrary frequency range and with arbitrary parameters. Combining Eqs. 16, 17, 23, and 24 yields:

$$Z = R_t \cdot \frac{\left[ 1 - \tan(\pi \alpha/2) \right]}{\zeta_0 + \zeta \cdot \omega^{\alpha_1 - \alpha_0}} \cdot (1 + \zeta_0 \cdot (\delta L)^2 \cdot \omega^{\alpha_1 - \alpha_0})$$

where $\zeta_0 = \tanh(\delta L)/(\delta L)$.

This impedance formula contains relationships with viscoelastic parameters of the parenchyma and loading materials on the micro and macro levels, also incorporating parameters of the porosity and permeability. This equation thus characterizes the link between the impedance and the structural and material parameters of the lung.

**DISCUSSION**

In this paper, we developed an approach of the analysis of dynamic lung biomechanics based on consideration of the Navier-Stokes equations for gas transport, in conjunction with the viscoelastic equations for porous material at the micro and macro level (4, 14, 16, 18, 24). A solution to this problem based on homogenization theory (4) was published a few years ago by Owen and Lewis for the mechanical system of the lung (16), but, unfortunately, this work stopped short of the analysis needed for application to interpreting the lung impedance. The analysis we present now provides the theoretical basis of both the structural damping and standard viscoelasticity models as applied to the lung. We also show how the tissue impedance can be related to the intact lung.

There have been several attempts to connect macroscopic rheology of the parenchymal tissue with the fibrous microstructure of the cell on the biophysical level (1, 15) and also with the molecular theory of polymer viscoelasticity (19). It was shown that either of these approaches can describe viscoelastic deformations of the parenchymal tissue that follow predictions of the CP model. For this purpose, there are at least two ways to characterize the parenchymal tissue impedance: with either a statistical model or a phenomenological description of viscoelastic tissue behavior. In our approach, because we analyzed lung impedance using a porous structure based on deterministic considerations, it was more appropriate to utilize the phenomenological model for the parenchymal tissue impedance. This approach also enabled either of the two common phenomenological models (the CP or RC) to fit with the analysis. In accordance with published descriptions of these models (6, 16), they can be characterized with elements defined either by standard viscoelasticity (SV for the RC model) or structural damping (SD for the CP model). The SV framework uses constant parameters, and SD framework uses time-variable or parametric elements. One parametric model of the viscoelastic deformation was considered within the constraints of quasi-linear viscoelasticity (7) and was shown to have characteristics approximating the CP model. For the purpose of the jointly considering lung input and tissue impedances, we showed how the same mathematical expressions for the parametric model of structural damping could be applied to either case, as arising from the micromechanical deformation. This analysis supports the work of Suki et al. (19) that originally probed the molecular mechanism underlying the CP model. However, more recent analogies of the time variability of parenchymal viscoelasticity to glasses and polymers can also be fit well with our present analytical framework. For example, Cavaille et al. (5) have shown that the compliance of polymers...
contains four components, among which are two associated with correlated motions of molecules with time-constant parameters as power functions of time. In our model of structural damping we use an identical time variable function for the phenomenological compliance.

One further consideration with regard to our analysis is that we have only considered the whole lung as a homogeneous material, ignoring the potential for parallel regional variations that can lead to heterogeneous behavior. This is an added level of complexity, but if enough information is available on the nature of the heterogeneity, it would surely be possible to use the same analytical model to describe these parallel components. In this case, the lung impedance would be considered as an ensemble of the similar impedances with different parameters and in different one-dimensional directions. This analysis, however, is beyond the scope of the present work.

In conclusion, we have applied and extended the theoretical solution describing the dynamics of a porous medium, with parenchyma viscoelasticity presented by Owen and Lewis (16), to derive a general formula for the impedance of the lung. This result utilizing a variant of the parametric model of structural damping provides an explicit relationship between impedance and parameters of viscoelasticity on micro (alveolar wall) and macro (acinar) levels. We have also shown that the dynamic impedance of the lung in the general case is tightly coupled with parenchymal tissue impedance and with loading impedance. In situations in which loading impedance is significantly greater than tissue impedance, the lung input impedance is then proportional to the parenchymal tissue impedance. To describe the CP model of parenchymal viscoelasticity, we used one variant of the parametric model of structural damping. With this approach, our results allow further analysis of lung impedance with viscoelastic tissue parameters at any level of complexity. This analysis may be useful in directing specific tests of different models used to explain experimental results designed to measure viscoelastic parameters of lung structure under normal and pathological conditions.

APPENDIX

One variant of the parametric model of structural damping. The concept of structural damping was first suggested in the physiological literature by Fredberg and Stamenovic (6). This term is used to describe the phenomenon of frequency independence of the elastic and viscous parameters of biological material (e.g., parameters $E_p$ and $\mu_{SDp}$ in Table 1). Under conditions of structural damping, the lung impedance can be interpreted within the framework of the CP model. In this model, identical frequency behavior of both parameters can exist with the assumption that one unified physical element is responsible for elasticity and viscosity (6). However, their analysis did not describe characteristics of this element that would be consistent with power law of the CP model. It turns out that for this physical element to fit the CP model, one needs to incorporate a slight frequency dependency of the elastic and viscous characteristic of the biological material. On the basis of these considerations and as motivated in the DISCUSSION, we suggest an alternative development of the structural damping concept. This model is analyzed by use of time variable viscosity and elasticity. Although these parameters are proportional to each other on the micro and macro levels (as linked by the constant $\sigma$ in Table 1), for our purposes it is more convenient to work with the macroparameters of the elasticity $E_p$ and viscosity $\mu_{SDp}$. If desired, information at the micro level can be easily obtained from the known constant, $\sigma$.

To further clarify our concept of the parametric model of structural damping, let us consider the simpler situation in one dimension of a parenchymal strip in vitro with cross-sectional area $S_T$, length $L_T$, and internal force $\tau(t)$. The linear deformation $u(t)$ of this material will lead to a strain of $u(t)/L_T$ and stress of $\tau(t)/S_T$. Mechanical properties of the material are represented with elasticity $E_p$ and viscosity $\mu_{SDp}$. To describe the dynamic behavior of standard viscoelasticity in this material, we start from the Maxwell model, describing the relationship between values of the velocity of the linear deformation $u(t)$, $i(t) = du/dt$, and $\tau(t)$, the internal force in the material, in the following form:

$$\frac{S_T}{L_T} \cdot i(t) = \left( \frac{1}{E_p} \cdot \frac{d}{dt} + \frac{1}{\mu_{SDp}} \right) \cdot \tau(t)$$

This formula shows that conventional deformation is described with two separate elements of viscosity and elasticity. With a parametric model of structural damping, we assume the presence of just one elastic element with the following time dependence of capacitance, $c(t) = E_p^{-1}(t)$:

$$c(t) = C_m \cdot \xi \cdot (1 - \tau(t)^k)$$  \hspace{1cm} (A1)

where $C_m^{-1}$ is a constant with units of Young’s modulus; $\tau_s \leq t, k < 1$, and $\xi$ has units of s$^{-1}$.

We include in this consideration a function of the volume, $Q(t)$:

$$Q(t) = c(t) \cdot \tau(t), \text{ and hence } \frac{S_T}{L_T} \cdot i(t) = \frac{d}{dt}Q(t).$$

We then have:

$$\frac{S_T}{L_T} \cdot i(t) = \left[ c(t) \cdot \frac{d}{dt} + C_m \cdot \xi \cdot k \cdot (t - \tau(t))^{-1} \right] \cdot \tau(t)$$  \hspace{1cm} (A2)

Equation A2 is a differential equation with time-varying coefficients. Its solution is well known and can be written with help of the following function $\varphi(t, \tau)$:

$$\varphi(t, \tau) = \exp \left[ \int_{\tau_0}^{\tau} \frac{C_m \cdot \xi \cdot k (\eta - \tau_0)^{-1}}{c(\eta)} \cdot d\eta \right] = \exp \left[ -k \cdot \int_{\tau}^{\tau_0} \frac{d\eta}{\eta - \tau_0} \right]$$  \hspace{1cm} (A3)

Using this function, we have the following solution of Eq. A2:

$$\tau(t) = \varphi(t, \tau_0) \cdot \tau_0 \cdot \frac{S_T}{L_T} \int_{\tau_0}^{\tau} \frac{\varphi(t, \tau)}{c(\tau)} \cdot i(\tau) d\tau, \hspace{1cm} (A4)$$

where the initial tension $\tau_0 = 0$. Then, from Eqs. A3 and A4, and $c(\tau)$ from Eq. A1, we have:

$$\tau(t) = \left( C_m \cdot \xi \right)^{-1} \cdot \frac{S_T}{L_T} \int_{\tau_0}^{\tau} \frac{i(\tau) d\tau}{(t - \tau)^2}.  \hspace{1cm} (A5)$$

We are seeking specific solution of Eq. A2 under synchronized conditions between the momentum $\tau$ at the onset of the pulse function and the momentum associated with the development of the parametric effect. In this case, the current $\tau_0(t)$ will be considered as a limit of $\tau$ as $\tau_s \rightarrow \tau$. That is, we have at $t_0 = 0$:

$$\tau_0(t) = \left( C_m \cdot \xi \right)^{-1} \cdot \frac{S_T}{L_T} \int_{0}^{\tau_0} \frac{i(\tau) d\tau}{(t - \tau)^2}.  \hspace{1cm} (A6)$$

This result describes a convolution relation between $\tau_0(t)$ and $i(t)$. Taking the Fourier transform of both sides of Eq. A6 gives the following:
The function following relationship:

where $\alpha_0 = 1 - k$; $\Gamma(\alpha_0)$ is the gamma function.

In accordance with the theory of the CP model (9, 10), the function $Z_n(\omega)$ can be considered as

$$Z_n(\omega) = \frac{S_T}{L_T} \cdot (q_n - jh_n) \cdot \omega^{-m}$$  \hspace{1cm} (A9)

where

$$q_n = \Gamma(\alpha_0) \cdot (C_n \xi^2)^{-1} \cdot \cos(\pi \alpha / 2);$$

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The function $Z_n(\omega)$ is the tissue strip impedance, and the viscoelastic parameters, $E_n$ and $E_p$, are coupled with this function through the following relationship:

$$Z_n(\omega) = \frac{S_T}{L_T} \cdot (\bar{\mu}_p - jE_p) \cdot \omega^{-1}$$  \hspace{1cm} (A10)

where

$$\bar{\mu}_p = q_p \cdot \omega^2; \quad E_p = p_p \cdot \omega^2$$  \hspace{1cm} (A11)

We can represent Eq. A10 in the following form:

$$Z_n(\omega) = \frac{S_T}{L_T} \cdot \rho_0 \omega^2 L^2 (\bar{\mu} - jE)$$  \hspace{1cm} (A12)

In the context of our model and in accordance with Eq. 14 in the text, we have the following normalized values in Eq. A12:

$$\bar{\mu} = \bar{\mu}_p (p_p \omega^2 L^2)^{-1} \cdot \omega^{-1+(m+1)} = g_k \cdot \omega^{-1+(m+1)}$$  \hspace{1cm} (A13)

$$E = E_p \cdot (p_p \omega^2 L^2)^{-1} \cdot \omega^{-1+(m+1)} = h_k \cdot \omega^{-1+(m+1)}$$

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The relationships in Eq. A13 are the general equations of the parametric model of structural damping.

Glossary

- $B$ transformation coefficient in the tissue impedance relationship
- $c(t)$ capacitance of the parametric model of the structural damping
- $C_M$ constant quasi-capacitance of the parametric model of the structural damping
- $D$ inversely proportional value to the coefficient diffusion
- $E$ normalized Young’s modulus of parenchymal tissue
- $E_p$ physical Young’s modulus of parenchymal tissue
- $E$ macroscopic Young’s modulus
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- $\tau_0(\omega) = Z_n(\omega) \cdot i(\omega)$ equation of state
- $\tau_0(\omega)$ and $i(\omega)$ are Fourier transforms of $\tau_0(t)$ and $i(t)$, respectively. We use the subscript $t$ to indicate the special situation of an isolated tissue strip; $Z_n(\omega)$ is the Fourier transform of the kernel of Eq. A5:

$$Z_n(\omega) = \frac{S_T}{L_T} \cdot (C_n \xi^2)^{-1} \cdot \int_0^\infty e^{-\eta t} \eta \, d\eta$$

where $\alpha_0 = 1 - k$; $\Gamma(\alpha_0)$ is the gamma function.

$$\Gamma(\alpha_0) \cdot (C_n \xi^2)^{-1} \cdot \omega^{-m}$$  \hspace{1cm} (A8)

In accordance with the theory of the CP model (9, 10), the function $Z_n(\omega)$ can be considered as

$$Z_n(\omega) = \frac{S_T}{L_T} \cdot (q_n - jh_n) \cdot \omega^{-m}$$  \hspace{1cm} (A9)

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