

# Novel interpretations of respiratory impedance in COPD based on computational fluid dynamics

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**Abstract:** Although it has been conventionally believed that negative frequency dependence of respiratory resistance and deep decline of respiratory reactance during expiratory phase in COPD are due to ventilation inhomogeneity caused by small airway obstructions, there have been neither experimental theoretical evidence. We have recently confirmed that the airflow limitation site in COPD is the intra-mediastinal airway (intra-thoracic trachea, main bronchi, and right lobar bronchi) whose membranous part is displaced inward during expiration by the use of 4D-CT (see our another presentation). Therefore, we have hypothesized they are also due to the might occur in the membranous part of the intra-thoracic trachea in emphysema by FOT, because it may be easily flip-flopped. In order to validate this hypothesis theoretically, we constructed an oscillated 4D lung model with tracheal deformation and reproduced the respiratory impedance by the use of computational fluid dynamics.

**Keywords:** Pulmonary function test, Forced oscillation technique, frequency dependence, Reactance, Convective acceleration.

## 1. Introduction

Respiratory impedance measurement by the use of forced oscillation technique (FOT) noninvasively provides quantitative information regarding ventilation mechanics during usual breath at rest. In chronic obstructive pulmonary diseases (COPD), there are two apparent features during expiratory phases. One is negative frequency dependence of resistance, and the other is deep decline of reactance. According to the conventional textbook, they are caused by small airway closure (1). However, we have recently revealed that the expiratory airflow limitation in COPD occurs at the intra-mediastinal airway (IMA) by the use of 4D-CT images (see our another presentation). Therefore, interpretation of respiratory impedance based on conventional theory should be reconsidered.

Figure 1 shows the Electron beam CT during expiration at rest in an emphysema patient (courtesy of Prof. H. Kurosawa, Tohoku University), where the dorsal part of the trachea is shifted inward at the beginning of expiration and keeps its concave shape until the end of expiration.

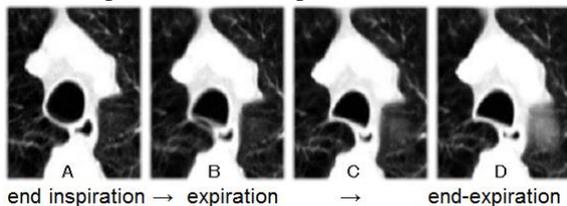


Fig.1. Dynamic CT images during resting expiration in emphysema.

The dorsal part has no cartilage and is called “membranous part”. In normal condition, intra-thoracic pressure is always negative over respiratory cycle and the expiratory flow was driven only by the elastic recoil of the lung tissue. Therefore, the tracheal membranous part is all ways convex at rest. However, in emphysema, the expiratory muscles work so as to compensate the loss of elastic recoil and the intra-thoracic pressure becomes positive. Therefore, the tracheal membranous part becomes slightly concave due to positive intra-thoracic pressure in addition to the hyperinflation of the emphysematous

lung. Such a configuration change of the trachea is thought to be the cause of expiratory airflow limitation. Furthermore, negative frequency dependence of resistance can be explained as follows.

Cheek oscillation is well known as a cause of negative frequency dependence of resistance in normal subjects (1). There is an investigation that the natural oscillation number of the human cheek is about 16 Hz (2). According to this knowledge, we think that resonant oscillation of the cheek will occur when forced oscillation (FO) with 10-20 Hz is given in the oral cavity and the FO will not propagate through the airway. Thus, we have hypothesized that the concave membranous part of the trachea beneath the thoracic inlet must behave in the same way as the cheek and cause negative frequency dependence of resistance. In order to validate the above hypotheses, we simulated FOT by the use of Kitaoka’s 4D lung model and computed the airflow impedance by the use of computational fluid dynamics (CFD).

## 2. Methods

### 3-1. Construction of 4D finite element lung models

A 4D whole-lobe lung model was produced as shown in Fig.2 (3). The model consists of the airway tree from the intra-thoracic trachea to the lobar bronchi and five lung lobes.

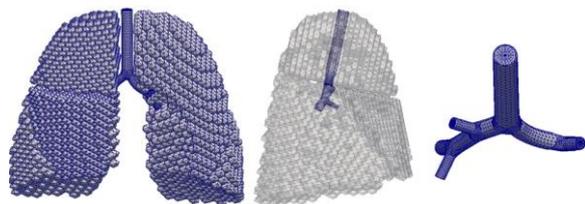


Fig.2. A 4D finite element lung model.

The model consists of 282,272 tetras and 88,343 nodes whose positions are assigned at every step. The functional residual capacity (FRC) of the model is assigned at 2.83 L, and 0.54 L of air is expired over 2.0 sec. The 2.0 sec period is divided into 320 steps with the time step of 0.00625 sec.

FO is assumed to be propagated simultaneously to the whole lung and modeled by superimposing the lung tissue

displacement due to the oscillation on the breathing motion. The frequency of FO is assigned at 5, 10, and 20 Hz. Although some of FOT devices in clinical use produce broad-band pulse waves, we use a single continuous sine wave for the simplicity of analyses. The amplitude of the airflow rate caused by oscillation is assigned at 0.05 L/s.

In order to reproduce negative frequency dependence of the resistance, the central part of the trachea is assumed to oscillate resonantly with 15 Hz when 10- or 20-Hz FO is given. Although the tracheal deformation during expiration in emphysema is not axisymmetric as shown in Fig.1, in the present simulation, the tracheal deformation is assumed as axisymmetric during expiration for simplicity. The expiratory flow rate is also assumed constant for simplicity. The diameter of the whole trachea is assumed to reduce by 20 % during expiration and the central tracheal wall is assumed axisymmetrically displaced with the volume rate amplitude of 0.04 L/s. In order to reproduce deep decline of reactance with 5-Hz FO (X5) at the beginning of expiration, we constructed a dynamic and non-axisymmetric model for the tracheal deformation (Figure 3). The flow rate is not constant but the peak flow rate is 0.5 sec after the beginning of expiration.

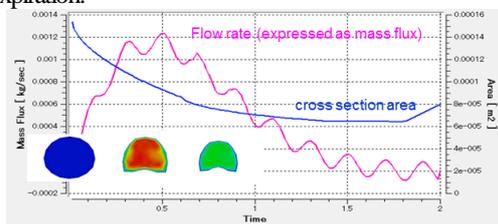


Fig.3. Dynamic and non-axisymmetric model for the tracheal deformation

### 3-2. Airflow computation under moving boundary conditions

Airflow simulation was performed by the use of finite element CFD software (AcuSolve, Altair Engineering Inc., USA). The airflow velocity distribution was obtained by solving incompressible Navier-Stokes equation under moving boundary conditions using arbitrary Lagrangean-Eulerian method and the least square Galerkin method. Turbulence was modeled by dynamic Large Eddy Simulation (LES) method. The pressure of the upper end of the trachea was assigned zero.

### 3-3. Calculation of the respiratory impedance caused by airflow

The pressure fluctuation clinically measured by forced caused by airflow is equal to that of the alveolar pressure. The former corresponds to the simulated airflow rate through the trachea, and the latter to the average of the simulated lobar pressures. Therefore, division of two complex numbers obtained by Fourier transformations of the simulated data gives the impedance caused by airflow.

## 3. Results and Discussion

Figure 4 shows simulated results with 20-Hz FO with (right) and without (left) resonant oscillation of the central part of the tracheal wall. Negative frequency dependence during expiration with resonant oscillation of the tracheal wall is well reproduced (Figure 5). In reality, about 1.5 cmH<sub>2</sub>O/L/s will be added for the

## upper airway resistance and tissue resistances

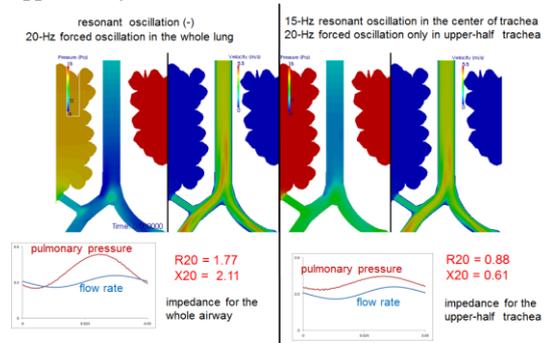


Fig. 4. Oscillation simulation with(right) and without(left) resonant oscillation of the tracheal wall.

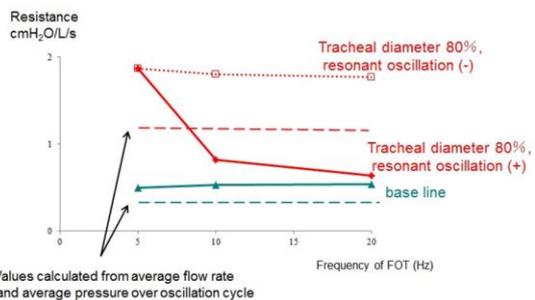


Fig. 5. Relationship between frequency of FO and measured respiratory resistance.

Table 1 shows simulated results for dynamic and non-axisymmetric model of the tracheal deformation. All values are added for R5 by 1.5 corresponding to the sum of upper airway resistance and tissue resistances, and for X5 by - 0.6 corresponding to the elastance. Deep decline of X5 is well reproduced. Convective acceleration generated by the tracheal deformation works as an apparent negative force and decreases the value of reactance.

Table 1. Simulated results for dynamic tracheal deformation

Time(s)	0.2~0.4	~0.6	~0.8	~1.0	~1.2	~1.4
R5(cmH <sub>2</sub> O/L/s)	3.22	3.67	3.70	3.42	2.85	2.36
X5(cmH <sub>2</sub> O/L/s)	-1.02	-1.65	-1.22	-0.71	-0.52	-0.53

## 4. Conclusions

Our simulation study strongly suggests that frequency dependence of resistance and deep decline of reactance during expiration in emphysema are caused by the tracheal dynamic deformation, although the airflow limitation in emphysema has been believed for several decades to occur at small airways. This is the time to rebuild ventilation mechanics based on 4D imaging and CFD.

## Reference

- Oostveen E, et al. The forced oscillation technique in clinical practice: methodology, recommendations and future developments. *Eur Respir J* 22: 1026-1041, 2003.
- Witham EM, et al. The effects of vibration frequency and direction on the location of areas of discomfort caused by whole-body vibration. *Applied Ergonomics* 4 ; 231-239, 1978.
- <http://www7b.biglobe.ne.jp/~lung4cer>