Pneumothorax Influenced 3D Lung Deformations

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Abstract: In this paper we propose a method to simulate morphological changes caused by both closed and tension pneumothoraces. We consider a clinical parameter, the pneumothorax-index (P), the degree of lung collapse, as the input to the simulation. Specifically, we simulate the changes in size and shape of the affected lung. Once the index is obtained, the increase in ventilation rate and the change in the pressure-volume relationship of the affected lung are then computed. For tension pneumothoraces, the air continuously flows into the pleural cavity and thus every inhalation is followed by the changes seen for a closed pneumothorax, until the affected lung collapses. The subsequent closure of pulmonary veins and resulting hypoxia of the affected lung is also presented. Results show a real-time visualization of closed and tension pneumothoraces using a high-resolution 3D model obtained from a normal human subject.

1. Introduction

Medical visualization is a critical component in planning procedural interventions and predicting patient outcomes.[1] Current physically based simulation techniques, such as Finite Element Modeling (FEM) and Finite Difference Modeling (FDM), extend the utility of visualizing the complex anatomy and physiology into both 3D space and the fourth dimension of time.[2] The success of such medical simulations is evidenced by the fact that over one third of all medical schools in the United States augment their teaching curricula using patient simulators.[3] In this paper, we describe such a simulation, which illustrates the complex medical condition of pneumothorax, the presence of air in the pleural cavity of the lung, including its time course and different classifications. The two mechanistic variants of pneumothorax are open (hole in the chest wall) and closed (hole in the visceral pleura or chest lining) pneumothoraces, which lead to different symptoms and treatment methods. The visualization is currently presented for 3D lungs and the mathematical component shows scope for including the effect of changes in cardio-vascular functions. The simulation parameters in this paper are currently set to experimental values obtained from the medical literature.

2. Proposed Method for 3D Lung Deformation

In this section we outline the methodology adopted for the dynamic simulation of 3D lungs deformation. This method is subdivided into two stages. In the first stage we used the method discussed in [4] to parameterize the change in lung volume for a change in pressure (referred to as trans-pulmonary pressure). In the second stage we used the method discussed in [5] to estimate the change in the global lung shape for an increase in lung volume. This is obtained using a physically based deformation method.

Within the context of computer animation, a green's function based deformation was chosen since it has been observed that lung deformations do not undergo vibrations.[6]

Fig 1a shows the shape of the normal human lungs before inflation and Fig 1b shows the shape at the end of inhalation. The PV curve used for this simulation is as shown in Fig 1b.

![Fig 1: The shape of normal lungs (a) at the start of inhalation, (b) at the end of inhalation.]

2.1 Closed Pneumothorax

The 3D deformable lung models under normal breathing lung conditions are driven by the normal human pressure-volume (PV) curve.[4] A ventilation rate of 12 breathing cycles per minute is taken as the normal breathing rate. We use a pneumothorax index, $P$, as input, which conveys a degree of lung collapse. The value of this index ranges from 0 to 1. We assume that the volume of air in the pleural space is considered to be proportional to the ratio of the difference between the radius of hemithorax and the lungs, and the radius of the hemithorax. The change in the volume of air inside an affected lung, $dV$ (in the range from 0 to 1) for a given index value, $P,[7]$ is given as

$$dV = (P)^{0.33}$$

(1)

This corresponding change in lung shape for the change in volume ($dV$) is obtained by scaling down the 3D surface points of the model along their normals from the bilum, which is the entry point for air into the lungs. The modified 3D model is now displaced vertically from the apex so that it sits on the diaphragm.[8] We also assume that in an upright position, the vertical displacement is proportional to the lateral displacements of the lungs described in [8]. This vertical displacement ($A$) was shown (in [8]) to be

$$A = \frac{(P \times 100 - 4.2)}{4.7 \times 2}$$

(2)

Let $\tau$, and $\tau_e$ be the time taken for a single cycle of inhalation and exhalation respectively. The subsequent decrease in the time taken for one breathing cycle of the lungs ($dT$) can be given as

$$dT = \tau \times dV$$

(3)
and

\[ T_i = T_i - \Delta T \]  
\[ T_e = T_e - \Delta T \]  

(4)

(5)

where \( T_i \) and \( T_e \) are the modified time for a single cycle of inhalation and exhalation respectively, and \( T \) is the normal time taken for one breathing cycle.\(^7\) The tidal volume of air flow into the affected lung \( (V_i') \) during inhalation in one breathing cycle is set to be

\[ V_i' = V_i \times (1 - \alpha) \]  

(6)

The tidal volume of the unaffected lung \( (V_e') \) is set to be

\[ V_e' = V_e - \alpha \]  

(7)

where \( \alpha \) is a constant that refers to the increase in volume due to the extra effort in breathing. The range of values for \( \alpha \) can vary for every human subject between 0-2500 milliliters \( / \) and clinically accurate range is yet to be determined. The subsequent changes in the PV curve caused by the additional work during inspiration and the decrease in work during expiration (\( \{10\} \)) are modeled using a method discussed in \( \{7\} \). Such a PV relation allows modeling any degree of lung collapse. The PV relation during inhalation and exhalation is represented using a second-order differential equation with a variable parameter. This parameter is further computed as a linear summation of products of a set of control parameters and trigonometric basis functions that represents the summary muscle resistance. The values of control parameters are extracted from patient's specific clinical data.\(^{11}\) Fig 2a shows the values of a set of control constants used for generating a normalized PV curve of a normal subject as shown in Fig 2b. We simulate a PV curve that represents pneumothorax by varying the normal control constants. One key property of the control constants that we use is that the values as a sequence converge to zero and the rate of convergence is observed to be faster under higher drive conditions. We thus increased the rate of convergence of control parameters during exhalation since the presence of air in the pleural cavity increases the drive to exhale. Similarly we decreased the rate of convergence during inhalation. Multiplying every parameter with its logarithm and dividing it by the logarithm of the first parameter yields a decrease in the rate of change during inhalation. Similarly, multiplying every parameter with its exponent and dividing it by the exponent of the first parameter yields a decrease in the rate of convergence during exhalation. Let \( c^{in} \) and \( c^{ex} \) be the array of control constants during inhalation and exhalation and let \( a_i^{in} \) and \( a_i^{ex} \) be the modified control constants respectively. For a dV\( ^4 \) amount of air entering the pleural cavity, a method to vary the control constants is given by

\[ a_i^{in} = c_i \times \frac{(\alpha - \alpha^i) \times \log(\frac{\alpha^i}{\alpha})}{\log(\frac{\alpha^i}{\alpha})} \]  

\[ a_i^{ex} = c_i \times \frac{(\alpha - \alpha^i) \times \log(\frac{\alpha^i}{\alpha})}{\log(\frac{\alpha^i}{\alpha})} \]  

(8)

(9)

where \( \alpha \) is a proportionality constant that relates the change in control constants to the amount of additional work. Experimental results are shown for small degrees of lung collapse \( (P < 0.01) \). For an experimental analysis the value of \( \alpha \) is set to 0.1; Fig 2a shows the value of modified control constants used for a dV of 50 ml \( (P = 0.008) \). The PV curve generated for the modified control constants is shown in Fig 2b. PV curve variations can thus be meticulously modeled in order to model even small degrees of lung collapse.

\[ dV^i = dV \times e^{-\alpha^i}, \]  

(10)

where \( \alpha \) is a constant which controls the rate of air-flow inside the pleural cavity. The negative exponential component represents the decrease in the rate of airflow caused by the decrease in the trans-pulmonary pressure range. The subsequent decrease in the time taken for the inhalation and exhalation are given by replacing dV by dV\( ^i \) in equations (3), (4) and (5). The change in 3D lung shape is obtained as explained for closed pneumothorax. We now introduce dV\( _{sum} \) which represents the volume of air in the pleural cavity. After a few breathing cycles dV\( _{sum} \) reaches a volume \( V^{'max} \) after which the affected lung cannot inhale or exhale. In order to model the lateral movement of the affected lung we introduce the parameter \( q^i \), which represents the percentage volume of the bounding box of the left \( (i=1) \) and right \( (i=2) \) lungs which is inside the respective lungs. Let \( x^i, y^i \) and \( z^i \) be the dimensions of the bounding box of the \( i \)th lung in X, Y and Z at the end of the \( i \)th inhalation, respectively. We can now represent \( q^i \) as

\[ q^i = \frac{x^i \times y^i \times z^i}{x_0 \times y_0 \times z_0} \]  

(11)

The magnitude of the global displacement in volume is equal to the volume of air directly entering the pleural cavity and is computed as follows. The displacement of the bounding box of the affected lung \( (i=1) \) towards the mediastinum (along the X-axis), denoted by ds\( _x \), is given by the relation,

\[ dV^i = dx^i \times dy^i \times dz^i \]  

(12)
The displacement of the lung towards the mediastinum can now be represented by replacing \( d_y \) with \( q_i \cdot x \cdot \gamma \) in the above equation and simplified as,

\[
ax = \frac{dy}{\gamma'} \cdot b_z' + b_x' \cdot x
\]

(13)

Let \( \beta \) a cardiovascular factor that represents the maximum global displacement by the lung in the state of arrest at which the superior vena cava closes. The closing of the vena cava leads to the collection of blood in the lung and its hyper-expansion until its volume \( V_2 \) reaches \( V_{\text{max}} \), after which it attains the state of arrest. Thus both lungs have reached a state of arrest. \([12,13]\) The value of \( \beta \) is set to be equal to the average diameter of the vena cava (i.e. 18 mm) for simulation purposes. We now discuss the simulation of closed pneumothorax influenced on a patient's left lungs. For simulation purposes, the value of \( P \) and \( a \) are set to 0.1 and 100nl, respectively. Fig. 3a shows the shape of lungs in an upright position before inhalation. It can be seen that the patient's left lung is lower in volume and is shorter than the right lung. Fig 3b shows the shape of lungs at the end of exhalation. It can be seen that the patient's right lung is more expanded as compared to the normal lung in Fig.2b, which is caused by the value of \( a \). An illustration of hyper expansion during tension pneumothorax is shown as in Fig.3c. In this case the patient's left lung is at \( V_{\text{min}} \) and the right lung is at a volume of \( V_{\text{max}} \). The lungs have reached a state of arrest.

3. Conclusion

A method for successfully modeling closed and tension pneumothorax has been presented. The proposed method can simulate lung deformations in a physically accurate manner in real-time. Such deformations can be visualized using advanced visualization environments for effective training and visual guidance in clinical maneuvers. Future work will involve a clinical validation of this method, and will help determine the value of the constants \( \alpha \), \( \varphi \) and \( \beta \) for normal and abnormal human subjects.

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**References**


