Original Article

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TANAFFOS

Evaluation of the Tracheal Stenosis Effects on Airway Resistance and Work of Breathing Using Computational Fluid Dynamics

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Background: Bronchoscopy is one of the most accurate procedures to diagnose airway stenosis which is an invasive procedure. However, a quick and non-invasive estimation of the percent area of obstruction (%AO) of the lumen is helpful in decision-making before performing a bronchoscopy procedure. We hypothesized that there is a relationship between %AO and tracheal resistance against fluid flow.

Materials and Methods: By measuring airway resistance, %AO could be estimated before the procedure. Using computational fluid dynamics (CFD), this study simulates the fluid flow through trachea models with web-liked stenosis using CFD. A cylindrical segment was inserted into the trachea to represent cross-sectional areas corresponding to 20%, 40%, 60%, and 80% AO. The fluid flow and pressure distribution in these models were studied. Our CFD simulations revealed that the tracheal resistance is exponentially increased by %AO.

Results: The results showed a 130% and 55% increase in lung airway resistance and resistive work of breathing for an 80% AO, respectively. Moreover, a curve-fitted relationship was obtained to estimate %AO based on the measured airway resistance by body plethysmography or forced oscillation technique.

Conclusion: This pre-estimation is very useful in diagnostic evaluation and treatment planning in patients with tracheal stenosis.

Keywords: Tracheal stenosis; Bronchoscopy; Airway resistance; Work of breathing

INTRODUCTION

Tracheal stenosis (TS) is a narrowing of the endotracheal lumen caused by trauma during surgery or in intensive care units, infection, tumor, or aspirated foreign bodies. This life-threatening condition most commonly occurs due to prolonged intubation or tracheotomy (1). Patients with tracheal stenosis may have diverse symptoms based on clinical presentation. Until 30% stenosis, the patients usually remain asymptomatic (1). Those characteristically present with complaints of dyspnea and wheezing are easily misdiagnosed as asthma. TS can be categorized according to the Myer-Cotton grading scale which utilizes bronchoscopy to measure the %AO of the lumen: grade 1, <50%AO; grade 2, 51–70%AO; grade 3, 71–99%AO, and grade 4, complete occlusion (2, 3). In addition to this grading scale, Freitag et al proposed a classification system for stenosis based on structural and dynamic types and degree of stenosis, location, and transition zone (4).

Diagnostic assessment for TS includes pulmonary function tests followed by flexible or rigid laryngoscopy

and bronchoscopy (5). Interpretation of spirometry results is often complicated by concomitant lung diseases (e.g., COPD and asthma). So, spirometry is not a reliable diagnostic technique.

There are different approaches to assessing the degree of airway obstructions (6). CT scan can detect the exact location, the extent of stenosis, and the nature of surrounding soft tissues (6). Although the trachea diameter can be assessed by virtual bronchoscopy (a noninvasive well-tolerated procedure by all patients), it is expensive. So, a quick and non-invasive estimation of %AO could assist the surgeon in choosing the most suitable procedure. In this regard, various researchers attempted to find an index for it. Jamaati et al. found significant relationships between plethysmography variables and stricture severity reported by rigid bronchoscopy in patients with post-intubation tracheal stenosis (7). Horan et al. found a strong correlation between the diameter of tracheal stenosis and respiratory impedance measured by forced oscillation technique (FOT) in patients with neurologic injury (8). Verbanck et al. showed that the slope of the resistance-flow curve exploited from the FOT test was well correlated with minimum tracheal lumen area both in normal subjects and COPD patients (9). So, flow dependence of resistance could be a valuable index to detect upper airway obstruction which is not confounded by the presence of concomitant peripheral airway obstruction. Handa et al reported that resistance at 5–20 Hz and reactance at 5 Hz could be used to discriminate between variable and fixed central airway obstruction (10).

Various therapies for TS include dilation, laser resection, stenting, balloon bronchoplasty, and resection anastomosis (11). Laser resection and stent placement are expensive and require expertise only at professional care centers, but balloon bronchoplasty is a relatively simple procedure performed under sedation and at the bedside. The estimation of %AO might be helpful in clinical decision-making for treating tracheal stenosis.

There are several studies on CFD simulations of tracheal stenosis. CFD simulations in anatomically realistic models of tracheal stenosis showed decreased pressure and increased shear stress in the region of a stenosis (12, 13). Brouns et al showed a significant pressure drop in case of severe constriction (more than 70% AO) (14). Poynot et al compute the flow rate within a stenotic trachea relative to that of a normal trachea induced by a fixed pressure difference based on area reduction (15). However, the effects of this increased pressure drop on the lung resistance and work of breathing were not computed.

The purpose of this study was to computationally simulate models of the narrowed trachea to characterize the breathing consequences of tracheal stenosis based on the increased lung resistance and work of breathing. We are looking for a relationship between lung resistance (which could be measured noninvasively during body plethysmography or forced oscillation technique (FOT)) and tracheal area reduction. This relation might be helpful in clinical decision-making for treating tracheal stenosis.

MATERIALS AND METHODS

Model geometry: An idealized model of the trachea with a diameter of 1.8 cm and length of 12 cm was used as a non-stenosed trachea. The so-called "weblike" stenoses of varying constriction were artificially inserted in the upper third of the trachea. A cylindrical segment was inserted into the trachea to represent cross-sectional areas corresponding to 20%, 40%, 60%, and 80% airway obstruction (AO). Four different lengths of 3, 10, 15, and 20 mm for the obstruction region were assumed.

Computational fluid dynamics (CFD) simulations: Steady-state airflow simulations were conducted in Ansys Fluent 2021 for airflow rates corresponding to breathing at rest. The following boundary conditions were used: (1) inlet velocity at the trachea with sinusoidal profile, (2) no-slip conditions (zero velocity) at the wall, and (3) a constant zero outlet pressure. The k-omega turbulence model was used with a turbulent scale of 1 mm and 5% turbulence intensity at the inlet. Previous studies have shown that the standard k-omega turbulence model can accurately reproduce fluid flow in the human upper airway.

We have assumed the rest condition includes minute ventilation of 15 lit/min, respiratory rate of 15 min^{-1}, and inspiratory to expiratory ratio of 1:1. So, the inhalation flow

rate is 1 lit/s which corresponds to a constant velocity of 3.94 m/s

Tracheal resistance, R, can be found by the ratio of the pressure drop from the inlet to the outlet of the trachea, ΔP, to the inhalation flow rate, f, as Eq. (1)

$$R = \frac{\Delta P}{f} \quad (1)$$

Rohrer's equation can be used to describe the resistance of lung airways, R_L (Eq. 2). f is inspiratory flow rate and K_1 is related to the laminar resistance, while K_2 compensates for the turbulent effects on the resistance. The average K_1 and K_2 constants for normal lung airways are 1.85 and 0.43, respectively.

$$R_L = K_1 + K_2 f \quad (2)$$

We have assumed that tracheal resistances are different between studied models. So, the reminder resistance of the lung airways can be found by extracting the resistance of our tracheal model without stenosis from the calculated lung airway resistance. So, the ratio of tracheal resistance to lung airway resistance could be evaluated and compared between different degrees of tracheal obstruction.

The resistive work of breathing (WOB) is required to overcome the respiratory frictional resistance to airflow during breathing by Eq. (10). With our breathing flow assumption, the total resistive WOB can be computed by total respiratory resistance.

We have followed the previous approach to find respiratory resistance including the resistance of lung airways, and pulmonary and chest wall tissue resistance (16).

The resistance of the pulmonary and chest wall tissues, DR_L and DR_w, can be expressed by the following exponential function (17):

$$DR = R_2(1 - e^{-t_i/\tau_2}) \quad (3)$$

where R and τ_2 are the resistance and time constant of the viscoelastic properties of the pulmonary or chest wall tissues. The mean values of R_2 and τ_2 for DR_L and DR_w in normal subjects are 3.44 cmH2O.L-1.s, 1.13 s, and 2.12 cmH2O.L-1.s, 1.29s, respectively. So, the total resistance of the respiratory system, R_{rs}, can be found in Eq. 14:

$$R_{rs} = R_L + DR_L + DR_W \quad (4)$$

The tracheal resistive work can be computed by Eq (5). Since we have used the flow rate of 1 lit/s and inspiratory time of 1 s, the work of breathing (WOB) because of tracheal resistance in CmH2O.L will be equal to $R_{trachea}$ in cmH2O.s.L-1.

$$WOB_{trachea}^{resistive} = ft_i R_{trachea} \quad (5)$$

The total WOB has two components; the resistive WOB needed to overcome the resistance of the respiratory system and the elastic WOB required to inflate the lung.

$$WOB = \int_0^{t_i} f.(R_{rs}.f + V/C_{rs})dt = \int_0^{t_i} R_{rs}.f^2 dt + V_T^2/(2C_{rs}) \quad (6)$$

in which C_{rs} is the respiratory compliance. The first term of Eq. (6) is the resistive work and the second term is the elastic work. Including the elastic work for a normal lung with C=0.1 lit/cmH2O in our computations, the percent of the increase in total WOB can be evaluated.

RESULTS

The contours of velocity and static pressure in the stenosed trachea are shown in Figures 1 and 2. The simulation results show the peak velocity of 30 m/s at the stenosis section with 80% AO where the main pressure drop occurred, probably caused by the disturbed flow near the stenosis. The increased pressure drop in stenosis reflects the high energy needed to drive the flow through these narrow regions. Energy loss associated with such flow expansion after the constriction will be significant. Reversed velocity shown in Figure 1 occurred in the recirculation zones downstream of the obstruction region.

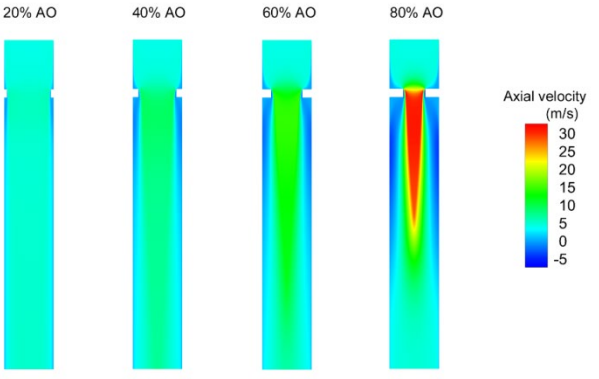

Figure 1. Contours of air velocity with varying levels of simulated tracheal stenosis with 3mm length.

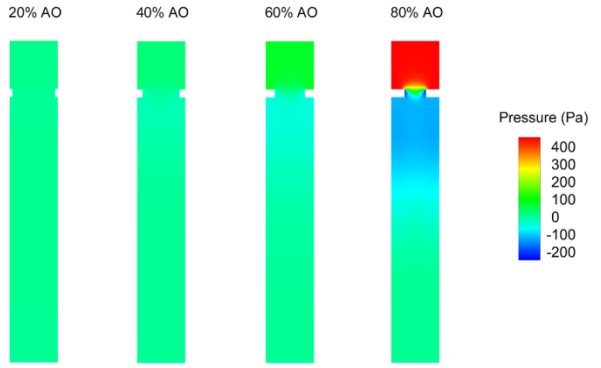

Figure 2. Contours of air static pressure with varying levels of simulated tracheal stenosis with 3mm length

The variation of pressure distribution along the tracheal axis for 80% AO is shown in Figure 3.

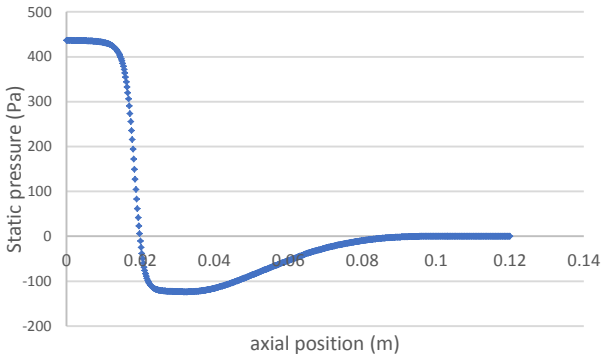

Figure 3. Variation of pressure along the tracheal axis

The effect of obstruction length (3, 10, 15, 20 mm) and %AO on the tracheal resistance is shown in Figure 4. Tracheal resistance increased more than 500% (from 0.049 to 0.302 Pa.s/ml) when obstruction severity increased from 60% to 80% in our model. The increase in obstruction length would decrease the length of the recirculating zone. So, the pressure drop decreases with increasing the obstruction length. Obstruction length had a more significant effect on the tracheal resistance in severe obstructions than in mild obstructions. As shown, the airway resistance decreases with increasing the obstruction length from 3 to 10 mm. However, it hardly changes by a further increase in the obstruction length to 20 mm. So, assuming obstruction lengths of more than 10 mm, the only effective parameter in changing the airway resistance is %AO. So, in the following, we have reported our results for an obstruction length of 10 mm.

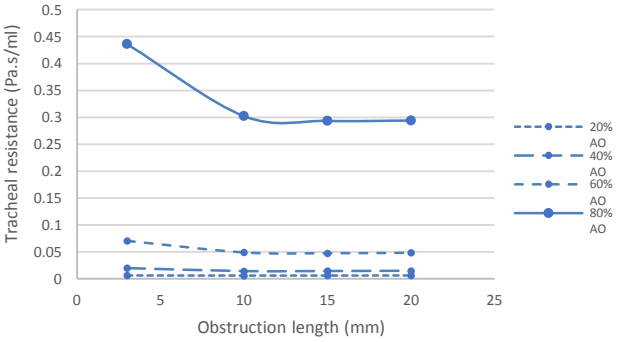

Figure 4. The effect of obstruction length on tracheal resistance in different degree of obstruction

The ratio of tracheal resistance to lung airway resistance in normal subjects is shown in Figure 5. It can be seen that although tracheal resistance is a minor component in lung resistance (less than 5%), it can be significant by increasing %AO and will be increased to 132% of lung resistance by 80% AO.

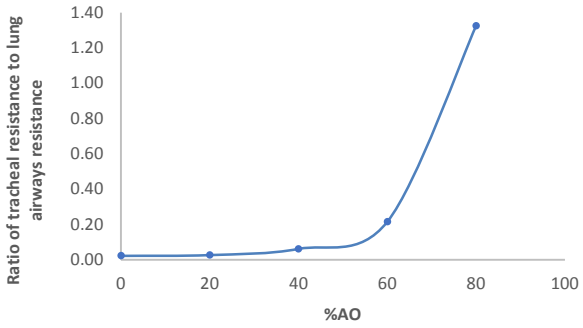

Figure 5. The ratio of tracheal resistance to lung airways resistance as a function of %AO in simulated stenosed models

The following relation between the percent increase in lung airway resistance, %RI, and %AO can be found by curve fitting of Figure 6 with a correlation coefficient of 0.999.

$$\%RI = 0.28 - 0.074(1 - \exp(0.09328 \times \%AO)) \quad (7)$$
$$\rightarrow \%AO = 10.7241 \ln(1 - \frac{(0.28 - \%RI)}{0.074})$$

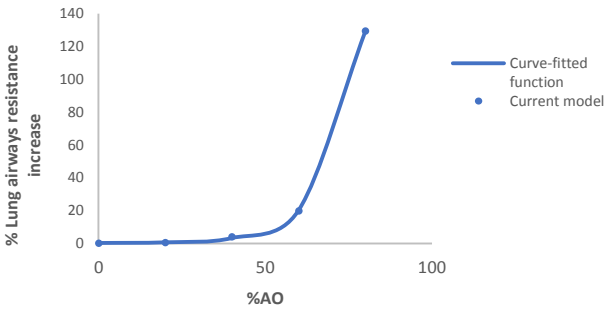

Figure 6. Percent of increase in lung airway resistance (%RI) as a function of % AO in simulated stenosed models; current model and curve fitted function of

The increase in resistive WOB and total WOB between different %AO is shown in Figures 7 and 8. As shown, 80% AO will increase the resistive WOB and total WOB by 55% and 28%, respectively.

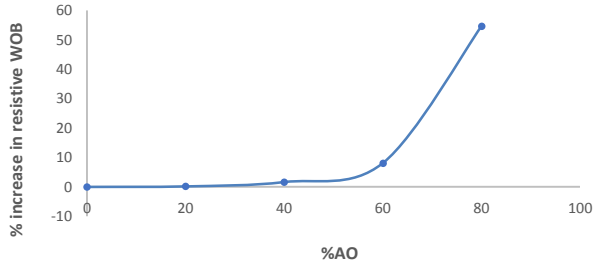

Figure 7. Percent of increase in resistive WOB as a function of %AO

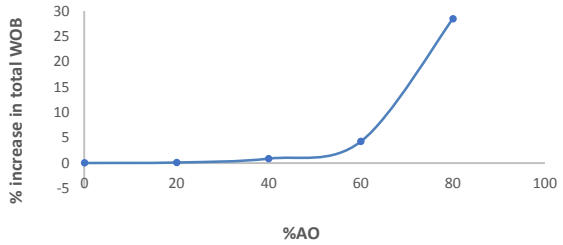

Figure 8. Percent of increase in total WOB as a function of %AO

DISCUSSION

The general shape of the pressure variation along the stenotic airway consisted of a significant pressure drop at the obstruction region and pressure recovery in the distal airway. The most pressure drop occurred across the obstruction region due to flow acceleration and wall friction. The initial pressure rise was associated with flow deceleration, then pressure recovered due to the viscous process in the recirculating region which shows a plateau level. This pressure recovery has been also observed in aortic stenosis (18).

As expected, by increasing the severity of the obstruction, the recirculating and low-pressure regions will be more significant. So, more pressure drops will be observed in higher degrees of obstruction. Tracheal resistance in the stenotic trachea is notably greater than that of normal trachea, agreeing with previous literature (19). The effect of obstruction diameter on the tracheal resistance is greater than the obstruction length. This behavior has been also reported in subglottic stenosis (20).

The curve-fitted exponential relationship found between the degree of tracheal stenosis and the degree of increase in lung airway resistance agreed with experimental measurements of the pressure difference between the proximal and distal sites of the stenosis and the degree of tracheal obstruction by Nishine et al (21). This relation could be used as a guide for estimated %AO using the measured resistance by body plethysmography.

The curve-fitted could help the surgeon estimate the %AO before performing a bronchoscopy to decide about the appropriate treatment. One limitation of this study is using one idealized trachea model excluding oral and nasal cavities, pharynx, and larynx. However, we believe that including upper airway geometries would not change the results much. Future simulation should be performed on a realistic respiratory airway model extending from oral to the end of the trachea including all the key details of the passage. Finally, this study was purely computational, and in vivo measurements were not available for validation. Future in vivo studies should be performed to confirm these findings.

CONCLUSION

This study suggests that the relationship between tracheal stenosis (%AO) and the degree of increase in lung airway resistance can be described as an exponential relationship. Our CFD simulations revealed that the tracheal resistance is exponentially increased by %AO. Thus, an 80% reduction in the tracheal cross-sectional area

is associated with a 130% increase in lung airway resistance.

A curve-fitted relationship for predicting the %AO based on the airway resistance is presented. This formula could help the surgeon estimate %AO before performing bronchoscopy to decide about the appropriate procedure. Moreover, according to our analysis, an 80% AO could increase the resistive WOB by 55%, an intolerable condition that needs urgent treatment.

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