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Power loss mechanisms in pathological tracheas

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ABSTRACT

The effort required to inhale a breath of air is a critically important measure in assessing airway function. Although the contribution of the trachea to the total flow resistance of the airways is generally modest, pathological alterations in tracheal geometry can have a significant negative effect. This study investigates the mechanisms of flow energy loss in a healthy trachea and in four geometries affected by retrosternal goitre which can cause significant distortions of tracheal geometry including constriction and deviation with abnormal curvature. By separating out the component of energy loss related to the wall shear (frictional loss), striking differences are found between the patterns of energy dissipation in the normal and pathological tracheas. Furthermore the ratio of frictional to total loss is dramatically reduced in the pathological geometries.

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1. Introduction

The work of breathing¹ is the energy provided by the subject to inhale a given volume of air. Whilst this has two components, elastic and resistive, it is the resistive which is affected by pathologies in the larger airways (Carroll, 2007). Characterisation of the reduction in patient breathing efficiency provides a useful tool in the assessment of how debilitating a goitre is for the patient and is thus an aid for surgical planning.

The total resistive losses in the airways represent a clinically relevant index that can be measured in patients. Understanding regional contributions to total airway resistance potentially allows the source of breathing difficulties to be isolated. In particular, the trachea represents a location that can be affected by a number of different pathologies that increase the resistive contribution to breathing. Resistive losses during inhalation can be further characterised. One component is the frictional losses from driving inhaled air along the airway walls. This term will be referred to as frictional losses throughout the remainder of this work; its magnitude can be quantified from the wall shear stress (WSS). Admittedly all other losses are ultimately due to friction e.g. viscous interactions, however it is convenient to use frictional loss only to refer to the local resistive effect of the walls. Wall frictional losses are an important measure in the trachea as they highlight

regions of constriction; in particular WSS will be elevated due to acceleration of the flow through the throat of the constriction. Other losses occur when the internal flow is not in equilibrium with the resistive stress imposed by the walls. These losses are due to features such as jets, fluctuations or regions of separation, as described in Section 3.3. Further losses are caused by viscous dissipation (Liu et al., 2004). We refer to all these as interior flow losses.

Goitres are an example of a large airway pathology, in which an enlarged thyroid gland presses on the trachea, potentially displacing and compressing it, which can cause breathing difficulties for the patient. Although the current work considers retrosternal goitre as an exemplar for the impact of airway pathologies on breathing, the methods and findings are equally valid for other pathologies e.g. tracheal stenosis. Surgical decision making is currently strongly subjective with knowledge lacking in regard to the influence of geometry on flow, thus breathing mechanics. Current surgical guidelines such as that given by the American Thyroid Association (Stang et al., 2012) rely on the ratio between diameters measured in the first tracheal ring and constricted segments, without considering other factors which may influence an increased tracheal resistance and work of breathing, such as tracheal curvature or the length of the constriction. Neglect of such factors represents potential limitations of the guidelines and an in-depth analysis of these factors is required to quantify their impact. Given the difficulty in measuring tracheal losses *in vivo*, computational fluid dynamics (CFD) offers not only a convenient tool to determine the significance of such factors, but a means to explore

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how they affect the dynamics of tracheal airflow and suggest alternatives to the current guidelines.

CFD simulations of flow in the trachea has seen significant attention for both healthy and diseased scenarios (Choi et al., 2009; Lin et al., 2007; Comerford et al., 2013; Bates et al., 2014; Malvè et al., 2011). Typically these studies investigate pressure drop in the large airways, however few studies have looked at the power losses and how this relates to the work of breathing. For example, how efficiently is flow driven through the trachea and how does this vary with pathology? Understanding the mechanisms by which different geometric features increase the tracheal losses and hence the work of breathing will allow characterisation of geometric variations and may lead to predictive tools for losses.

A number of studies have considered measures associated with energy loss in the trachea. Brouns et al. (2007) considered an idealised set of constricted tracheas and found a significant increase in tracheal resistance when pathological constrictions were larger than the glottal constriction. They analysed a pressure loss term that considered losses due to friction and geometric features.

Previously, Snyder et al. (1987) hypothesised that kinetic energy, rather than wall shear stress related losses account for the majority of energy losses in the canine central airways. Their study focused on pressure head loss which can be considered a surrogate for energy loss.

In this study we analyse the power loss that occurs in a healthy trachea as well as four pathological scenarios representing the effects of extended airway constriction and, or, curvature and displacement. The power loss is further broken down to ascertain the dominant mechanisms that potentially lead to goitre patients suffer debilitating consequences e.g. shortness of breath.

2. Methodology

2.1. CT data

Anonymised CT scans of the neck and chest were obtained retrospectively from an image database of goitre patients at Imperial College Healthcare Trust. Ethical approval to use the data from local joint research compliance office was waived and research and development approval was granted. Only scans containing the necessary anatomy and reported by a consultant radiologist as displaying a compressive goitre distorting tracheal anatomy from the first tracheal ring to below the carina were selected.

2.2. Geometries

Five geometries were selected for simulation: one normal (N) and four pathological cases (P1–P4). The 3D surface geometries were segmented in Mimics 17.0 (Materialise NV, Leuven, Belgium) using image thresholding, with additional manual manipulation to provide an anatomically correct geometry performed by a trainee ear, nose and throat (ENT) surgeon. Finally, the 3D surface was smoothed using Taubin's algorithm in the Vascular Modelling Toolkit (VMTK) (Piccinelli et al., 2009). The final geometries are shown in Fig. 1. All geometries include anatomy above the glottis, which allows realistic inflow conditions to the trachea (Choi et al., 2009).

These geometries were geometrically analysed using the Vascular Modelling Toolkit (VMTK) software following the methods described by Piccinelli et al. (2009). From this analysis, the centerline was extracted as well as the local curvature. Additionally, using a MATLAB (Mathworks, Natick MA, USA) based in-house tool, the cross-sectional area at each point along the centerline bounded by the wall of the trachea was computed, as was the deviation of the centerline from a straight line passing through the centerline at the first tracheal ring and the carina. Fig. 1 and Table 1 summarise the geometric attributes that distinguish each of these geometries.

Fig. 2 shows the level of deviation from a straight line for the considered tracheas. The normal case, N, deviates little from a straight line. Of the pathological geometries, two cases, P1 and P3 show a large magnitude of deviation from the normal trajectory of the trachea, whilst P2 and P4 deviate into an 'S' shape, as shown in Fig. 1.

2.3. Computation model

The geometries were spatially discretised with polyhedral and prismatic finite volumes in Star-CCM+ 8.04.007-r8 (CD-adapco). Solution of the air flow field was

performed by solving the unsteady Navier–Stokes equations. The solution of the flow field utilised an LES approach in which the field was filtered and the influence of the unresolved or subgrid-scale was incorporated through a sub-grid viscosity term. The eddy viscosity model chosen for the present study was the WALE (wall-adapting local eddy-viscosity) model. This model provides better behaviour (Nicoud and Ducros, 1999) of the eddy viscosity in the near wall region. To account

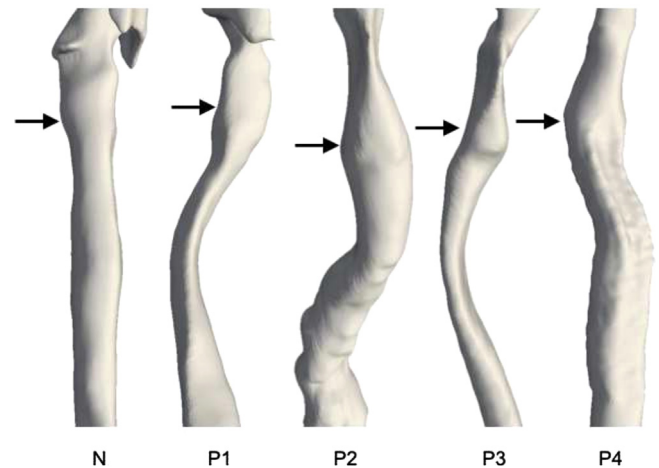


Fig. 1. Illustration of five tracheal geometries. Case N represents the healthy trachea and P1–P4 four pathological cases. Geometries are not to scale for illustrative purposes and have been rotated to demonstrate constriction and deviation by the goitre. The location of the first tracheal ring for each trachea and marks the upstream boundary of the region of interest in Fig. 2.

Table 1

The maximum deviation, mean curvature and constriction ratio in each of the tracheas, measured from the first tracheal ring to the carina. The constriction ratio is calculated as the percentage of the first tracheal ring cross sectional area remaining after subtracting the area of the smallest section of the trachea. The cases are characterised by the geometric effect the goitre has had on each trachea.

Case	Effect of pathology	Maximum deviation (mm)	Mean curvature (1/m)	Constriction (%)
N	–	1.9	7	26
P1	Curvature and constriction	13.8	15	55
P2	Curvature	5.9	23	17
P3	Curvature and constriction	11.6	19	60
P4	Curvature	5.0	17	20

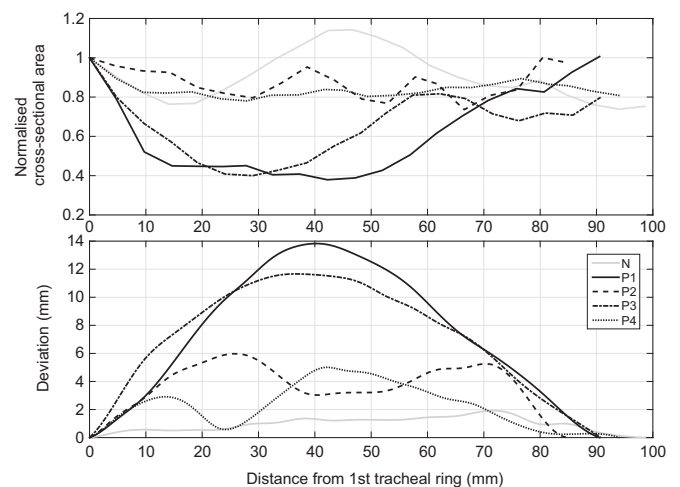


Fig. 2. Deviation of the tracheal centerline from a straight line drawn between points on the centerline within the first tracheal ring and one diameter above the carina.

for potential flow instabilities on the outlets of the domain (see e.g. Gravemeier et al., 2012), the meshes were extruded to prevent backflow. Three different flow rates were considered: 15, 30 and 60 l/min. Whilst the magnitude of losses changes with flow rate, the patterns described are similar in each case at all three flow rates. Therefore, simulations performed at 30 l/min are described throughout. These constant flow rates were distributed uniformly over the inlet of the domain. Bates et al. (2014) showed that whilst realistic breathing conditions are cyclical, for 90% of an inhalation, the flow patterns and distribution are constant, with the magnitude of flow velocity scaling with the flow rate. Therefore, quasi-steady simulations (unsteady simulations with constant boundary conditions) are sufficient whilst purely considering losses. At the outlets, zero pressure conditions were applied. Cases N and P1 include the bifurcation at the carina and hence have two outlets, whilst the others have one. These conditions are suitable when looking at flow in the trachea as the velocity and pressure fields are unaffected by the peripheral flow distribution (Tadjfar and Smith, 2004; Smith et al., 2003). No slip conditions were applied on the walls of the geometry. A second order implicit time stepping scheme was utilised. A total of 2500 time steps ($\Delta t = 0.1$ ms) were performed, in which 1500 instantaneous volume data steps in the interval $t \in [0.1, 0.25]$ were analysed statistically. The first 1000 time steps were discarded to allow start up transients to decay and turbulent flow to be established.

Convergence of the computation was performed both in space and time for case P1, which exhibits both curvature and substantial constriction of the airway. For this case, a highly resolved mesh (~ 9 million volumes), which can be considered a pseudo direct numerical simulation (DNS), was analysed. The mesh was successively coarsened and the time step varied to find a good balance between accuracy and computational time. The primary quantities for convergence were the mean pressure loss along the trachea, and the amplitude and frequency of its fluctuations. These measures were identical in the benchmark and final meshes. Also considered were cross sectional distributions of mean velocity and its variance and the mean dissipation. These measures showed similar magnitudes, and overall distribution patterns, with small local differences. The final mesh had 2.2 million elements, 5 prismatic boundary volumes with an off-wall spacing of 0.15 mm and a geometric progression of 1.3. A time step of 0.1 ms was used.

2.4. Validation

The computational model has previously (Bates, 2014) been verified against experiments of flow through a replica trachea, manufactured by rapid-prototype (case N). Comparing the pressure-flow relationship of the experimental data with that of the CFD prediction produced an R^2 value 0.994. This value suggests that these computational simulations provide a good prediction of losses within the airway.

2.5. Tracheal losses

Flow energy losses in the trachea can be attributed to different mechanisms: the effect of friction at the wall, internal rearrangement of the flow and the dissipation of fluctuations. Fig. 3 shows a control volume of the trachea through which different power losses can be defined.

The total losses are obtained from the energy flux. The transport of energy by the flow through a cross-section of the airway (e.g. the areas marked as A_1 or A_2 in Fig. 3) corresponds to the energy flux, and is defined by

$$\phi_E = \int_A (\mathbf{U} \cdot \mathbf{n}) \left(P_s + \frac{1}{2} \rho |\mathbf{U}|^2 \right) dA \quad (1)$$

where $\mathbf{U}$ is the mean velocity vector, $\mathbf{n}$ is the normal to the cross-sectional plane (e.g. $\mathbf{n}_1$ in Fig. 3), P_s the static pressure. Previously, the use of energy flux in flow analysis has been used to evaluate total cavopulmonary connection haemodynamics by (Whitehead et al., 2007). The rate of energy dissipation can be deduced from the derivative of the energy flux along the airway. Consider a control volume defined as the airway enclosed between two neighbouring planes and an annulus (the tracheal wall) joining them. The total power loss per metre to drive the flow between these planes (e.g. ① and ② in Fig. 3) can be expressed in terms of the difference between the energy influx and efflux

$$E_T = \frac{\partial \phi_E}{\partial x} \approx \frac{\phi_{E,2} - \phi_{E,1}}{l} \quad (2)$$

Frictional energy losses in the trachea are imposed by virtue of the no-slip condition at the airway wall. Mathematically frictional losses (E_F) are related to the wall shear stress and can be defined on the small tube of airway wall shown above. The power losses per metre are given by

$$E_F = \frac{(\mathbf{U} \cdot \mathbf{n})}{l} \int_{A_w} |\boldsymbol{\tau}_w| dA_w \quad (3)$$

where $\boldsymbol{\tau}_w$ is the wall shear stress vector.

Finally, from the above definitions we can define the interior flow losses in the trachea as:

$$E_U = E_T - E_F \quad (4)$$

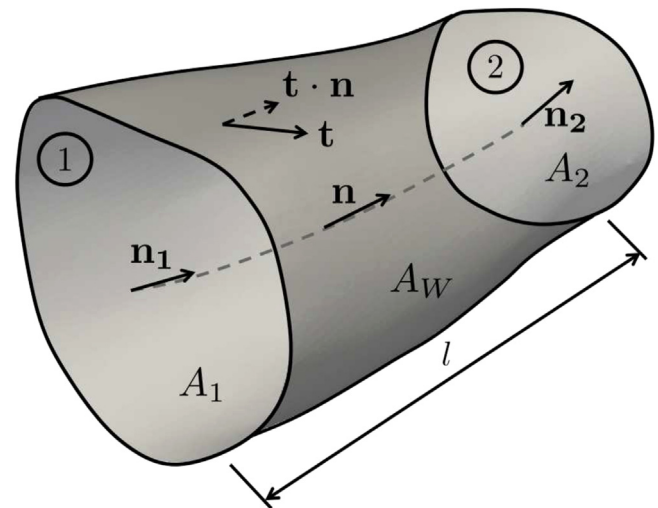


Fig. 3. Control volume in which total and frictional power losses are calculated. Cross-sectional planes ① and ② of area A_1 and A_2 are used to calculate energy influx and efflux. Area, A_w is used to determine the frictional losses by integrating the traction vector ($\mathbf{t}$) projected onto the wall.

2.6. Work of breathing

The work of breathing is an inspiratory measure of the power required to overcome resistance and capacitance of the lung. The resistive component represents the power required to drive air through the trachea and bronchi. The capacitive component is the work required to overcome the elasticity of the parenchyma and chest wall. When there is an increase in either of these components the patient requires more effort to breathe. In this study we will focus purely on the resistive component; the capacitive component is independent of the compressive goitre.

The tracheal component to the resistive work of breathing cannot be measured *in vivo*, but can be predicted via CFD modelling. This value allows the effect of the tracheal pathology to be isolated from any other conditions affecting breathing, such as chronic obstructive pulmonary disease (COPD) and hence the possible surgical outcome of goitre removal may be predicted. This value is equal to the loss of power in the flow through the trachea and is calculated as the difference in energy flux at the first tracheal ring and the carina.

3. Results and discussion

3.1. Total and frictional power losses

Fig. 4 details the rate of total and frictional losses for the normal and four pathological cases. The losses indicated that all pathological cases behave differently from the normal case. Total and local frictional losses are nearly identical where the flow is well distributed over the airway lumen. However, as dissipation is dominated by interior flow rearrangement, local frictional losses may be low or near zero when the bulk of the flow is contained within a jet in the centre of the lumen. In this case, flow near the wall (and hence the wall shear stress vector) is dominated by recirculation regions where flow moves in the upstream direction.

In case N the laryngeal jet forms as the flow exits the glottal constriction, (note this is upstream of the first tracheal ring and corresponds to negative positional values). As is widely reported in the literature (Lin et al., 2007; Longest and Vinchurkar, 2007; Ma and Lutchen, 2006; Zhang and Kleinstreuer, 2011), this jet is directed to the anterior wall by the alignment of the glottis. The rate of power losses in this region is dominated by unsteadiness (fluctuations in jet orientation) and turbulent dissipation (jet breakdown).

Frictional losses represent only a small proportion of the total loss in this region. Distal to this region, the interior flow losses rapidly diminish and total loss becomes dominated by frictional losses; the jet attaches to the anterior wall thus increasing wall shear stress and

the associated frictional losses ($5 \text{ mm} < x < 15 \text{ mm}$). Eventually, the jet cannot negotiate the wall and it detaches back into the bulk flow, leading to a rise in the rate of total power loss and an associated reduction in frictional losses. Further downstream, the frictional losses represent the majority of losses. At this stage, although not fully developed, the dynamics of the interior region of the flow are less intense so the flow comes closer to a state of local equilibrium.

Case P1 is both constricted and deviated. In the immediate post-glottal region ($x < -10 \text{ mm}$), interior flow losses dominate. However due to goitre induced constriction of the geometry, the WSS rises in the region downstream of the first tracheal ring ($x = 0 \text{ mm}$), causing a corresponding increase in the frictional losses. By the end of the constriction ($x \sim 40 \text{ mm}$, see location of minimum area in Fig. 2), the frictional losses dominate. The geometry then expands causing the interior flow losses to rise due to flow separation, finally decaying with reattachment towards the distal part of the trachea.

Case P2 is deviated, but has no constriction. The goitre causes the trachea to deviate into an 'S' shape (see Fig. 2), leading to highly disturbed flow with multiple regions of flow separation. Again, interior flow losses dominate the post glottal region and remain a high proportion of the total loss due to the deviated trachea falling in line with the glottis. This allows the jet to traverse further into the trachea. The jet impingement occurs at the point of inflection of the 'S' bend and flow immediately separates, thus causing frictional losses to remain a low proportion of the total loss. This low contribution of frictional loss to the total losses points to more energetic dynamics in the interior flow, as expected of velocity profile realignment imposed by curvature.

Case P3 has the largest total power loss, primarily due to the smallest glottal constriction and some geometric distortion within this region (see Fig. 1). The intrusion of the geometry into the glottal jet zone leads to a complex pattern of partial impingements and a highly complex and unsteady flow field in the upper region of the geometry ($x < 10 \text{ mm}$) and hence higher total losses. The total power loss decays rapidly as the flow adheres to the anterior wall. Just downstream, the frictional losses rise until the centre of the goitre induced constriction is reached at $x \sim 24 \text{ mm}$ (see location of

minimum area in Fig. 2); at this point the peak frictional loss is the highest of all tracheas due to the very high WSS through the throat of the constriction. Distal to this region, the geometry expands leading to flow separation and an increase in interior flow losses. Finally these again decay progressing towards the carina.

The geometry of P4 follows a similar 'S' shape to that of P2. The glottal jet is quite weak, since the aperture area is relatively large. At $x \sim 50 \text{ mm}$, P4 shows a large reduction in the rate of total power loss, as the flow reattaches. The flow does not reach an equilibrium, due to the irregularity in shape and surface, however the rate of total losses is small in this case due to the large lumen size.

From the power considerations we see that constriction is a major cause of power loss to goitre patients; however it is not the sole cause and other factors must be included. Taking case P2 for example, although the magnitude of the losses are not excessively high, the losses in the post-glottal region extend further into the trachea leading to elevated overall power losses.

3.2. Work of breathing

Table 2 details total and frictional power losses and their relative contribution for each case. The total power loss measurements can be considered the contribution that the trachea makes to the work of breathing for each subject i.e. it measures the efficiency of the trachea. For Case N the total frictional loss represents 86% of the power loss in the trachea, which agrees with the above-mentioned observations that the frictional losses dominate following jet impingement in a healthy geometry. This value is elevated compared with all pathological cases. The frictional value is higher than the equivalent pipe flow loss ('Poiseuille flow loss', interpreted loosely), as suggested by Pedley et al. (1970), as their analysis neglected the losses due to the glottal jet and assumed a uniform tracheal diameter.

Case P1 shows the frictional losses represent nearly half the total loss (49%). This geometry has a particularly long constriction region due to goitre compression that elevates frictional losses; however as described above the expansion and curvature in the distal regions reduces frictional losses. The 'S' shaped non-

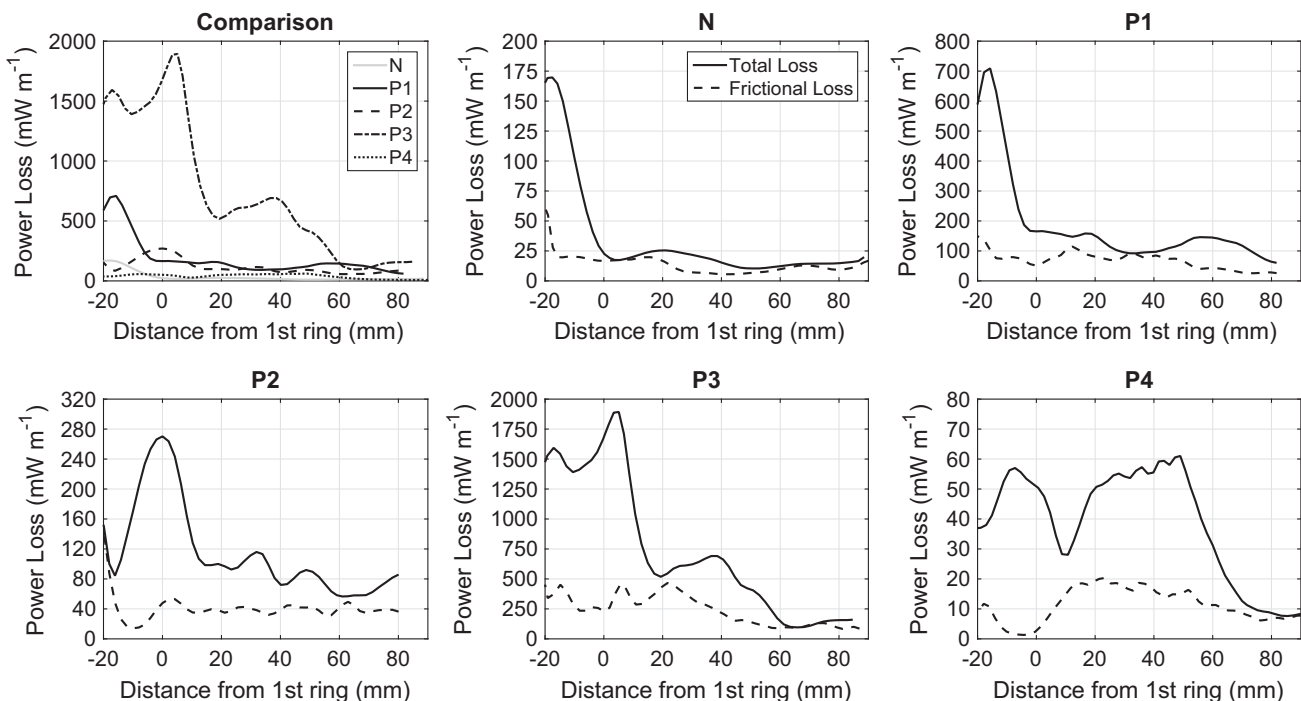


Fig. 4. Total and frictional power losses per metre along the length of each trachea.

compressed geometries (cases P2 and P4) show low frictional losses (21%), suggesting that non-compressed geometries are more susceptible to unsteadiness. Case P3 shows the highest overall loss, for the reasons given in Section 3.1. Whilst the frictional loss is just under a third of the total loss, it is much larger in the post-constriction section of the trachea. This case also contains the narrowest constriction of the considered geometries and hence gives the highest frictional loss of all the cases, as the wall shear stress is elevated through the high velocity, constricted section.

Comparison of these values for the power required to drive flow through the trachea with values in the literature for the work of breathing for a normal case (of order 30 mW to overcome air-flow resistance and of the order 300–350 mW for the total work rate (Milic-Emili and Petit, 1960; Loring et al., 2009)) reveal that the tracheal component of power loss becomes significant in pathological cases. It is also clear that elevated tracheal loss in the pathological cases raises the total work of breathing significantly. The impact of this increase for the subjects will depend on their ability to meet this increased power demand.

3.3. Unsteadiness

Many of the factors contributing to the interior power losses are due to the unsteadiness of the flow, for example, jets oscillate, separation points move and recirculation zones move. Therefore, it is revealing to quantify the unsteadiness of the flow through velocity fluctuations. Mapping the locations with high unsteadiness within the airway allows the mechanisms of these losses to be identified.

Fig. 5(a) details U_{rms} for case N, P1 and P2. These cases are chosen as they represent the three types of geometry considered in this study, N is healthy, P1 is an example of a constricted and deviated geometry, whereas P2 is only deviated. In the normal case we observe that the flow fluctuates in the post glottal region and then stabilises, which agrees with Fig. 4(a) where interior flow losses dominate in the post glottal region and then decay. For Case P1, we can make similar observations in the post-glottal region; however, due to the constriction in the geometry (location) the flow accelerates and unsteadiness reduces in the streamwise direction. Case P1 is also deviated by the goitre, this causes the high velocity fluid to separate and move towards the outer wall in the expansion region; the jet fluctuates in this region. The band of very low velocity fluctuation between these two elevated regions is the location where the frictional losses represent a high proportion of the total power loss (Fig. 4(b)). Finally, for case P2, we observe that U_{rms} is elevated throughout the geometry. The 'diffused' nature of the unsteadiness is caused by the highly distorted geometry.

Our analysis demonstrates that although the effect of the goitre on cases P1 and P2 was considerably different (i.e. one case distorted throughout and the other constricted and locally distorted), the flow in both cases was highly unsteady/ weakly turbulent compared to the normal case.

Table 2

Power losses during inhalation at 30 l/min from the first tracheal ring to the carina for the five chosen cases.

Case	Overall total power loss ($\times 10^{-5}$ W)	Overall frictional loss ($\times 10^{-5}$ W)	Ratio of losses (%)
N	101	87	86
P1	814	400	49
P2	809	166	21
P3	4674	1355	29
P4	293	63	21

A relationship can be identified between these patterns of unsteadiness and the results given in Table 2 and Fig. 4. Where the unsteadiness is low, the frictional losses dominate, for example throughout the trachea in case N or the constricted band in case P1, whilst regions with high interior flow losses correlate with regions of high unsteadiness. The implications of these findings are significant to both future CFD models, in showing that steady-state simulations are insufficient even with constant boundary conditions, and clinically, as discussed in Section 3.4.

3.4. Clinical significance

Currently, the guideline provided by the American Thyroid Association only takes into account a simple measurement of diameter reduction. Our study highlights that this metric fails to characterise losses on two of the four pathological cases. The use of power loss as a metric for understanding breathing difficulties readily allows both constricted and deviated cases to be identified. We have shown that a constricted trachea contributes to both elevation of wall-derived frictional losses over that of a healthy pathology and also raises other frictional losses downstream of the constriction e.g. due to viscous interaction. However, the more interesting observation here is that a deviated geometry can be of equal importance. For example, case P2 displays only a natural level of constriction (similar to a normal case), however, due to its curved nature, the overall power loss was similar to case P1 (55% constricted). This result also highlights the importance of curvature for power loss in constricted pipes: the maximum curvature for P1 and P2 was 0.021 with the former having a single large peak in curvature at the location of constriction and the latter two large peaks in curvature.

Future translation into the clinical setting could be realised by measuring a patient's total airway resistance (e.g. body plethysmography for total airway resistance or forced oscillation

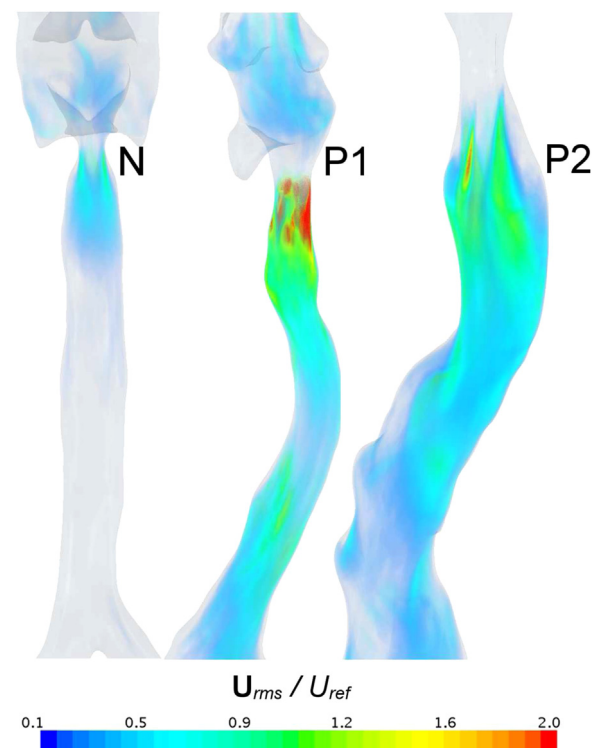


Fig. 5. Volume rendering of U_{rms} scaled by a reference velocity, U_{ref} , the mean velocity on a cross-sectional plane in the first tracheal ring. For cases N, P1 and P2, $U_{ref} = 2.19, 2.78, 2.59$ m/s respectively. P1 is an example of a constricted geometry and is similar to P3, whereas P2 is a curved geometry and is similar to P4.

technique for total lung resistance (Kaminsky, 2012)) or work of breathing (Grinnan and Truwit, 2005; Powell and Williams, 2012) and combining this with the CFD determined tracheal resistance or power loss in the trachea could establish the relative contribution of the tracheal component.

4. Conclusion

This study has demonstrated significant differences in the magnitude and mechanisms of power loss in normal tracheas and in those where the normal geometry is significantly altered by goitre. In normal tracheas, the pattern of energy loss corresponds to two distinct zones. Interior flow energy losses dominate in the region of the glottal jet, but quickly reduce downstream of jet impingement. Distal to the impingement site, frictional losses at the wall account for the majority of the losses, specifically 86% of the losses below the first tracheal ring in this case.

In the pathological cases, the ratio of power loss due to the wall friction is significantly reduced, as low as 21% in the cases considered. The total losses are also elevated. These differences are due to a range of different underlying mechanisms.

The first mechanism is found in cases where the tracheal lumen has been constricted by the goitre (cases P1 and P3). In these cases, the constriction causes elevated frictional losses that rapidly diminish as the geometry expands downstream of the constriction and increased interior flow losses arise due to separation.

The second mechanism is associated with deviation. Cases P2 and P4 exhibit deviation from the normal path of a trachea, but are not constricted. Here, the frictional losses are less highly elevated, however the interior losses are far higher than in the normal case. These losses occur due to the repeated disturbances imposed by the highly distorted geometry on the flow, resulting in successive separation zones.

The ratio of frictional to total losses can be seen as a measure of the relative intensity of the interior flow dynamics, so that a low ratio of frictional to total loss should correspond to more vigorous fluctuations in the flow. Fig. 5 shows the increased unsteadiness in the flow in examples of both types of pathology (P1 and P2). The prolonged zones of high fluctuating velocity in the pathological cases are in marked contrast to that in the normal anatomy.

The contribution of the tracheal component to the total airway flow resistance can rise from a small fraction of the total in normal subjects (of order 5%, comparing the normal case N with the values given by Loring et al. (2009)) to several times that of the normal total flow resistance in certain pathological cases (e.g. case P3). These increases in pathological cases can have important clinical consequences.

Conflict of interest statement

We wish to confirm that there are no known conflicts of interest associated with this publication.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.jbiomech.2015.11.033> and on request to rdm-enquiries@imperial.ac.uk

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