Modeling expiratory flow from excised tracheal tube laws

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Aljuri, Nikolai, Lutz Freitag, and José G. Venegas. Modeling expiratory flow from excised tracheal tube laws. J. Appl. Physiol. 87(5): 1973–1980, 1999.—Flow limitation during forced exhalation and gas trapping during high-frequency ventilation are affected by upstream viscous losses and by the relationship between transmural pressure (Ptm) and cross-sectional area (Atr) of the airways, i.e., tube law (TL). Our objective was to test the validity of a simple lumped-parameter model of expiratory flow limitation, including the measured TL, static pressure recovery, and upstream viscous losses. To accomplish this objective, we assessed the TLs of various excised animal tracheae in controlled conditions of quasi-static (no flow) and steady forced expiratory flow. Atr was measured from digitized images of inner tracheal walls delineated by transillumination at an axial location defining the minimal area during forced expiratory flow. Tracheal TLs followed closely the exponential form proposed by Shapiro (A. H. Shapiro, J. Biomech. Eng. 99: 126–147, 1977) for elastic tubes: Ptm = Kp [(Atr/Ar0)−n − 1], where Ar0 is Atr at Ptm = 0 and Kp is a parametric factor related to the stiffness of the tube wall. Using these TLs, we found that the simple model of expiratory flow limitation described well the experimental data. Independent of upstream resistance, all tracheae with an exponent n < 2 experienced flow limitation, whereas a trachea with n > 2 did not. Upstream viscous losses, as expected, reduced maximal expiratory flow. The TL measured under steady-flow conditions was stiffer than that measured under expiratory no-flow conditions, only if a significant static pressure recovery from the choke point to atmosphere was assumed in the measurement.

Flow limitation; tracheal cross-sectional area and collapse; wave speed

Theoretical and experimental studies have shown that forced expiratory flow becomes limited when gas velocity reaches the local wave speed at a point along the airway (1, 2). Such wave speed is a function of the airway’s transmural pressure-area relationship (also referred as tube law [TL]) and gas density (10). At high lung volumes, wave speed is generally reached within the trachea or the main bronchi. On theoretical grounds, it has been proposed that, depending on upstream viscous losses, expiratory flow could become limited at gas velocities lower than the corresponding local wave speeds (7, 10). Furthermore, if the tracheal TL is such that, with increasing effort local, wave speed increases faster than gas velocity, then flow might never become limited by the trachea. Under those conditions, flow might still become limited, but the limiting segment (also called the choke point) would occur at a more compliant peripheral location. Therefore, depending on the morphology of the tracheal TL and the magnitude of the upstream viscous losses, tracheal expiratory flow (Q) at maximal effort could have three different behaviors: limited by wave speed, limited by viscosity, or not limited. Given this dependency, we set out to test the theoretical relationship between choke point TL and expiratory flow limitation using excised animal tracheae of various stiffness and under various degrees of upstream airway resistance (Raw).

To isolate the flow limitation phenomena from unsteady changes in lung recoil and Raw that take place as a result of lung deflation, we used excised tracheae mounted in an apparatus that simulated a constant Raw and allowed quasi-steady levels of alveolar (PA) and pleural (Ppl) pressures.

Contrary to general belief, all the measured expiratory flows obtained during forced expiration were found to be higher than the theoretical maximal expiratory flows derived from the TL measured under static conditions. A possible explanation for this disagreement, as some investigators have pointed out (11, 12), is that increased wall tension caused by downstream migration of the choke point during expiration would effectively result in a choke point TL that would be stiffer than that measured at the same point under the no-flow condition. As a result, higher wave speeds and expiratory flows than those predicted by a TL measured under no-flow (static) conditions could be expected. With this in mind, we imaged the cross section of the trachea at the choke point and estimated the tracheal TL with digital planimetry under static and forced expiratory flow conditions with the assumption that the pressure downstream of the choke point was equal to atmospheric pressure (4, 10). However, we found that most forced expiratory flows measured were still higher than the theoretical maximal flows derived from the TL assessed when static pressure recovery downstream of the point of minimal cross-sectional area (Atr) was neglected. Therefore, we considered two additional hypotheses: 1) that the area expansion downstream of the choke point was sudden, with boundary layer separation and conservation of momentum and 2) that the area expansion occurred gradually with neglect of viscous losses and conservation of energy.

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1974 MODELING OF MAXIMAL EXPIRATORY FLOWS

Glossary

\[ A_{tr} \quad \text{Tracheal cross-sectional area} \]
\[ A_{tr0} \quad \text{A}_{tr} \text{ at } P_{tr} = 0 \]
\[ \alpha \quad \text{Normalized cross-sectional area} \]
\[ C \quad \text{Wave speed} \]
\[ U \quad \text{Flow velocity} \]
\[ S \quad \text{Speed index} \]
\[ Q \quad \text{Expiratory flow} \]
\[ P_{tm} \quad \text{Transmural pressure} \]
\[ P_A \quad \text{Alveolar pressure} \]
\[ P_{in} \quad \text{Choke point pressure} \]
\[ K_f \quad \text{Friction factor} \]
\[ \rho \quad \text{Gas density} \]
\[ TL \quad \text{Tube law} \]
\[ STL \quad \text{Static TL} \]
\[ DTL \quad \text{Dynamic TL} \]
\[ \beta \quad \text{Pressure recovery term} \]
\[ \text{Raw} \quad \text{Upstream airway resistance} \]
\[ MR \quad \text{Moderate Raw} \]
\[ HR \quad \text{High Raw} \]
\[ K_p,n \quad \text{Parametric factors related to the stiffness of the tube wall} \]

THEORETICAL CONSIDERATIONS

Total static pressure loss. Consider the trachea as a collapsible tube and that all upstream pressure losses from the alveoli to the carina can be attributed to a single resistance \( \text{Raw} \). For simplicity, let us assume that there is no elastic recoil; thus \( P_A = P_{pl} \). Also, disregard static pressure recovery from the point of minimal cross-sectional area (choke point) to atmosphere; then the static pressure in the choke point \( P \) equals atmospheric pressure \( P_A \). Accordingly, the overall pressure drop or driving pressure is equal to

\[ P_A = P_{up} + (1 + K_f) \frac{\rho U^2}{2} \]  (1)

where \( P_{up} = \text{Raw} \cdot \dot{Q}^2 \) is assumed to represent the upstream pressure loss, \( U \) is the air velocity, \( \rho \) is the density of air, and \( K_f \) is a turbulent friction factor that accounts for the local losses during the contraction of flow along the collapsing segment and is a function of tube geometry and Reynolds number. From Eq. 1, we may obtain

\[ \dot{Q} = A_{tr} \cdot U = A_{tr} \cdot \sqrt{\frac{2P_A}{\rho L}} \]  (2)

where the total loss factor \( L \) may be calculated as

\[ L = 1 + K_f + 2 \frac{A_{tr}^2 \text{Raw}}{\rho} \]  (3)

With the aforementioned assumptions, the \( P_{tm} \) difference is \( P_{in} - P_{pl} = P_A \). A local TL, representing the relationship between \( P_{tm} \) and \( A_{tr} \), normalized by the cross-sectional area at \( P_{tm} = 0 \) \( (A_{tr0}) \), is assumed to follow the relationship proposed by Shapiro (10)

\[ P_{tm} = K_p \cdot (\alpha^{-n} - 1) \]  (4)

where \( \alpha = (A_{tr}/A_{tr0}) \) and \( K_p \) and \( n \) are parametric constants related to the stiffness of the tube wall.

As shown by Shapiro (10), combining Eqs. 4 and 2 and setting \( \dot{Q} \cdot \partial P_{tm} = 0 \) it can be shown that for frictionless flow \( (K_f = 0, \text{Raw} = 0) \) the maximal flow rate that can pass through the airway segment is the product of \( A_{tr} \) times the local speed of propagation of long pressure waves \( (C) \) that depends on the gas density \( (\rho) \) and the TL and can be calculated as

\[ \dot{Q}_{max} = A_{tr} \cdot C = A_{tr} \cdot \sqrt{\frac{\alpha}{\rho} \cdot \frac{\partial P_{tm}}{\partial \alpha}} \]  (5)

In the presence of friction \( (K_f = (1/4) f > 0) \), if it is assumed by geometric similarity that the effective friction length \( (l) \) is proportional to the effective diameter \( (d) \) of the cross section and the friction coefficient \( (f) \) is a weak function of the Reynolds number, it can be shown by combining Eqs. 4 and 2 and setting \( \dot{Q} \cdot \partial P_{tm} = 0 \) that maximal flow \( (Q_{max}) \) would be reached at gas velocities \( (U_{crit}) \) lower than the local \( C \), where

\[ U_{crit} = \frac{1}{\sqrt{1 + K_f}} \cdot C \]  (6)

and thus

\[ \dot{Q}_{max} = A_{tr} \cdot U_{crit} \]  (7)

Once \( \dot{Q} \) has reached \( Q_{max} \) additional driving and transmural pressures can be applied before \( U \) reaches \( C \). This result, for a theoretical TL with \( n < 2 \), \( A_{tr} \) decreases more than \( C \) increases, creating a zone of negative effort dependency. Once \( U \) reaches \( C \) \( (\text{speed factor} (S) = U/C = 1; \text{Fig. 1}) \), further increases in \( P_A \) will no longer affect \( U \), since information cannot propagate upstream faster than \( C \) (1). In addition, the solution of Eq. 2 \( (\text{Fig. 1}) \) predicting a continued drop in \( U \) with \( P_A \) and \( S > 1 \) for the choke point should be no longer valid.

For the theoretical TL used, the curve of \( Q_{max} \) vs. \( P_A \) determined by solutions of Eq. 6 \( (\text{or Eq. 5 in the frictionless case}) \) is monotonically decreasing for increasing \( P_A \). Thus, for increasing values of \( \text{Raw} \), a lower \( Q_{max} \) \( (\text{or } Q_{max} = C \cdot A_{tr} \text{ for } K_f = 0) \) is reached while greater \( P_A \) is required.

The dependency of \( Q_{max} \) on the sole parameter \( n \) can be best illustrated for the case of \( \text{Raw} = 0 \) after substitution of Eq. 4 into Eq. 5 and normalization by the square root of \( (1 + K_f) \cdot \rho/(2 \cdot K_p \cdot A_{tr0}^2) \) as

\[ \sqrt{\frac{(1 + K_f) \cdot L}{2K_p A_{tr0}^2}} \cdot \dot{Q}_{max} = \sqrt{n \cdot \frac{2}{2 - n}} \cdot \frac{1}{2 - n} \]  (8)

showing that \( Q_{max} \) is real and finite only for \( 0 < n < 2 \). If \( \text{Raw} \) is included, numerical solutions of this theoretical
Q˙* show that, for a larger value of Raw, Q ˙* is always smaller (Fig. 2).

Including static pressure recovery. If the air flowing through a collapsible tube is treated as steady and one-dimensional and it is assumed that downstream from the choke point the expansion is sudden, boundary layer separation occurs until the flow gradually returns to a fully developed condition. For this particular flow, conservation of mass requires that

\[ A_{tr0} \cdot U_0 = A_U \cdot U \] (9)

where \( U \) is the air velocity at the choke point, and \( U_0 \) is the velocity at the point along the tube where the airflow returns to a fully developed condition. If it is further assumed that the average shear stress on the wall can be neglected because the fluid motion near the wall is very sluggish and random, the momentum equation can be written as

\[ A_{tr0} \cdot (P_{in} - P_0) = \rho A_U \cdot (U_0 - U) \] (10)

where \( P_{in} \) is the pressure at the choke point and \( P_0 \) is the pressure where the flow returns to a fully developed condition. Combination of Eqs. 9 and 10 yields

\[ P_{in} - P_0 = -\frac{\rho U^2}{2} \left( 2\alpha - 2\alpha^2 \right) \] (11)

If we assume, however, that the expansion downstream from the choke point occurs gradually and disregard any viscous losses, then conservation of energy requires that

\[ P_{in} + \frac{\rho U^2}{2} = P_0 + \frac{\rho U_0^2}{2} \] (12)

and combination of Eqs. 9 and 12 yields

\[ P_{in} - P_0 = -\frac{\rho U^2}{2} \left( 1 - \alpha^2 \right) \] (13)

Accordingly, depending on the assumption regarding the character of the area downstream of the choke point and assuming further that the upstream viscous losses are represented by a more realistic relationship (8)

\[ P_{up} = Raw_1 \cdot \dot{Q} + Raw_2 \cdot \dot{Q}^2 \] (14)

Equation 1 can be written as

\[ P_{A} = Raw_1 \cdot \dot{Q} + Raw_2 \cdot \dot{Q}^2 + (1 + K_f - \beta) \frac{\rho U^2}{2} \] (15)

where the parameter \( \beta \) corresponds to the fraction of the stagnation pressure recovered by the flow along the expansion downstream of the choke point: \( \beta = 2\alpha - 2\alpha^2 \) for a sudden area expansion leading to boundary layer separation, which we will refer to as minimal pressure recovery, and \( \beta = 1 - \alpha^2 \) for total static pressure recovery.

**Fig. 2.** Plots of \( \dot{Q}_{max} = C \cdot A_{tr} / \sqrt{1 + K_f} \) and experimental \( \dot{Q}_{max} \) normalized by \( \sqrt{1 + K_f} \cdot \rho / (2K_p \cdot A_{tr0}^2) \) vs. parameter \( n \) of Eq. 4. Dotted line, theoretical with no Raw; dashed line, with moderate Raw (MR); solid line, with high Raw (HR); \( x \), experimental with MR; \( \bullet \), with HR.
Finally, solving for $Q\dot{}$ in Eq. 15 after substitution of Eq. 16 yields
\begin{equation}
\dot{Q} = A_{tr} \cdot U
\end{equation}

\begin{equation}
\dot{Q} = A_{tr} \cdot \left[ -\frac{\text{Raw}_1 A_{tr}}{\rho L} + \sqrt{\frac{\text{Raw}_1 A_{tr}}{\rho L}} + \frac{2\text{PA}}{\rho L} \right]
\end{equation}

where $L$ is
\begin{equation}
L = (1 - \beta) + K_f + 2 \frac{A_{tr}^2 \text{Raw}_2}{\rho}
\end{equation}

\section*{METHODS}

Experimental preparation. Approval of the Animal Care Committee of the Massachusetts General Hospital was obtained for use of the animals and the protocol used for killing them. Tracheae from three dogs and two sheep were excised after the animals were killed with an intravenous mixture of KCl and pentobarbital sodium. The tracheae were cleaned from surrounding tissues and kept refrigerated for $<24$ h before the beginning of the study. The tracheae were suspended on an experimental apparatus that simulated upstream viscous losses and Ptm in static and dynamic conditions (Fig. 3). The tracheae were supported in the vertical position inside a clear Plexiglas cylindrical chamber (4 cm ID) that could be pressurized to simulate Ppl. The ends of the tracheae were mounted over and secured to thin-walled aluminum (laryngeal and carinal) fittings tailored to the largest possible diameter. The laryngeal fitting was connected to the smaller opening of a conical funnel and then to atmosphere via a 2-cm-ID side branch. A glass window covered the larger opening of the funnel, providing an axial view of the inner walls of the trachea. The carinal fitting was connected to one or two flow resistors in series made of packed 0.5-mm-ID 10-cm-long glass tubes for moderate resistance (MR) and high resistance (HR), respectively. The free end of the resistors was then connected to a 16-gallon rigid tank that was fed with air via an adjustable pressure regulator. To allow unobstructed view over its entire length, a minimal longitudinal tension was applied to the trachea by adjusting the distance between tracheal fittings just enough to overcome any natural bending.

Pressure transducers were connected with short stiff tubes (2 cm) to the tank, the tracheal supporting chamber, and the windowed funnel to measure the equivalent of $PA$, Ppl, and airway opening stagnation pressure, respectively. The alveolar tank and the pleural chamber were interconnected to maintain equal pressures during the forced expiratory maneuvers. $Q$ was measured at the exit of the funnel’s side branch with a pneumotachograph (Jaeger, Wurzburg, Germany) and a Valadyne differential pressure transducer. The flow and pressure signals were recorded digitally in a personal computer.

The pressure losses from the resistors were characterized by using in the apparatus a stiff tube instead of the trachea and fitting the experimentally measured $PA$-$Q\dot{}$ relation to Eq. 8. This fitting gave the parameters Raw$1 = 1.6$ and Raw$2 = 1.0$ for a single resistor (MR) and Raw$1 = 2.1$ and Raw$2 = 2.1$ for two resistors (HR).

Assessment of $A_{tr}$. The inner tracheal walls were recorded through the glass window with a black-and-white videocamera equipped with a zoom lens. To delineate the walls of a selected cross section of the trachea, a high-intensity flash ring placed around the supporting chamber was pulsed across a 2-mm-wide circular slit (Fig. 4). The axial location of the imaged cross section was selected during steady $Q$ by advancing the illuminated band along the axis of the trachea to the point showing the lowest $A_{tr}$, which should be close to the choke point. This location was left unchanged for the rest of the study.

Acquired video images were digitized with a frame grabber and stored and processed in an Amiga 1000 computer. $A_{tr}$ was calculated from those images by digital planimetry with a computer program that counted the pixels inside the illuminated walls. Area measurements were calibrated by simultaneously imaging, under identical conditions, a drawing of a 1-cm-side square placed at the same level of the lighted section.
Static measurements of choke point $A_{tr}$ were done by applying progressive levels of negative pressures to the inner walls of the trachea while the carinal end was kept closed. Measurements of $A_{tr}$, normalized by $A_{tr0}$ ($\alpha = A_{tr}/A_{tr0}$) were plotted against measured $P_{tm}$ and fitted by Eq. 4 (Fig. 5), yielding parameter values for $K_f$ and $n$.

During forced expiratory maneuvers, $A_{tr}$ was determined at incremental levels of $P_A$ during simultaneous recording of the pressures and $Q$ signals. For each $A_{tr}$ imaged, $P_{tm}$ was first calculated using Eq. 16 for three possible static pressure recovery conditions, no ($\beta = 0$), minimal ($\beta = 2\alpha - 2\alpha^2$), and total static pressure recovery ($\beta = 1 - \alpha^2$), and then fitted by Eq. 4 to yield the corresponding TL parameters during quasi-steady $Q$.

Plots of $Q$ vs. $P_A$ were fitted by Eq. 17 with use of measured values of $\alpha$ by adjusting $K_f$ for each of the static pressure recovery conditions mentioned above as shown in Fig. 6A in the trachea of dog 3 with MR. Such a fitting yielded $K_f > 0$, which minimized the mean squared error between the fitted model and experimental data of $Q$, as shown in Fig. 6B for the same trachea.

RESULTS

Static and dynamic TLs. Equation 4 provided excellent curve fitting to the experimental measurements of $\alpha$ (mean $r^2 = 0.99$). There was, however, substantial variability in $K_f$ (range 7.1–36) and $n$ (range 0.32–2.1) between the different tracheae. Dynamic TLs (DTLs) were stiffer than static TLs (STLs) for the case of total recovery ($\beta = 1 - \alpha^2$). In partial or no recovery, DTLs were, in some cases, more compliant, as shown by the parameter $n$ in Table 1.

TLs and flow limitation. We found that the fitting of the model with the assumption of minimal (mean $r^2 = 0.81$) and no pressure recovery (mean $r^2 = 0.71$) was poorer than that with the assumption of total recovery, which yielded excellent fitting (mean $r^2 = 0.996$; Fig. 7A). The poor fitting for the minimal or no-pressure recovery assumption was caused by a consistent underestimation of $Q$, despite a consistently smaller $K_f$ than that found for the total pressure assumption (Fig. 7B).

We also found that all tracheae showing STLs and DTLs with $n < 2$ exhibited a plateau in the $Q$ vs. $P_A$ plot for MR and HR, reflecting $Q$ limitation. The trachea from sheep 2 had an STL with $n = 2.1$ and did not exhibit a plateau at MR or HR (Fig. 8). In all tracheae, an increasing $Raw$ decreased $Q$ for all driving pressures, including $Q_{\text{max}}^\text{raw}$. In all cases with $n < 2$ except one (dog 3 with HR), $Q$ exceeded theoretical $Q_{\text{max}}^\text{raw}$ derived from Eq. 5 with the STL. When the stiffer DTL assuming total recovery was used, $Q$ always reached a maximum for $S < 1$.

DISCUSSION

We formulated a theoretical model to test the relationship between choke point TL and maximal $Q$ measured from excised animal tracheae at various degrees of...
For this purpose, we developed a simple experimental apparatus able to generate the required Ptm across the trachea while simultaneously allowing accurate imaging of the choke point. Because of the limited number of tracheae, one cannot characterize the elastic properties of dog or sheep tracheae. However, given the substantial (mechanical) differences among the five tracheae studied (Fig. 5) and the excellent fitting obtained with the model in all conditions, this limited number of studies gives sufficient evidence for the validity of the model relating TLs and flow limitation.

Jones et al. (4) used a similar imaging technique with a fiber-optic bronchoscope to measure Atr. This technique enabled them to assess the effect of time on airway compliance and to estimate the maximum flow from the measured TL of the compressed airway segment. Their study already suggested that this TL could determine maximum flow and, therefore, pointed out the importance of the experimental assessment of the airway’s TL.

The most important findings of our study are as follows: TLs of isolated tracheae followed quite well the mathematical representation proposed by Shapiro (10) for elastic tubes (Eq. 4). The TL assessed under quasi-steady flow conditions (DTL) was stiffer than the TL measured under quasi-steady no-flow conditions (STL) only if a significant pressure recovery was assumed. Only under these conditions could the measured Q be higher than those predicted by a STL be explained. We thus concluded that static pressure recovery from the choke point to atmosphere must have been substantial. Furthermore, we found that upstream viscous losses, represented by Raw, did affect maximal Q in these isolated tracheae.

Because our imaging technique allows us to assess the TL at a single axial location, we cannot rule out the possibility that the point of maximum Atr collapse during forced exhalation might have migrated axially, depending on the applied driving pressure. However, because the high-intensity flash ring used to define the inner walls of the trachea illuminated a relatively wide (~10-mm) section of the tracheal wall, it is likely that small migrations of the maximal collapse point did not affect our results, since only the minimal cross section was captured by the camera. The choke point TL was assessed only during progressively increasing driving pressure until the highest quasi-steady Q with our apparatus was obtained. Significant

![Fig. 7](http://jap.physiology.org/doi/10.2203.3.6)

**A** Logarithmic plots of minima of mean squared errors between fitted model and experimental data of Q in all tracheae for cases of no, minimal, and total static pressure recovery.

**B** Comparison between fitted model and experimental data of maximal Q for cases of no (*), minimal (○), and total static pressure recovery (†).

<table>
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Table 1. Values of Kp and n obtained from fitting of experimental Atr and Ptm to Eq. 4 with corresponding r² for all tracheae.
Hysteresis was observed during the decreasing phase of the flow, with lower flows for the same driving pressure, an observation described previously by Knudson and Knudson (6).

We found that in all cases studied the TL with \( A_{tr} \) measured during flow conditions and calculated with the assumption of no static pressure recovery was more compliant than the STL. This behavior could not be explained by any effects of longitudinal tension (11, 12), an unstable configuration of the flow-limiting segment during the transient (7), or effects of time on the change in \( A_{tr} \) (4). All these explanations would lead to the opposite result: a stiffer TL measured under flow conditions (DTL). We have to conclude that the assumption of \( P_{tm} = -P_A \) is erroneous. A fact supporting the hypothesis that a DTL should be stiffer than the STL is that, according to the wave-speed theory, the maximum frictionless flow that can pass through an airway segment is given by \( C \cdot A_{tr} \). In most of our experiments, \( Q \) exceeded this limit when the STL was used to derive \( C \) but never when the stiffer DTL, estimated assuming total pressure recovery, was used.

We also found that the model described by Eq. 17 could not describe the experimental data unless a significant fraction of the static pressure is recovered downstream of the choke point. Even for \( K_t \) set to 0, the calculated \( Q \) with the assumption of minimal recovery underestimated the observed \( Q \) (Fig. 7). Only for the case where total static pressure recovery was assumed, positive values of \( K_t \) were found and the model predicted well the experimental data. We cannot rule out and it is, in fact, likely that a fraction of the static pressure may have been lost downstream from the choke point. This would imply that our values of \( K_t \) were somewhat overestimated but still yield and excellent fit, since the pressure loss term includes the parameters \( \beta \) and \( K_t \) with opposite signs (Eq. 18). On the other hand, if one assumes a gradual contraction angle (30–60°), \( K_t \) should lie between 0.02 and 0.07 (3), and \( \beta \) can then be calculated by substituting \( K_t \) into Eq. 17 and solving for \( \beta \). Figure 9 shows a representative case for this calculated \( \beta \) with these two values of \( K_t \) plotted together with the theoretical predicted bounds for the cases of total and minimal static pressure recovery. These results agree with those of Kececioglu et al. (5), who found that \( \beta \) generally fell between the limiting curves of total pressure recovery and minimal pressure recovery for short and intermediate shocks for collapsible penrose tubes. We found, however, that the calculated values of \( \beta \) fell out of the theoretical predicted bounds for \( \alpha > 0.5 \). This is probably because friction losses due to tracheal collapse are insignificant compared with the other pressure drops in the system at these low levels of collapse. Thus, because the actual dependence of \( \beta \) on \( \alpha \) is not known and may vary from

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**Fig. 8.** Plots of fitted model and experimental data of \( Q \) vs. \( P_A \) in sheep 1 (A) and sheep 2 (B). Dotted lines, experimental \( Q \) with MR and HR. Fitted model of \( Q \) with use of experimental \( A_{tr} \) and corresponding \( P_A \) with assumption of total pressure recovery with MR (●) and HR (○) is shown. Also shown is fitted model of \( Q \) with use of fitted relationship of \( A_{tr} \) and \( P_A \) with assumption of total pressure recovery with MR (solid line) and HR (dashed line). Theoretical maximal frictionless expiratory flow \( (Q_{max} = C_{STL} \cdot A_{tr}) \) is derived from static tube law (STL; +), and theoretical maximal frictionless expiratory flow \( (Q_{max} = C_{DTL} \cdot A_{tr}) \) is derived from stiffer dynamic tube law (DTL) estimated assuming total pressure recovery with MR (○) and HR (×).

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**Fig. 9.** Plots of \( \beta \) vs. \( \alpha \) in trachea of sheep 1 with MR. Solid line, theoretical predicted bound for case of total static pressure recovery; dashed line, theoretical predicted bound for case of minimal static pressure recovery. \( \beta \) was derived from Eq. 17 for \( K_t = 0.02 \) (○) and \( K_t = 0.07 \) (×).
trachea to trachea, it is not possible for this model to predict $Q$ on the basis of values of $K_r$, estimated from first principles. It seems clear nonetheless that the static pressure from the choke point to atmosphere is not totally lost, as assumed previously (1, 2, 7, 8, 10–12). The stiffness of the tracheal cartilaginous rings and longitudinal tension forces probably prevented a sharp transition of $A_{tr}$, allowing significant static pressure recovery.

Our experimental data agree qualitatively with the derivations of Shapiro (10) showing that for a very high Reynolds number a TL with $n < 2$ is required for flow limitation to occur. Lambert (7) also showed that, for wave-speed limitation to occur, $m$ (the reciprocal of $n$) should be $>0.5$. We found that all but one trachea had $n < 2$ in the STL and exhibited $Q$ limitation with MR and HR. The trachea of sheep 2 ($n = 2.1$) did not exhibit a plateau at MR or HR (Fig. 8). If an Raw is included in the simplified model proposed by Shapiro, then flow limitation still occurs for TLs with $0 < n < 2$ and $Q_{\text{max}}$ decreases as Raw is increased (Fig. 2). This theoretical result also agrees with our data showing that, in all cases, measured $Q_{\text{max}}$ decreases with increasing Raw. However, measured and theoretical $Q_{\text{max}}$ differ qualitatively, because this theoretical result does not include static pressure recovery. Excellent agreement could be achieved only if a substantial degree of static pressure recovery from the choke point to atmosphere took place, as discussed previously (Fig. 7B).

Elliot and Dawson (2) tested the wave-speed theory assuming frictionless $Q$ and found $Q_{\text{max}}$ values that were somewhat lower than $C \cdot A_{tr}$. Given that the experimental apparatus used by Elliot and Dawson did not include Raw, it is not surprising that the observed maximal $Q$ were found close to, although slightly lower than, $C \cdot A_{tr}$. Although they attributed this result to possible errors in the measurements of $A_{tr}$, the small departure of $Q_{\text{max}}$ from $C \cdot A_{tr}$ could have also been explained if $K_r$ was nonzero (Eq. 6). In our experiments, $Q$ always limited at values lower than $C \cdot A_{tr}$ because of the high upstream viscous losses generated by the MR and HR.

We conclude that an exponential form of the TL similar to that proposed by Shapiro (10) for collapsible tubes and Lambert (7) for airways characterized well the compliant properties of excised tracheae measured under static and flow conditions. Experimental data obtained from excised sheep and dog tracheae showed that flow limitation occurred only in tracheae with $n < 2$, whereas no flow limitation was reached for tracheae with $n > 2$. We developed a simple lumped-parameter model of $Q$ limitation that included the effects of upstream and choke point friction pressure losses. The model agrees with wave-speed theory in the limit when Raw and $K_r \rightarrow 0$ but predicts $Q_{\text{max}} < C \cdot A_{tr}$ for the more realistic cases where Raw and $K_r > 0$. This prediction is confirmed by experimental data. These data strongly suggest that a large fraction of the static pressure is recovered downstream of the choke point.

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