Good short-term agreement between measured and calculated tracheal pressure


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Background. Tracheal pressure (P_tr) is required to measure the resistance of the tracheal tube and the breathing circuit. P_tr can either be measured with a catheter or, alternatively, calculated from the pressure–flow data available from the ventilator.

Methods. Calculated P_tr was compared with measured P_tr during controlled ventilation and assisted spontaneous breathing in 18 healthy and surfactant-depleted piglets. Their lungs were ventilated using different flow patterns, tidal volumes (V_T) and levels of positive end-expiratory pressure.

Results. In terms of the root mean square error (RMS), indicating the average deviation of calculated from measured P_tr, the difference between calculated and measured P_tr was 0.6 cm H2O (95%CI 0.58–0.65) for volume-controlled ventilation; 0.73 cm H2O (0.72–0.75) for pressure support ventilation; and 0.78 cm H2O (0.75–0.80) for bi-level positive airway pressure ventilation.

Conclusion. The good agreement between calculated and measured P_tr during varying conditions, suggests that calculating P_tr could help setting the ventilator and choosing the appropriate level of support.


Keywords: lung; lung, tracheal pressure; ventilation, controlled mechanical; ventilation, spontaneous

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For inspiratory gas to enter the lungs, a pressure gradient between the alveoli and the airway opening is required. For inspiratory gas to traverse the tracheal tube a gradient between tracheal pressure (P_tr) and airway opening is required. It is this driving pressure difference that we are interested in for setting the ventilator, particularly in modes designed to assist spontaneous breathing. To create the pressure difference that drives gas into the lungs, the patient lowers P_tr, particularly if respiratory drive is high.1 The greater the flow and the smaller the tracheal tube diameter, the greater the driving pressure difference between airway opening and P_tr must be, to overcome the resistance of the tracheal tube. With flow patterns other than constant inspiratory flow, the relationship between the pressure at airway opening (P_prox) and P_tr is complex, and there is no simple rule to infer P_tr from P_prox at the bedside. The logical option is to either measure P_tr directly4–10 or to calculate P_tr non-invasively from flow and pressure data available from the ventilator.11–18 We have compared directly measured with calculated tracheal pressure under different conditions.

Materials and methods
The study followed NIH guidelines in the laboratories of the Department of Surgical Sciences/Section of Anaesthesiology and Critical Care Medicine, Uppsala University. The Local Ethics Committee for Animal Experimentation reviewed and approved the study. We
studied 18 healthy male and female piglets of Swedish landrace breed (25 (SD 2) kg). After the experiments the animals were killed with an overdose of potassium chloride.

**Study plan**

One group of piglets (n=12) was allocated to volume-control ventilation (VC) before and after broncho-alveolar lavage, to study whether lavage-induced changes of the impedance of the respiratory system would affect the reliability of the calculated $P_t$. We later added a second study with assisted spontaneous breathing. We felt that in clinical practice, tracheal pressure is more relevant with assisted ventilation than with VC, and that the complex flow patterns of assisted modes would be a more vigorous test of the value of the $P_t$ algorithm than the constant inspiratory flow of VC. Three healthy piglets therefore received pressure support ventilation (PS), and another three received bi-level positive airway pressure ventilation (BiPAP). Ventilation was given for 20 min before measurements were performed. The relationship between calculated and measured $P_t$ was assessed.

Tracheal pressure was calculated using two different methods: according to Guttmann and colleagues,\(^4\) henceforth called ‘calculated $P_{Rohrer}$’ and according to Jarreau and colleagues,\(^2\) henceforth called ‘calculated $P_{Blasius-Ito}$’ (see Appendix). When referring to calculated $P_t$, the simpler term ‘calculated $P_t$’ is used.

**Ventilator settings**

In the VC group, tidal volumes ($V_T$) of 150, 250, 300, 400, and 500 ml and either zero PEEP (healthy animals; group labelled ‘ZEEP’), or 12 cm H$_2$O PEEP (surfactant-deficient animals; group labelled ‘PEEP’) were consecutively applied. Ventilator frequency was 20 min$^{-1}$. In the PS group (healthy animals; ‘PS’), PEEP was set to 5 cm H$_2$O and the pressure support to 5 cm H$_2$O above PEEP level.

In the BiPAP group (healthy animals; ‘BiPAP’) the settings were: PEEP at 5 cm H$_2$O for 3 s; upper pressure level 10 cm H$_2$O for 3 s; set frequency 6 min$^{-1}$. $F_{O_2}$ was 0.5 throughout. In the PS and the BiPAP group, trigger was set to the maximal flow-trigger sensitivity.

**Respiratory measurements**

The animals’ lungs were ventilated through a tracheal tube (#7, Mallinckrodt, Athlone, Ireland), connected to a Servo 300 ventilator (Siemens-Elema, Solna, Sweden) by a 60 cm rigid tubing system. In the PS and BiPAP group, airway pressure and flow were continuously measured with a Fleisch pneumotachograph (Pneumotachometer Series 1110; Hans Rudolph, Kansas City, MO) placed between the tracheal tube and the Y-piece of the ventilator circuit. In the VC group, flow and airway pressure were measured with the Servo 300 built-in transducers. A comparison between flow/pressure data simultaneously recorded with the Fleisch head and the Servo 300 transducers showed excellent agreement during VC. Tracheal pressure was directly measured 2–3 cm above the carina with a catheter tip transducer (OML intracranial pressure monitoring kit, model 110-4B, Integra Neurosciences Camino, San Diego, CA), positioned outside the tracheal tube under direct inspection after the tracheostomy, before placing the tracheal tube. The Camino transducer measures pressure at the tip of the catheter, in an axial direction. End-inspiratory and end-expiratory hold manoeuvres (5 s) were also performed. Assuming that pressure would equilibrate within 5 s, the pressure values at the end of a 5-s hold were used to correct the measured $P_t$ data if necessary. If after a 5-s hold a pressure difference was detected, this difference was treated like an offset, and it was subtracted from the pressure readings of the Camino sensor.

All signals were sampled at 200 Hz, passed to a data acquisition system (AcqKnowledge, version 3.7.2, BioPac System, Inc., Santa Barbara, CA), and then exported to an MS-Excel worksheet for off-line analysis. At each measurement period, data were continuously sampled for 2 min. The measured $P_t$ was compared off-line to the calculated $P_t$ using the pressure–flow data acquired at the proximal outlet of the tracheal tube.

**Anaesthesia and fluid management**

After pre-medication (tiletamine 3 mg kg$^{-1}$, zolazepam 3 mg kg$^{-1}$, xylazine 2.2 mg kg$^{-1}$, atropine 0.04 mg kg$^{-1}$ intramuscularly), anaesthesia was induced with 500 mg ketamine, 0.5 mg atropine, and 20 mg morphine i.v.. Anaesthesia was maintained with ketamine infusion (20 mg kg$^{-1}$ h$^{-1}$) and morphine 0.5 mg kg$^{-1}$ h$^{-1}$, and neuromuscular block obtained by continuous infusion of pancuronium bromide (0.25 mg kg$^{-1}$ h$^{-1}$). The animals were given a solution of 4.5 g litre$^{-1}$ NaCl with 25 g litre$^{-1}$ glucose (Rehydrex, Pharmacia Infusion AB, Uppsala, Sweden) at 10 mg kg$^{-1}$ h$^{-1}$ and a 5 mg kg$^{-1}$ bolus of dextran-70 (Macrodex 70, Pharmacia Infusion AB, Uppsala, Sweden) to ensure normovolaemia.

**Lavage**

Lavage was done with 11 broncho-alveolar lavages (1.2–1.5 litre normal saline, corresponding to 50–60 ml kg$^{-1}$) which resulted in a $P_{aO_2}/F_{O_2}$ of less than 20 kPa. After lavage, the animals were allowed to stabilize for 20 min.

**Re-expansion**

Immediately after lavage the lungs were re-expanded by a 5-min period of pressure-controlled ventilation (PEEP 25 cm H$_2$O and a peak inspiratory airway pressure of 50 cm H$_2$O).
Data presentation

Results are presented as mean and 95% CI (95% lower to 95% upper) if not otherwise indicated. The agreement between measured and calculated $P_{tr}$ was assessed in terms of the root mean square error (RMS), giving the mean deviation for all data points under consideration. Bias and limits of agreement for distinct single pressure points were assessed according to the Bland and Altman method. The data were defined as follows. During VC: point 1 = 0.1 s past start of inspiration; point 2 = peak pressure; point 3 = 0.25 s past peak pressure; point 4 = 0.5 s past peak pressure; point 5 = 1 s past peak pressure; point 6 = last end-expiratory point. During BiPAP: point 1 = 0.2 s past start of inspiration; point 2 = peak pressure; point 3 = 0.2 s past peak pressure; point 4 = 0.15 s past start of expiration; point 5 = 0.25 s past start of expiration; point 6 = last end-expiratory point. During PS: point 1 = 0.10 s past start of inspiration; point 2 = peak pressure; point 3 = 0.10 s before end of inspiration; point 4 = 0.15 s past start of expiration; point 5 = 0.25 s past start of expiration; point 6 = last end-expiratory point. Statistical significance was assumed with $P \leq 0.05$.

Results

Eighteen animals were studied (VC, $n=12$; PS, $n=3$; BiPAP, $n=3$), and more than 2000 single breaths recorded, 759 of which were analysed. Fifty-seven breaths were excluded because of drifting of the transducer measuring $P_{tr}$. A further 300 breaths were not used because the tip of the $P_{tr}$ catheter touched the tracheal wall or moved during the respiratory cycle, leaving 402 breaths for the calculation according to $^4$ (VC, 277; PS, 81; BiPAP, 44). In 30 breaths for each group, the calculation of $P_{tr}$ was performed according to $^2$ ($P_{Blasius-Ito}$) and analysed for agreement with the results obtained by the method according to $^4$ ($P_{Rohrer}$).

Comparison of two methods to calculate tracheal pressure

The average deviation of $P_{Rohrer}$ $^4$ from $P_{Blasius-Ito}$ $^2$ was 0.3 cm H$_2$O (95% CI 0.15–0.4) over the whole respiratory cycle. Agreement was equally good for inspiration and expiration and for VC, PS, and BiPAP, respectively (see Figs 1–3).

Comparison of calculated and measured tracheal pressure $P_{Rohrer}$ (see Table 1, and Figs 1–3)

In terms of RMS, the mean difference between calculated $P_{tr}$ ($P_{Rohrer}$) – measured $P_{tr}$ was 0.6 cm H$_2$O (95% CI 0.58–0.65) for the breaths during VC with ZEEP; 0.6 cm H$_2$O (0.57–0.63) during VC with PEEP 12; 0.73 cm H$_2$O (0.72–0.75) during pressure support; and 0.78 cm H$_2$O (0.75–0.80) during BiPAP. During VC, the mean difference was the same in inspiration and expiration, whereas during the spontaneous breathing modes PS and BiPAP the deviation of $P_{Rohrer}$ was more during inspiration (0.93 cm H$_2$O (0.91–0.94) with PS, and 1.00 cm H$_2$O (0.97–1.04) with BiPAP) than during expiration.
Bland–Altman for $P_{\text{Rohrer}}$ (see Fig. 4 and Tables 2–5)

The comparison of single pressure points indicated good overall agreement, with the most bias during VC with ZEEP for the data point 0.25 s past peak in expiration ($-0.63$ cm H$_2$O ($-0.75$ to $-0.49$)) and the smallest bias for the pressure point 0.5 s past peak pressure ($-0.07$ cm H$_2$O ($-0.15$ to $-0.01$)). The situation was similar for VC with PEEP 12. The limits of agreement for VC (both without and with PEEP) in the worst case ranged from $-1.23$ to $1.53$ cm H$_2$O (peak inspiratory pressure, VC ZEEP) and in the best case from $-0.81$ to $0.79$ cm H$_2$O (1 s past peak pressure, VC 12 PEEP). Figure 4 shows Bland–Altman plots for six selected...
pressure points. For the spontaneous modes, bias and limits of agreement were greater for all pressure points under consideration with a maximal bias of 1.31 cm H2O (1.27–1.34) (0.1 s after start of inspiration during pressure support).

**Discussion**

We found that calculated tracheal pressure agreed well with measured tracheal pressure. Over the entire respiratory cycle, calculated $P_{Rohrer}$ was 0.6–1.0 cm H2O greater (RMS) than measured $P_{tr}$.

**Methodological considerations**

Some methodological problems could not be solved to our satisfaction.

First, the transducer for measuring $P_{tr}$ is not specifically designed for that purpose. The device is designed for measuring intracranial pressure and has a temperature-related drift, which in these animals, whose temperature was 37.5°C at start and sometimes became 41°C, could be important (the manufacturers manual indicates a temperature coefficient of 3 mm Hg over temperature range 22–38°C).

![Fig 4](image) Agreement between calculated and measured tracheal pressure ($P_{Rohrer}$ calculated according to 4) during VC with a PEEP of 12 cm H2O in surfactant-deficient animals. Bias and limits of agreement are indicated for distinct pressure points: 0.1 s after start of inspiration; peak airway pressure; 0.25 s past peak; 0.5 s past peak; 1 s past peak; last end-expiratory point.

**Table 1** Agreement between measured and calculated tracheal pressure ($P_{Rohrer}$ calculated according to 4) in terms of RMS indicating the average difference between measured and calculated $P_{Rohrer}$ over the entire respiratory cycle, and for inspiration and expiration separately. For number of animals and breaths see text. VC: volume-controlled ventilation; BiPAP: bi-level positive airway pressure ventilation. Values are mean (lower to upper 95% confidence interval) VC without PEEP.

<table>
<thead>
<tr>
<th>Mode</th>
<th>RMS (entire respiratory cycle) (cm H2O)</th>
<th>RMS (inspiration) (cm H2O)</th>
<th>RMS (expiration) (cm H2O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VC PEEP 0</td>
<td>0.61 (0.57–0.65)</td>
<td>0.59 (0.55–0.63)</td>
<td>0.61 (0.56–0.67)</td>
</tr>
<tr>
<td>VC PEEP 12</td>
<td>0.60 (0.57–0.62)</td>
<td>0.58 (0.54–0.62)</td>
<td>0.58 (0.55–0.62)</td>
</tr>
<tr>
<td>PS</td>
<td>0.73 (0.72–0.76)</td>
<td>0.93 (0.91–0.94)</td>
<td>0.57 (0.56–0.59)</td>
</tr>
<tr>
<td>BiPAP</td>
<td>0.78 (0.75–0.80)</td>
<td>1.00 (0.97–1.04)</td>
<td>0.64 (0.61–0.66)</td>
</tr>
</tbody>
</table>
As our concern was not the reliability of direct $P_{tr}$ measurement, we did not systematically screen all the measured $P_{tr}$ data for malposition and potential artefacts to determine the absolute number of erroneous measurements to which our current equipment is obviously prone. Despite the basic methodological concerns with direct $P_{tr}$ measurement discussed below, we know, that a dedicated tool for measuring $P_{tr}$ would probably have given better-measured $P_{tr}$ data.

The second concern was with the bias flow of the Servoventilator 300. Flow-triggering requires this bias flow of 40 ml s$^{-1}$. The algorithm does not discriminate between the main flow and the bias flow, and the sum of both is included in the calculation of $P_{Rohrer}$. During early inspiration, flow is high (>1500 ml s$^{-1}$) with PS or BiPAP, and the error introduced by the bias flow is relatively small. However, at end-expiration the flow rate is getting small and the relation between the bias flow and the entire flow increases. This can explain the weak agreement of measured and calculated $P_{tr}$ at some of the pressure points. As far as device-specific methods for regulating flow on the calculation of $P_{tr}$ can be taken into account, the present results can be extended to other types of ventilators, too.

Cardiogenic oscillations were another disturbing factor. Measurement of $P_{tr}$ was performed at the carina close to the beating heart. Calculation of $P_{tr}$, in contrast, uses pressure and flow data sampled at the proximal tracheal tube outlet, distant from the beating heart with the dampening effect of airways and tracheal tube interposed. Cardiogenic oscillation affects the measured $P_{tr}$ more than the calculated $P_{Rohrer}$, and an association between measured and calculated $P_{tr}$ data may be considered at the very moment when a heartbeat distorts the measured $P_{tr}$. At low lung volumes the heartbeats influence the measured $P_{tr}$ even more strongly, which can explain the weak association of some of the pressure points at end-expiration.

The combination of two studies, one with VC and an additional one with assisted spontaneous breathing, PS and BiPAP, comprising different numbers of animals is certainly open to statistical criticism. We justify this with our intention to test the agreement between calculated and measured $P_{tr}$ under an extended spectrum of flow patterns.

Finally, this is a short-term study only. A clinical validation would have to take long-term effects like progressive tracheal tube narrowing by secretions into account. An estimation of the potential error caused by tracheal tube narrowing is therefore added (see below).

### Table 2 Bias and limits of agreement according to Bland and Altman for different pressure points (calculated $\Delta P_{tr}$ measured tracheal pressure, $P_{Rohrer}$ calculated according to 4) during VC without PEEP

<table>
<thead>
<tr>
<th>Point (definition)</th>
<th>Bias (cm H$_2$O)</th>
<th>Limits of agreement (cm H$_2$O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Point 1 (0.1 s past start of inspiration)</td>
<td>0.23 (0.14 to 0.32)</td>
<td>-0.85 to 1.31</td>
</tr>
<tr>
<td>Point 2 (Peak pressure)</td>
<td>0.15 (0.03 to 0.26)</td>
<td>-1.23 to 1.53</td>
</tr>
<tr>
<td>Point 3 (0.25 s past peak)</td>
<td>-0.63 (-0.75 to -0.49)</td>
<td>-2.19 to 0.93</td>
</tr>
<tr>
<td>Point 4 (0.5 s past peak)</td>
<td>-0.07 (-0.15 to 0.01)</td>
<td>-1.01 to 0.87</td>
</tr>
<tr>
<td>Point 5 (1 s past peak)</td>
<td>0.21 (0.12 to 0.29)</td>
<td>-0.83 to 1.25</td>
</tr>
<tr>
<td>Point 6 (last end-expiratory point)</td>
<td>0.50 (0.39 to 0.61)</td>
<td>-0.82 to 1.82</td>
</tr>
</tbody>
</table>

### Table 3 Bias and limits of agreement according to Bland and Altman for different pressure points (calculated $\Delta P_{tr}$ measured tracheal pressure, $P_{Rohrer}$ calculated according to 4) during VC with 12 cm H$_2$O PEEP

As our concern was not the reliability of direct $P_{tr}$ measurement, we did not systematically screen all the measured $P_{tr}$ data for malposition and potential artefacts to determine the absolute number of erroneous measurements to which our current equipment is obviously prone. Despite the basic methodological concerns with direct $P_{tr}$ measurement discussed below, we know, that a dedicated tool for measuring $P_{tr}$ would probably have given better-measured $P_{tr}$ data.

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Cardiogenic oscillations were another disturbing factor. Measurement of $P_{tr}$ was performed at the carina close to the beating heart. Calculation of $P_{tr}$, in contrast, uses pressure and flow data sampled at the proximal tracheal tube outlet, distant from the beating heart with the dampening effect of airways and tracheal tube interposed. Cardiogenic oscillation affects the measured $P_{tr}$ more than the calculated $P_{Rohrer}$, and an association between measured and calculated $P_{tr}$ data may be considered at the very moment when a heartbeat distorts the measured $P_{tr}$. At low lung volumes the heartbeats influence the measured $P_{tr}$ even more strongly, which can explain the weak association of some of the pressure points at end-expiration.

The combination of two studies, one with VC and an additional one with assisted spontaneous breathing, PS and BiPAP, comprising different numbers of animals is certainly open to statistical criticism. We justify this with our intention to test the agreement between calculated and measured $P_{tr}$ under an extended spectrum of flow patterns.

Finally, this is a short-term study only. A clinical validation would have to take long-term effects like progressive tracheal tube narrowing by secretions into account. An estimation of the potential error caused by tracheal tube narrowing is therefore added (see below).

### Table 4 Bias and limits of agreement according to Bland and Altman for different pressure points (calculated $\Delta P_{tr}$ measured tracheal pressure, $P_{Rohrer}$ calculated according to 4) during BiPAP ventilation

<table>
<thead>
<tr>
<th>Point (definition)</th>
<th>Bias (cm H$_2$O)</th>
<th>Limits of agreement (cm H$_2$O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Point 1 (0.20 s past start of inspiration)</td>
<td>1.22 (1.15 to 1.28)</td>
<td>0.84 to 1.60</td>
</tr>
<tr>
<td>Point 2 (Peak pressure)</td>
<td>-1.27 (-1.35 to -1.20)</td>
<td>-1.71 to -0.83</td>
</tr>
<tr>
<td>Point 3 (0.20 s past peak)</td>
<td>-0.20 (-0.33 to -0.08)</td>
<td>-0.92 to 0.52</td>
</tr>
<tr>
<td>Point 4 (0.15 s past start of expiration)</td>
<td>0.51 (0.37 to 0.65)</td>
<td>-0.29 to 1.31</td>
</tr>
<tr>
<td>Point 5 (0.25 s past start of expiration)</td>
<td>-0.1 (-0.20 to -0.01)</td>
<td>-0.64 to 0.44</td>
</tr>
<tr>
<td>Point 6 (last end-expiratory point)</td>
<td>0.13 (0.05 to 0.20)</td>
<td>-0.29 to 0.55</td>
</tr>
</tbody>
</table>

### Table 5 Bias and limits of agreement according to Bland and Altman for different pressure points (calculated $\Delta P_{tr}$ measured tracheal pressure, $P_{Rohrer}$ calculated according to 4) during PS ventilation

<table>
<thead>
<tr>
<th>Point (definition)</th>
<th>Bias (cm H$_2$O)</th>
<th>Limits of agreement (cm H$_2$O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Point 1 (0.10 s past start of inspiration)</td>
<td>1.31 (1.27 to 1.34)</td>
<td>1.01 to 1.61</td>
</tr>
<tr>
<td>Point 2 (Peak pressure)</td>
<td>-0.13 (-0.17 to -0.10)</td>
<td>-0.43 to 0.17</td>
</tr>
<tr>
<td>Point 3 (0.10 s before end of inspiration)</td>
<td>-0.16 (-0.19 to -0.14)</td>
<td>-0.40 to 0.08</td>
</tr>
<tr>
<td>Point 4 (0.15 s past start of expiration)</td>
<td>0.42 (0.40 to 0.44)</td>
<td>0.24 to 0.60</td>
</tr>
<tr>
<td>Point 5 (0.25 s past start of expiration)</td>
<td>-0.08 (-0.10 to -0.06)</td>
<td>-0.24 to 0.08</td>
</tr>
<tr>
<td>Point 6 (last end-expiratory point)</td>
<td>-0.02 (-0.04 to 0.00)</td>
<td>-0.22 to 0.18</td>
</tr>
</tbody>
</table>
Can direct measurement of tracheal pressure be regarded as a gold standard at present?

Apart from the above, direct measurement of $P_{tr}$ has drawbacks that make it difficult to regard it as a true gold standard. Thin measuring catheters are easily obstructed, and thick catheters affect flow pattern and increase the tracheal tube resistance. (To avoid the latter, the catheter in this study was placed outside the tracheal tube, whereas in all previous studies that we know of the $P_{tr}$ catheter was placed inside the ETT.) Catheters that measure $P_{tr}$ at the tip of the catheter in an axial direction (like the one used in the present study) overestimate the expiratory pressure because of the Bernoulli effect. With expiratory gas flowing towards the measuring site of the catheter, there is an additional dynamic pressure component that depends on the square of flow velocity. Moreover, the cross-sectional area changes abruptly at the transition of the tracheal tube to the trachea. Here kinetic energy dissipates by flow separation, leading to additional pressure loss. As a consequence, $P_{tr}$ should be measured where the gas flow is fully developed. This requires the catheter to be advanced at least 3 cm beyond the tip of the tracheal tube. Positioning the measuring site radial to the flow direction (using catheters with a lateral opening) also has drawbacks because the velocity profile is inhomogeneous—it is parabolic when flow is laminar, and it is flat when transitional flow is present. Apart from those basic methodological concerns it is not easy to position the catheter correctly, and one might easily find the catheter stuck against the tracheal wall or obstructed by secretions. We also observed that the position of the catheter could change with lung volume (that is, with tidal volume and/or with PEEP).

Different methods to calculate tracheal pressure

Different methods have been proposed for calculating $P_{tr}$. From a theoretical standpoint it is certainly more satisfying to take all relevant variables (gas physical properties, the inner geometry of the ETT and its curvature) into account rather than limiting the analysis to the mathematical description of empirical pressure–flow curves using the Rohrer coefficients (depending on length and inner diameter (ID) of the native tracheal tube measured ex-vivo). When it comes to the bedside, however, it is cumbersome, if not completely impossible, to determine the curvature of the tracheal tube or the changes in gas physical properties oscillating within the respiratory cycle. Jarreau and colleagues have suggested dimensional forms of the more complex equations given in their paper for practical estimation of the pressure decrease over the tracheal tube. In the dimensional form of the equations only variables available at the bedside (flow, length, and ID of the native tracheal tube) are used and whether flow is turbulent or not is estimated from a rough approximation. Using this approach we found an excellent agreement between $P_{tr}$ calculated with the method suggested by Guttmann and colleagues and the method suggested by Jarreau and colleagues (see Figs 1–3 and Appendix). Changing $F_{O2}$ and, hence, the density of the gas, affects resistance, which is particularly important in paediatric tubes, but for clinical purposes in adults the effect is negligible. While the gas density of room air is about 89% of the density of pure oxygen, density of helium is 12% of pure oxygen. Thus, the use of gases with major difference in density would have to be taken into account when calculating $P_{tr}$.

How large are potential errors when tracheal pressure is calculated?

All the methods for calculating $P_{tr}$ depend on knowledge of the true actual ID of the tracheal tube. If secretions progressively narrow the tracheal tube while the algorithm continues to assume a native, unobstructed tracheal tube an error will result. The narrowing increases the flow-dependent resistive pressure decrease over the tracheal tube ($\Delta P_{tr}$), and the calculated $P_{tr}$ now overestimates the true $P_{tr}$. The potential error is estimated in Figure 5. The $\Delta P_{ETT}$ of the native tracheal tube with ID 8 is plotted for increasing flow rates. An assumed narrowing from ID 8 to 7.5 mm increases the tracheal tube-related resistance by about 25%, shifting the pressure–flow curve upwards. The difference between the $\Delta P_{ETT}$ curves with 7.5 and 8.0 mm ID, respectively, yields the potential error (thick bottom line). With a constant inspiratory flow of 18 litre min$^{-1}$ (used in
the present study) and a narrowing from 8 to 7.5 mm ID, the $P_{\text{Rohrer}}$ overestimates the true $P_{\text{tr}}$ by 0.11 cm H$_2$O; with 48 litre min$^{-1}$ the overestimation is 1.06 cm H$_2$O, and with 90 litre min$^{-1}$ the overestimation is 3.98 cm H$_2$O. If this is added to the 0.6–1 cm H$_2$O difference between $P_{\text{Rohrer}}$ and measured $P_{\text{tr}}$, it appears that up to an inspiratory flow rate of 48 litre min$^{-1}$ and with a minor narrowing (8–7.5 mm ID) the error is still acceptable for clinical purposes. With more complex flow patterns and/or more pronounced narrowing, estimation of the potential error is less straightforward, however. A comparison of the actual $\Delta P_{\text{ETT}}$ over time plot with the same plot of the initial native tracheal tube is of help. A comparison of the flow-dependent upward shift of the $\Delta P_{\text{ETT}}$ (see Fig. 5) with the native condition is more sensitive than monitoring tracheal tube obstruction with peak airway pressure ($P_{\text{peak}}$). As discussed above, the effects of tracheal tube obstruction are flow-dependent. Not until the narrowing is severe enough to accelerate the flow rate up to a threshold above which flow changes from laminar to turbulent, will the increase in $P_{\text{peak}}$ become clinically obvious. We acknowledge, however, that progressive narrowing by secretions, and, hence, knowledge of the true actual ID, pose a problem for calculation of $P_{\text{tr}}$.

Calculation or measurements of tracheal pressure?

Different methods have been suggested to determine the true actual ID of a tracheal tube. This information must then be used in the calculation of $P_{\text{tr}}$ to obtain reliable results when the tracheal tube is narrowed.$^1$ Acoustic reflection has been proposed$^{16-18}$ to determine the actual ID of the tracheal tube. We have suggested detecting tube obstruction from the expiratory flow signal.$^{19}$ If such methods are applied, we do not see a fundamental objection to calculating $P_{\text{tr}}$ rather than directly measuring it.$^{20}$ Clinically, this is even more relevant as the direct $P_{\text{tr}}$ measurement has the methodological difficulties mentioned above. Even if future long-term studies show that direct measurement is accurate, inserting another catheter into the trachea (or flushing an airway catheter on a regular basis to avoid obstruction) has to be balanced against the added risk of infection. The development of miniaturized pressure transducers based on fibre-optic technology is a potential step forwards,$^7$ but they would have to be stable and comparable with calculation methods that are cheap and require no more than the pressure–flow data available at the ventilator.

Breath-by-breath information on $P_{\text{tr}}$—whether measured or calculated—helps in setting assisted modes of spontaneous breathing,$^{21-25}$ and other modes might also benefit from information on $P_{\text{tr}}$.

We conclude that calculating tracheal pressure is reliable and the results agree well with those determined by direct measurement. Calculation of $P_{\text{tr}}$ uses pressure–flow signals that are already available, is non-invasive, and can be done breath-by-breath. The resistive pressure decreases over the tracheal tube can be determined and the ventilator can be set with respect to tracheal pressure. If common clinical problems like progressive narrowing of the tracheal tube by secretions can be taken into account appropriately, this method can aid respiratory monitoring without the possible errors of direct tracheal pressure measurement.

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Appendix

For gas to flow through a tracheal tube a difference must exist between the airway pressure $P_{\text{prox}}$ at the proximal end of the tracheal tube and the tracheal pressure $P_{\text{tr}}$ at the distal outlet of the tracheal tube. This pressure difference is the pressure decrease across the tracheal tube: $\Delta P_{\text{ETT}}$.

For the inspiratory and expiratory $\Delta P_{\text{ETT}}$ it follows:

$$\Delta P_{\text{ETT-in}} = P_{\text{aw-in}} - P_{\text{tr-in}}$$

$$\Delta P_{\text{ETT-ex}} = P_{\text{aw-ex}} - P_{\text{tr-ex}}$$

with ‘in’ and ‘ex’ denoting inspiration and expiration, respectively. The inspiratory $\Delta P_{\text{ETT}}$ is positive, the expiratory $\Delta P_{\text{ETT}}$ is negative.

From equations (1) and (2) the inspiratory and expiratory $P_{\text{tr}}$ can be derived:

$$P_{\text{tr-in}} = P_{\text{aw-in}} - \Delta P_{\text{ETT-in}}$$

$$P_{\text{tr-ex}} = P_{\text{aw-ex}} + \Delta P_{\text{ETT-ex}}$$

Two values are, hence, necessary for calculating $P_{\text{tr}}$, airway pressure ($P_{\text{prox}}$) and $\Delta P_{\text{ETT}}$. Calculation of $\Delta P_{\text{ETT}}$ requires a mathematical description of the non-linear pressure–flow curve of the tracheal tube. This has been done either by using empirical equations such as the Rohrer equation$^{14}$ or by using equations based on general physical laws such as the Ito and Blasius formula.$^2,26-28$ We investigated the approach using the Rohrer coefficients.$^5$ For comparison, the approach based on the Blasius formula$^2$ was also checked.

Rohrer equation

The Rohrer equation was first used to describe the airflow resistance of the lung$^{14}$ and can describe the pressure–flow curve of a single tube or a tubing system. It combines a linear with a non-linear pressure–flow dependence. According to the Rohrer equation the inspiratory and expiratory pressure decrease across the tracheal tube $\Delta P_{\text{ETT}}$ is described as:
Tracheal pressure can be calculated reliably using the Blasius–Ito approach in its simplified and dimensional form as proposed by Jarreau and colleagues. According to fluid dynamic theory the total inspiratory pressure decrease across the tracheal tube is divided into two components, the first component $\Delta P_{ke}$ being caused by the change in kinetic energy between two distinct cross sections (here tube connector and tracheal tube), and the second component $\Delta P_{vd}$ being the friction pressure decrease:

$$\Delta P_{ETT-in} = \Delta P_{ke} + \Delta P_{vd}$$

(7)

The first component $\Delta P_{ke}$ assuming a change in kinetic energy between a standard tracheal tube connector and a tracheal tube of ID $\geq 3$ mm is given by:

$$\Delta P_{ke} = -0.98 \cdot V^2 \cdot (D^{-4} - 5.72)$$

(8)

where $V$ is the flow rate (litre s$^{-1}$), and $D$ is the ID of the tracheal tube (cm).

The second component $\Delta P_{vd}$ is calculated using the Blasius formula in its simplified and dimensional form:

$$\Delta P_{vd} = 0.0104 \cdot L \cdot D^{-4.75} \cdot V^{1.75}$$

(9)

where $L$ is the length of the tracheal tube (cm), and $V$ is the flow rate (litre s$^{-1}$).

This gives the following simplified formula:

$$\Delta P_{ke} = -0.98 \cdot V^2 \cdot [D_{ETT}^{-4} - D_{CONN}^{-4}]$$

when $D \geq 3$ mm

$$\Delta P_{ke} = -0.98 \cdot V^2 \cdot [1.5 \cdot D_{ETT}^{-4} - D_{CONN}^{-4}]$$

when $D = 2.5$ mm

where $D_{ETT}$ is the diameter of the tracheal tube and $D_{CONN}$ is the diameter of the tracheal tube connector (usually 1.15 cm).

All $P_u$ calculations were performed with a tracheal tube of 32 cm length and of 7 mm ID.

References

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