Pulmonary Mechanics in Normal Subjects Following Endotracheal Intubation

Thomas J. Gal, M.D.*

To determine the effects of endotracheal intubation on airway mechanics in healthy individuals, measurements of pulmonary function were made while semirecumbent subjects performed dynamic respiratory maneuvers. Eight healthy male volunteers were studied under three test conditions: 1) breathing through a 25-mm ID mouthpiece (control); 2) with an 8.0-mm ID endotracheal tube adaptor in the mouthpiece (external resistance); 3) with an 8.0-mm ID endotracheal tube 25 cm long in place. Decreases in peak inspiratory flow and peak expiratory flow during flow-volume loops were the only significant changes seen with the external resistance. Peak flows were also decreased by intubation, but to a significantly greater extent (P < 0.01). Forced vital capacity (FVC) decreased significantly with tracheal intubation to 89 ± SEM 2% per cent of control. Forced expiratory volume in 1 sec (FEV₁) was also decreased significantly, whether expressed as actual volume or as FEV₁/FVC. Compliance of the lung was unchanged after intubation, but flows below mid-vital capacity during maximal expiratory flow-volume (MEFV) curves were decreased significantly from control. Responses to breathing helium were abnormal with intubation in many subjects, although mean responses did not differ significantly from control. Mean values for closing volume and slope of Phase III (ΔN₂ per cent/l) were likewise not significantly different from control, but in at least three subjects were indicative of peripheral airway obstruction. The decreased peak flows and FEV₁ with intubation reflect the tube's behavior as a significant fixed upper-airway resistance. The decreased FVC and expiratory flows below mid-vital capacity indicate that with intubation many subjects showed diffuse airway constriction superimposed on the fixed resistance of the tube. (Key words: Airway; Intubation, endotracheal; Lung; closing capacity; compliance; function; volume closing.)

Endotracheal intubation may influence airway function in several ways. First, the tube represents a mechanical burden to a spontaneously breathing patient, since it decreases airway caliber and increases resistance to breathing. Recent work suggests that this increased resistance is not significant during quiet breathing but is likely to exert a marked effect during deep breathing and maximal effort. On the other hand, the endotracheal tube may paradoxically increase peak flow rates during forced expiration by preventing dynamic compression of the trachea. Finally, mechanical irritation of the larynx and trachea by the tube may alter the function of the lower airways by inducing reflex constriction distal to the tube. This effect may extend to the smaller peripheral airways (<2 mm diameter) and affect intrapulmonary gas distribution.

The endotracheal tube has been assumed to behave solely as a fixed upper-airway resistance. Despite the widespread use of endotracheal intubation as a therapeutic modality, there has been no study that evaluated its possible effects on the function of the intrapulmonary airways distal to the tube. The present study was performed to identify alterations in normal airway function produced by endotracheal intubation and compare them with changes produced by a fixed external airway obstruction of the same diameter as the endotracheal tube. The aim was to delineate whether changes in pulmonary mechanics with intubation were merely similar to those of a simple fixed resistance or were influenced by further effects on airways distal to the tube. The mechanical properties of these airways were examined by measurements such as lung elastic recoil, maximal flow during forced vital capacity maneuvers, and closing volume.

Methods

Eight healthy nonsmoking volunteers between the ages of 22 and 29 years were studied in the fasting state after informed consent had been obtained. Approval was granted by the Human Studies Committee of the University of Virginia. All measurements were made with the subjects in the semirecumbent position, since it represents the posture in which most patients whose tracheas are intubated must breathe. Subjects were tested under three conditions of breathing: 1) through a 25-mm ID mouthpiece (control); 2) through an 8.0-mm ID endotracheal tube adaptor inserted into the same mouthpiece (external resistance); 3) through an 8.0-mm ID endotracheal tube 25 cm long (Shiley Laboratories). Topical anesthesia of the airway was produced by lidocaine, 4 per cent, 8–10 ml, inhaled from an ultrasonic nebulizer (Mistogen®). The tube was inserted to its full depth with assurance against endobronchial intubation. The cuff was inflated to produce a leak-free condition with at least 20 cm H₂O applied pressure. Subjects inspired to near total lung capacity and then relaxed against an occluded mouthpiece. The absence of a leak was verified.

* Assistant Professor.

Received from the Department of Anesthesiology, University of Virginia, Charlottesville, Virginia 22908. Accepted for publication May 22, 1979. Presented in part at the Annual Meeting of the American Society of Anesthesiologists, New Orleans, October 1977. Supported in part by a grant from American Society of Anesthesiologists (Parker B. Francis Foundation), and by funds from the American Lung Association.

Address reprint requests to Dr. Gal.
by maintenance of a constant airway pressure (sensed by an aneroid manometer) for at least 5 sec.

Upper airway function was assessed with standard spirometry using a waterless rolling-seal spirometer (model 840, Ohio Medical Products). The spirometer was calibrated with a 3-l super-syringe and had a dynamic resistance less than 0.2 cm H₂O at 10 l/sec and a frequency response flat within 10 per cent to 15 Hz. Vital capacity (VC), forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV₁) were measured. Subjects performed at least three FVC maneuvers during each testing condition. The largest values for FEV₁ and FVC were used in the results provided the FVC from which FEV₁ was obtained was within 200 ml of the largest FVC. All volumes were corrected to body temperature and pressure saturated with water vapor (BTPS). Maximum-effort flow–volume loops were obtained with the spirometer and a fast-response X-Y recorder (Hewlett-Packard 7041A). Flow at the mouth derived by electronic differentiation of the spirometer's volume signal was plotted on the Y-axis and volume on the X-axis. The largest values for peak inspiratory flow (V₁max) and peak expiratory flow (Vₑₐₓₐₑₕ) were used in the results.

The lower (intrathoracic) airways were evaluated by maximum expiratory flow–volume (MEVF) curves in which flows and volumes were simultaneously plotted during a maximal FVC maneuver. Flow was measured at 10 per cent intervals of vital capacity. At least three efforts whose values for FVC varied by less than 5 per cent were aligned at total lung capacity, and maximal values for expiratory flow at 10 per cent intervals of FVC were used to construct a composite MEVF curve for each subject. These composite curves were constructed for each of the three experimental conditions. The largest values at each interval on the MEVF curve were used to minimize gas compression artifacts from excessive effort. To identify possible constriction in the smaller peripheral airways, MEVF curves were obtained after subjects performed three full inspiratory vital capacity maneuvers with a low-density gas mixture (80 per cent helium, 20 per cent oxygen). The response to helium was determined by measuring flow rates at 50 per cent of vital capacity after helium breathing (Vₑₓₓₐₑₓₐₑₕ Hₑ). These were compared with Vₑₓₓₐₑₓₐₑₕ obtained under the same test conditions when subjects breathed air (Vₑₓₓₐₑₓₐₑₕ air). The increase in flow with helium was termed ΔVₑₓₓₐₑₓₐₑₕ and expressed as a percentage of the flows with air: ΔVₑₓₓₐₑₓₐₑₕ per cent = (Vₑₓₓₐₑₓₐₑₕ Hₑ – Vₑₓₓₐₑₓₐₑₕ air) ÷ Vₑₓₓₐₑₓₐₑₕ air × 100. The largest value for Vₑₓₓₐₑₓₐₑₕ Hₑ was used to calculate ΔVₑₓₓₐₑₓₐₑₕ provided the FVC from which it was obtained was within 5 per cent of that with air. The MEVF curves after breathing helium were superimposed on the air curves obtained under the same test conditions. Curves whose FVC values were unequal were matched at residual volume. The point on the two curves where flows became identical and continued to residual volume was identified as the volume of isoflow (Vₑₓₓₐₑₓₐₑₕ), which was expressed as percentage of FVC above residual volume. Vₑₓₓₐₑₓₐₑₕ for each testing condition was recorded as the mean of three such comparisons.

The closing volume test was performed using Anthonisen’s modification of Fowler’s single-breath nitrogen test. Subjects exhaled to near residual volume (RV), then inhaled 100 per cent oxygen to total lung capacity and slowly exhaled to residual volume. Flow monitored by visual display was kept below 0.5 l/sec. Expired N₂ sampled at the mouthpiece by a rapid-response N₂ analyzer (Hewlett Packard Model 57302A) was displayed on the Y-axis of the X-Y recorder and volume on the X-axis. Closing volume (CV) was defined as the volume between the volume at which an abrupt increase in expired N₂ occurred and RV. This was expressed also as percentage of vital capacity, CV/VC per cent. The slope of the Phase III alveolar plateau was also calculated from a line of best fit from 30 per cent of expired vital capacity to onset of CV and expressed as percentage change in N₂ concentration per liter (ΔN₂ per cent/l). Each subject repeated the test at least three times. Values for CV and slope of Phase III were obtained as the means of two or three satisfactory tracings that agreed closely, i.e., with the highest VC, slow expiratory flow, and positive identification of the abrupt change in slope at CV.

Transpulmonary pressure (Pₑ) was estimated as the difference between the pressure at the airway opening and esophageal pressure. A balloon 10 cm long, containing 0.5 ml air, was passed into the mid-esophagus and connected to a differential pressure transducer (Validyne MP45, range ± 50 cm H₂O). The opposite side of the transducer was connected to a pressure tap in the mouthpiece. Lung elastic recoil was estimated by quasi-static pressure–volume curves. Pₑ was simultaneously plotted against lung volume on an X-Y recorder while subjects inspired to total lung capacity and then slowly exhaled (about 0.2 l/sec) to residual volume. Individual pressure–volume curves were constructed as the means of two to four correctly recorded curves, i.e., with the greatest inspiratory capacity and without sudden shifts of measured Pₑ due to esophageal muscle contraction. To characterize the effect of lung recoil on the ability to generate airflow, Pₑ was plotted against maximum expiratory flow at the same lung volume using MEVF and pressure–volume curves to construct maximum-flow static recoil.
(MFSR) curves. From the MFSR curves the slope of the curve between 50 and 30 per cent of vital capacity was calculated. This represents the conductance of the upstream airway (G_w) which extends from the alveoli to the equal pressure point, i.e., where transmural pressure is zero. According to the model of Mead and associates, expiratory flow (V_{E,max}) = \( P_0 \times G_w \).

To estimate the role of expiratory effort in generating maximal flow, dynamic transpulmonary pressure was measured during performance of all FVC maneuvers. Here the difference between esophageal and airway opening pressures was sensed by a differential pressure transducer (Validyne MP-45, range 0 ± 250 cm H_2O), and the esophageal balloon volume was 1.0 ml. The mean of at least three determinations of maximum pressure was recorded for each experimental condition.

Resistances for the endotracheal tube and the adapter used as the external resistance in the study were estimated during constant flows of 0.5, 1.0, 1.5, and 2.0 l/sec. The difference between upstream pressure (proximal to the entrance of the resistive element) and downstream pressure (outside the distal end) was sensed in the appropriate range by one of two MP-45 transducers (ranges 0 ± 5 and 0 ± 50 cm H_2O).

Data were analyzed by a paired two-tailed t test. The significance of differences between the control state and the other two test conditions was assessed at the 95 per cent confidence level.

### Results

Control measurements with conventional spirometry (table 1) were unaffected by the external resistance. With tracheal intubation slow vital capacity was unchanged. Forced vital capacity decreased significantly to 89 ± SEM 2 per cent of control. FEV_1 was decreased to 79 per cent of control when expressed as actual volume. The decrease in the ratio FEV/FVC per cent was likewise significant despite a decreased FVC. During flow-volume loops peak flows were decreased significantly by both external resistance and intubation. Both peak inspiratory flow (V_{I,max}) and peak expiratory flow (V_{E,max}) were decreased to about half their control values with intubation, significantly more so than with the external resistance.

Table 1. Results of Spirometry and Flow–Volume Loops during Three Test Conditions (Means ± SEM for Eight Subjects)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Control</th>
<th>External Resistance</th>
<th>Tracheal Intubation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slow vital capacity (VC) (l)</td>
<td>5.8 ± 0.3</td>
<td>5.8 ± 0.4</td>
<td>5.6 ± 0.3</td>
</tr>
<tr>
<td>Forced vital capacity (FVC) (l)</td>
<td>5.8 ± 0.3</td>
<td>5.7 ± 0.3</td>
<td>5.1 ± 0.3*</td>
</tr>
<tr>
<td>Forced expiratory volume in 1 sec (FEV_1) (l)</td>
<td>4.4 ± 0.2</td>
<td>4.2 ± 0.3</td>
<td>3.5 ± 0.2*</td>
</tr>
<tr>
<td>FEV_1/FVC (per cent)</td>
<td>77 ± 2</td>
<td>76 ± 2</td>
<td>68 ± 1*</td>
</tr>
<tr>
<td>Peak inspiratory flow (V_{I,max}) (l/sec)</td>
<td>7.4 ± 0.6</td>
<td>4.6 ± 0.4*</td>
<td>3.6 ± 0.2*</td>
</tr>
<tr>
<td>Peak expiratory flow (V_{E,max}) (l/sec)</td>
<td>8.7 ± 0.3</td>
<td>5.6 ± 0.4*</td>
<td>4.4 ± 0.4*</td>
</tr>
</tbody>
</table>

* Significant difference from control by t test for paired data, \( P < 0.01 \).

The mean control value for closing volume was unchanged by intubation, whether expressed as absolute volume (520 ± 30 ml) or as percentage of vital capacity (11 ± 1 per cent). Although the mean slope of Phase III was unchanged from the control value of 1.5 ± 0.1 per cent N_2/1 with intubation, three subjects (1, 2 and 5) showed distinct positive increases in slope.

Lung elastic recoil did not change significantly with intubation (fig. 3). Neither recoil pressure at total lung capacity nor the slope of the pressure–volume curve in the range of tidal volume (30–40 per cent VC) was altered. V_{E,max} at corresponding levels of P_0 below 70 per cent VC was decreased with intubation compared with control and external resistance (fig. 4). Upstream half of vital capacity, flows with the external resistance did not differ from control but appeared to be decreased with intubation. These decreased flows are also evident when values are plotted at 10 per cent intervals of vital capacity oriented above residual volume (fig. 2). With intubation flows between 50 per cent of vital capacity, residual volume was significantly lower than with the other conditions. The difference was also demonstrable with flows expressed in units of FVC/sec to normalize for differences in forced vital capacity.

When helium was breathed, the normal \( \Delta V_{max} \) to 50 per cent in the control state was decreased in five subjects with intubation and increased in three, but the mean value (36 ± 4 per cent) was not significantly different from control (43 ± 2 per cent). Volume of isoflow (V_{isoV}) increased in five subjects with intubation and decreased in three. In two of the subjects with decreased V_{isoV}, flow on the helium curve decreased below that on the air curve, before becoming equal at V_{isoV}. The mean value with intubation (20 ± 2 per cent) did not significantly differ from control. In contrast, V_{isoV} decreased significantly with the external resistance.

The mean control value for closing volume was unchanged by intubation, whether expressed as absolute volume (520 ± 30 ml) or as percentage of vital capacity (11 ± 1 per cent). Although the mean slope of Phase III was unchanged from the control value of 1.5 ± 0.1 per cent N_2/1 with intubation, three subjects (1, 2 and 5) showed distinct positive increases in slope.
Fig. 1. Composite maximum expiratory flow-volume curves for eight normal subjects during the three test conditions. Curves are aligned at total lung capacity (TLC) and maximum expiratory flow is plotted at 10 per cent intervals of measured forced vital capacity from TLC to residual volume (RV).
Fig. 2. Mean values for maximum expiratory flow are plotted at 10 per cent intervals of vital capacity above residual volume. Data are from eight normal subjects. Bars indicate ± 1 SEM. In the left panel, flow is plotted in l/sec, whereas in the right panel it is expressed as fractions of forced vital capacity per sec (FVC/sec).

Conductance ($G_{\text{aw}}$) calculated from the slope of the MFSR curves between 50 per cent and 30 per cent of VC was $0.6 \pm 0.2$ l/sec/cm H$_2$O for control, compared with $0.7 \pm 0.1$ l/sec/cm H$_2$O with the external resistance. The mean $G_{\text{aw}}$ with intubation ($0.45 \pm 0.11$ l/sec/cm H$_2$O) was not decreased significantly from control.

Maximum transpulmonary pressure during FVC maneuvers was $69 \pm 5$ cm H$_2$O in the control state, $72 \pm 5$ cm H$_2$O with the external resistance, and $74 \pm 10$ cm H$_2$O with intubation. No significant difference in expiratory effort could be demonstrated with intubation.

At a flow of 0.5 l/sec the resistance of the tube was 2.6 cm H$_2$O/l/sec, while that of the adaptor (external resistance) was 1.8 cm H$_2$O/l/sec (fig. 5). At a flow of 1.0 l/sec the resistances were 5.8 and 3.2 cm H$_2$O/l/sec, respectively.

**Discussion**

The results from this study indicate that tracheal intubation alters forced expiratory flows to a greater extent than and in a different fashion from an external

![Fig. 3. Mean deflation quasi-static pressure-volume curves for eight subjects before and after intubation. Data points for transpulmonary pressure are plotted at intervals of 10 per cent of control vital capacity. Bars represent 1 SEM.](image)

![Fig. 4. Maximum-flow static recoil (MFSR) curves during the three test conditions. Simultaneous determinations of expiratory flow and transpulmonary pressure are plotted at intervals of 10 per cent of vital capacity (VC) between 70 and 30 per cent VC. Values are plotted as mean ± SEM for both flow and pressure. Shaded area indicates range of normal values taken from data of Cherniack (Pulmonary Function Testing, Philadelphia, W. B. Saunders, 1977, p 207).](image)
Further explanations for a decreased FVC include decreased compliance of the lung and decreased inspiratory effort resulting in a decreased total lung capacity. The latter would appear to account for the decreases in FVC found in Subjects 5 and 6, but slow vital capacity values for both subjects during intubation were within 200 ml of control values for FVC. These relatively small changes in slow vital capacity and the lack of alterations in the normal pressure-volume behavior of the lung do not support the likelihood of a decreased total lung capacity. Furthermore, measurements of thoracic gas volume in seated subjects whose tracheas are intubated confirm that total lung capacity is not significantly decreased.† Thus, the decreased FVC with intubation appears to be best explained by an associated increase in residual volume, most likely as a result of airway constriction.

In contrast to effects of FVC, the increased resistance of the tube may have been sufficient to account for decreases in FEV₁ observed during intubation. If maximal flows were decreased over a greater portion of vital capacity, FEV₁, which is a time integration of flows, would be decreased. Over the initial portion of vital capacity, flows with an endotracheal tube held externally are decreased more than with the external resistance. One might rightfully argue that the resistive pathway provided by the endotracheal tube exceeded that of the external resistance (fig. 5). Thus, a strict quantitative basis for comparing flows with intubation did not exist, particularly those highly dependent on effort. Nevertheless, even measurements with the endotracheal tube held externally do not provide a truly valid quantitative comparison with the tube in situ. This is because the tube does not add external resistance to normal airway resistance while in the trachea, but rather substitutes for the normal resistance of the segment from mouth to trachea, which accounts for 30–40 per cent of normal airway resistance.¹³

The decreased FVC with intubation cannot be accounted for solely by the imposed resistance of the tube unless expiratory efforts were submaximal. In five subjects this was clearly not the case, since maximum transpulmonary pressures were increased in the intubated state. One subject (3) showed a small decrease, while in the remaining two (5, 6) demonstrated larger decreases. In the latter two subjects this decreased expiratory effort can adequately account for the decrease in peak flow, but cannot fully explain the decreased flows over the less effort-dependent portions of the FVC. The MEFV curves (fig. 2) for these two subjects are somewhat convex to volume axis in the final third of vital capacity, and suggest that subjects reached flow limitation as residual volume was approached rather than "dropping off" their curves at high residual volume, as is observed with weakness or decreased effort.¹⁴

resistance, but from about the mid-point of vital capacity to residual volume neither alters flow notably from control (fig. 6).

With intubation, expiratory flows were decreased to relatively constant values over the initial portion of vital capacity in a fashion described by Miller and Hyatt for a fixed obstruction of the upper airway.\(^\text{16}\) The decreased flows below the mid-point of vital capacity suggest an additional factor superimposed on this fixed upper-airway obstruction. The most likely possibility is diffuse airway constriction, since lung elastic recoil, the other determinant of expiratory flow over this range of lung volume, was unchanged. One of the possible sites for this constriction was in the small peripheral airways. The response to breathing helium provides a physiologic framework for demonstrating obstruction in these airways.\(^\text{16}\) The effects of helium inhalation can be explained by the concept of equal pressure points (EPP) proposed by Mead.\(^\text{11}\) A normal \(\Delta V_{\text{max}}\) with helium and a normal or decreased \(V_{\text{Iov}}\) imply that the site of increased resistance lies in the upper airway or large central airways, where flow is turbulent and highly dependent on density. A less than normal \(\Delta V_{\text{max}}\) and increased \(V_{\text{Iov}}\) suggest that the major site of flow resistance is in the small peripheral airways, where flow is laminar and not affected by the low density of helium. Although it is not possible to demonstrate from mean changes that peripheral airway obstruction occurred with intubation, the individual responses (table 2) showed different patterns. Many were compatible with small-airway constriction. On the other hand, a normal \(\Delta V_{\text{max}}\) and \(V_{\text{Iov}}\) may not entirely exclude the possibility of peripheral airway obstruction if resistance of the tube is severe enough to shift the equal pressure points mouthward, thereby increasing the range of lung volumes over which flow is density-dependent. This possibility is supported by recent data from patients with diffuse obstructive lung disease who demonstrated large helium-induced flow increases at mid-vital capacity when severe upper airway obstruction was simulated.\(^\text{17}\)

Although not specific, the closing volume test has been described as a sensitive indicator of constriction in small airways.\(^\text{18}\) Similarly, the slope of Phase III is a sensitive measure of non-uniform ventilation even in mild peripheral airway disease,\(^\text{19}\) although, like closing volume, it also reflects abnormalities in the elastic properties of airways. When taken collectively, these tests do not appear to indicate peripheral airway constriction with intubation. Nevertheless, three subjects (1, 2 and 5) demonstrated distinct increases in the slope of Phase III ("alveolar plateau"), indicating altered intrapulmonary gas distribution and probable small-airway constriction. In all three, closing volume decreased and onset of Phase IV was less distinct. These changes are still compatible with bronchoconstriction and "air trapping," which result in a failure to establish the normal nitrogen concentration gradient.\(^\text{20}\) It is also possible that residual volume increased and produced an increased closing capacity in other subjects with no change in CV/VC per cent. Since measurements of absolute lung volumes were not performed in this study, one can only speculate about this possibility.

**Table 2. Responses to Breathing Helium during the Three Test Conditions**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>External Resistance</th>
<th>Intubation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(\Delta V_{\text{max}}) (Per Cent)</td>
<td>(V_{\text{Iov}}) (Per Cent)</td>
<td>(\Delta V_{\text{max}}) (Per Cent)</td>
</tr>
<tr>
<td>Subject 1</td>
<td>50</td>
<td>20</td>
<td>45</td>
</tr>
<tr>
<td>Subject 2</td>
<td>43</td>
<td>23</td>
<td>35</td>
</tr>
<tr>
<td>Subject 3</td>
<td>40</td>
<td>20</td>
<td>34</td>
</tr>
<tr>
<td>Subject 4</td>
<td>43</td>
<td>14</td>
<td>40</td>
</tr>
<tr>
<td>Subject 5</td>
<td>50</td>
<td>11</td>
<td>36</td>
</tr>
<tr>
<td>Subject 6</td>
<td>42</td>
<td>24</td>
<td>30</td>
</tr>
<tr>
<td>Subject 7</td>
<td>43</td>
<td>25</td>
<td>32</td>
</tr>
<tr>
<td>Subject 8</td>
<td>35</td>
<td>17</td>
<td>38</td>
</tr>
<tr>
<td>Mean</td>
<td>43</td>
<td>19</td>
<td>36</td>
</tr>
<tr>
<td>SEM</td>
<td>2</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

\(\Delta V_{\text{max}}\) per cent = Relative increase in flow with helium compared with air at 50 per cent of vital capacity; \(\Delta V_{\text{max}}\) per cent = \(\Delta V_{\text{max}}\) Hel \(\times V_{\text{max}}\) air + \(\Delta V_{\text{max}}\) air \(\times 100\). \n
\* Significant difference when compared with control by t test for paired data, \(p < 0.05\).

† Flow on helium curve decreased below air curve before flows became equal at volume of isoflow (\(V_{\text{Iov}}\)).
FVC and flow would have occurred, since the MEFV curve would have been shifted over the volume axis for its entire course. Static recoil pressure at TLC would also have been decreased. Static recoil pressure at TLC was, however, unchanged by intubation. Although slow vital capacity decreased with intubation, the decrease was minimal (180 ± 40 ml) and not sufficient to support this possible explanation of the decreased flow rates.

Another factor that must be considered in interpreting flows is alveolar gas compression. Ingram and Schilder demonstrated that flow was underestimated when plotted against expired volume measured at the mouth by a spirometer, as in this study, compared with changes in thoracic gas volume measured by body plethysmography. They attributed this discrepancy to alveolar gas compression. During forced expiration the extent of gas compression is related to increased effort, which causes compression of the chest. At most lung volumes, the volume of expired gas measured with a spirometer is less than the decrease in lung volume measured by plethysmography. A given value for flow will therefore be recorded at a larger apparent lung volume. In this study this effect was minimized somewhat because maximum values for flow were used in all results. Nevertheless, this factor cannot be excluded, particularly in considering the five subjects whose expiratory efforts increased while they were intubated. The artifact created by alveolar gas compression would also increase the slope of the lower portion of the MFSR curve, since $V_{E,max}$ would be more greatly underestimated at lower lung volumes. This could explain the apparent increases in $G_{as}$ observed in three subjects with intubation.

Since topical anesthesia of the airway was achieved prior to intubation, its influence on airway dynamics must also be considered. Ultrasonic aerosols increase airway resistance in patients with obstructive pulmonary disease and in healthy anesthetized patients whose tracheas are intubated. Similar bronchoconstriction might have been anticipated from the lidocaine mist used in this study, but data from other healthy subjects indicated slight bronchodilation, presumably from direct or indirect effects of the anesthetic on airway smooth muscle tone. The same effect may have rendered the airways more collapsible with intubation and contributed to decreasing expiratory flows.

In summary, endotracheal intubation in normal subjects decreased peak inspiratory and expiratory flows in a fashion similar to a fixed upper-airway obstruction. In contrast to a simple upper-airway obstruction, the endotracheal tube decreased FVC and expiratory flows at lung volumes below mid-vital capacity. These flows are less dependent on subject effort and not apt to be altered by an artificially added resistance such as the tube. Therefore, the findings suggest the presence of an additional factor in the form of diffuse obstruction in the airways distal to the tube. Responses to helium breathing, nitrogen washout, and calculations of upstream conductance identified constriction of smaller peripheral airways in individual subjects, but other factors such as alveolar gas compression and increased collapsibility of airways may have also contributed to the decreases in measured flow rates.

The author gratefully acknowledges the advice and suggestions of Dudley F. Rochester, M.D., and Robert M. Epstein, M.D., in preparing this manuscript.

References

16. Gelb AF, Klein E: The volume of isoflow and increase in maximal flow at 50 per cent of forced vital capacity during helium–oxygen breathing as tests of small airway dysfunction. Chest 71:396–399, 1977