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Pulmonary Mechanics in Normal Subjects Following Endotracheal Intubation

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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 (FEV_1) was also decreased significantly, whether expressed as actual volume or as FEV_1/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 (ΔN_2 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 FEV_1 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[1] 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.[2] 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.[3] 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 (Shilley 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 H_2O applied pressure. Subjects inspired to near total lung capacity and then relaxed against an occluded mouthpiece. The absence of a leak was verified

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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 H_2O 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 (FEV_1) were measured. Subjects performed at least three FVC maneuvers during each testing condition. The largest values for FEV_1 and FVC were used in the results provided the FVC from which FEV_1 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 ($\dot{V}_{I\,max}$) and peak expiratory flow ($\dot{V}_{E\,max}$) were used in the results.

The lower (intrathoracic) airways were evaluated by maximum expiratory flow–volume (MEFV) 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 MEFV curve for each subject. These composite curves were constructed for each of the three experimental conditions. The largest values at each interval on the MEFV curve were used to minimize gas compression artifacts from excessive effort.[4] To identify possible constriction in the smaller peripheral airways, MEFV 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 ($\dot{V}_{max\,50}$ He). These were compared with $\dot{V}_{max\,50}$ obtained under the same test conditions when subjects breathed air ($\dot{V}_{max\,50}$ air). The increase in flow with helium was termed $\Delta\dot{V}_{max\,50}$ and expressed as a percentage of the flows with air: $\Delta\dot{V}_{max\,50}$ per cent = ($\dot{V}_{max\,50}$ He − $\dot{V}_{max\,50}$ air) ÷ $\dot{V}_{max\,50}$ air × 100.[5] The largest value for $\dot{V}_{max\,50}$ He was used to calculate $\Delta\dot{V}_{max\,50}$ provided the FVC from which it was obtained was within 5 per cent of that with air. The MEFV 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.[6] The point on the two curves where flows became identical and continued to residual volume was identified as the volume of isoflow ($V_{iso\,\dot{V}}$), which was expressed as percentage of FVC above residual volume. $V_{iso\,\dot{V}}$ for each testing condition was recorded as the mean of three such comparisons.

The closing volume test was performed using Anthonisen's modification[7] of Fowler's single-breath nitrogen test.[8] 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 N_2 sampled at the mouthpiece by a rapid-response N_2 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 N_2 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 N_2 concentration per liter (ΔN_2 per cent/l).[9] 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 (P_L) was estimated as the difference between the pressure at the airway opening and esophageal pressure.[10] 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 H_2O). 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. P_L 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 P_L due to esophageal muscle contraction. To characterize the effect of lung recoil on the ability to generate airflow, P_L was plotted against maximum expiratory flow at the same lung volume using MEFV 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_{us}) 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,[11] expiratory flow ($\dot{V}_{E\,max}$) = $P_L \times G_{us}$.

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 adaptor 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 ($\dot{V}_{I\,max}$) and peak expiratory flow ($\dot{V}_{E\,max}$) were decreased to about half their control values with intubation, significantly more than with the external resistance.

Composite MEFV curves with flows plotted at 10 per cent intervals of FVC are displayed for each subject in figure 1. The curves are matched at total lung capacity as recommended when FVC values are not equal.[12] During the initial half of vital capacity, flows were decreased by the external resistance and even more so by intubation. On the other hand, during the latter 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.

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

	Control	External Resistance	Tracheal Intubation
Slow vital capacity (VC) (l)	5.8 ± 0.3	5.8 ± 0.4	5.6 ± 0.3
Forced vital capacity (FVC) (l)	5.8 ± 0.3	5.7 ± 0.3	5.1 ± 0.3*
Forced expiratory volume in 1 sec (FEV_1) (l)	4.4 ± 0.2	4.2 ± 0.3	3.5 ± 0.2*
FEV_1/FVC (per cent)	77 ± 2	76 ± 2	68 ± 1*
Peak inspiratory flow ($\dot{V}_{I\,max}$) (l/sec)	7.4 ± 0.6	4.6 ± 0.4*	3.6 ± 0.2*
Peak expiratory flow ($\dot{V}_{E\,max}$) (l/sec)	8.7 ± 0.3	5.6 ± 0.4*	4.4 ± 0.4*

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

When helium was breathed, the normal $\Delta\dot{V}_{max\,50}$ 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 ($V_{iso\,\dot{V}}$) increased in five subjects with intubation and decreased in three. In two of the subjects with decreased $V_{iso\,\dot{V}}$, flow on the helium curve decreased below that on the air curve, before becoming equal at $V_{iso\,\dot{V}}$. The mean value with intubation (20 ± 2 per cent) did not significantly differ from control. In contrast, $V_{iso\,\dot{V}}$ 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/l 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. $\dot{V}_{E\,max}$ at corresponding levels of P_L below 70 per cent VC was decreased with intubation compared with control and external resistance (fig. 4). Upstream

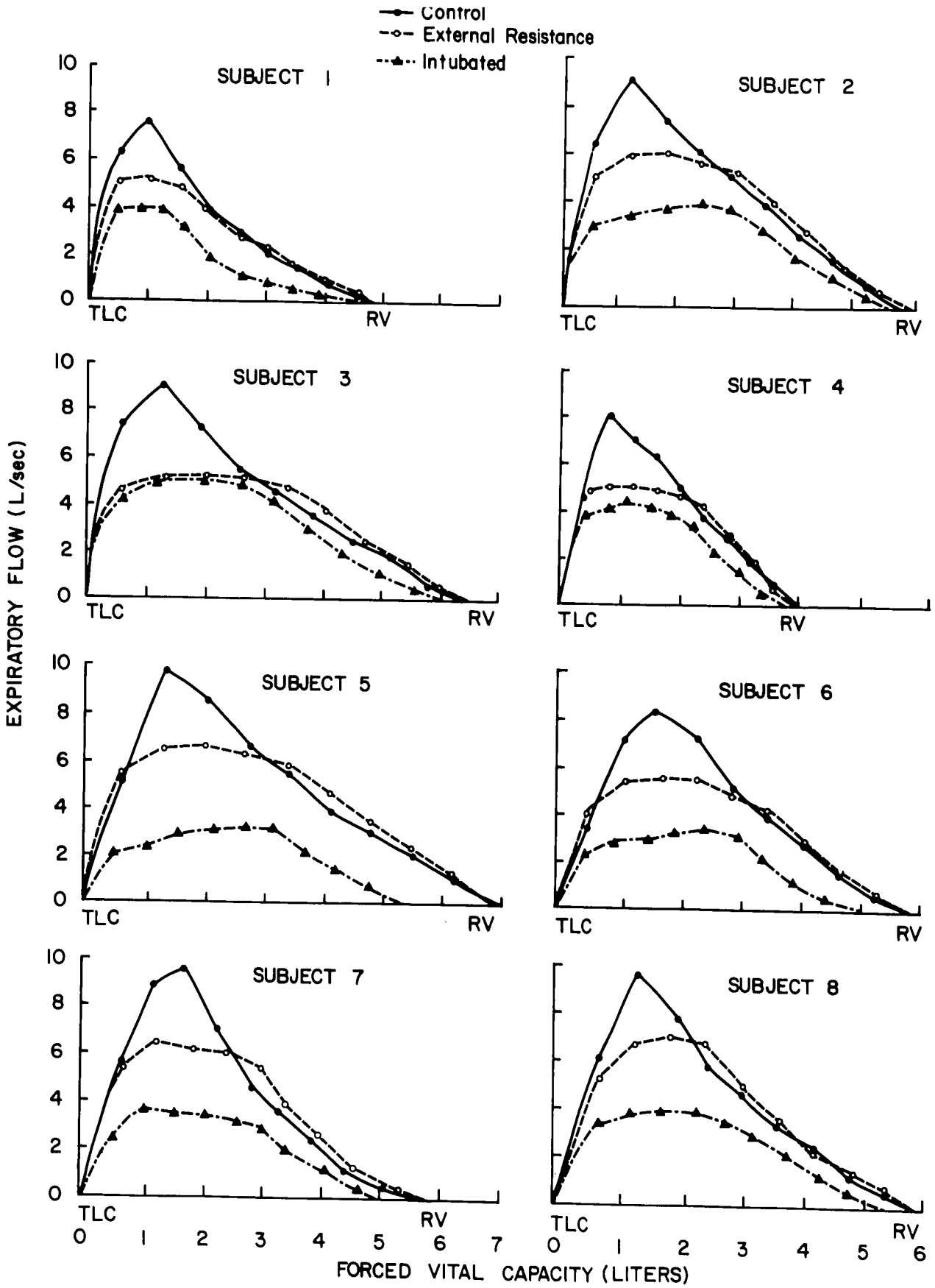

FIG. 1. Composite maximum expiratory flow–volume curves for eight normal subjects during the three test conditions. Curves are aligned at total lung capacity (TLC) and maximum expiratory flow is plotted at 10 per cent intervals of measured forced vital capacity from TLC to residual volume (RV).

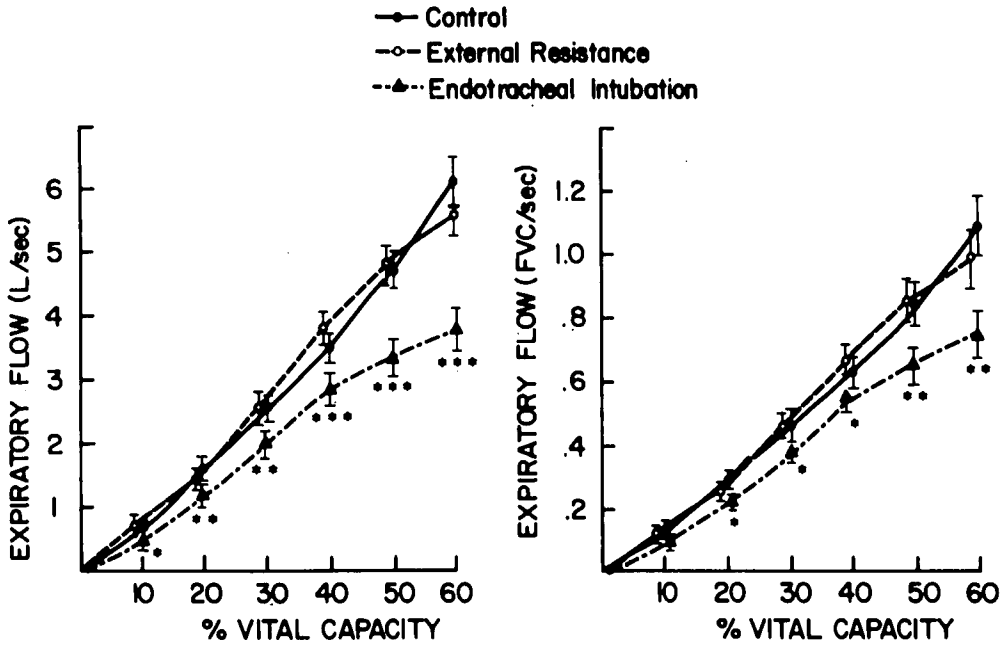

FIG. 2. Mean values for maximum expiratory flow are plotted at 10 per cent intervals of vital capacity above residual volume. Data are from eight normal subjects. Bars indicate ±1 SEM. In the left panel, flow is plotted in l/sec, whereas in the right panel it is expressed as fractions of forced vital capacity per sec (FVC/sec).

conductance (G_{us}) calculated from the slope of the MFSR curves between 50 per cent and 30 per cent of VC was 0.6 ± 0.2 l/sec/cm H_2O for control, compared with 0.7 ± 0.1 l/sec/cm H_2O with the external resistance. The mean G_{us} with intubation (0.45 ± 0.11 l/sec/cm H_2O) was not decreased significantly from control.

Maximum transpulmonary pressure during FVC maneuvers was 69 ± 5 cm H_2O in the control state, 72 ± 5 cm H_2O with the external resistance, and 74 ± 10 cm H_2O 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_2O/l/sec, while that of the adaptor (external resistance) was 1.8 cm H_2O/l/sec (fig. 5). At a flow of 1.0 l/sec the resistances were 5.8 and 3.2 cm H_2O/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

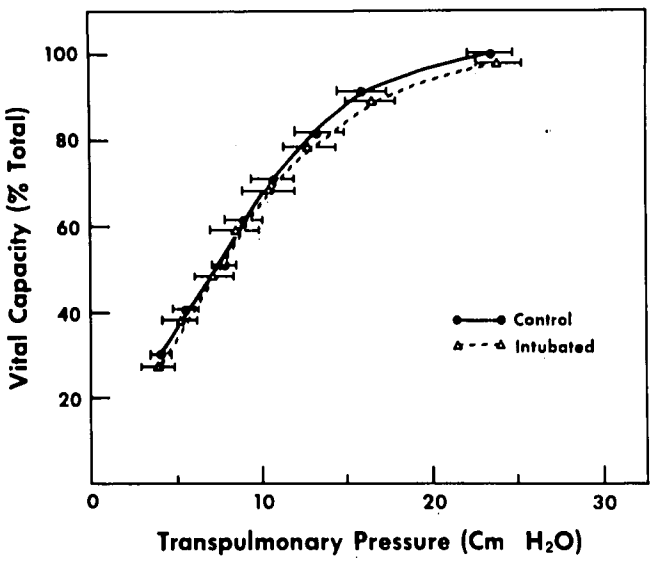

FIG. 3. Mean deflation quasi-static presure–volume curves for eight subjects before and after intubation. Data points for transpulmonary pressure are plotted at intervals of 10 per cent of control vital capacity. Bars represent 1 SEM.

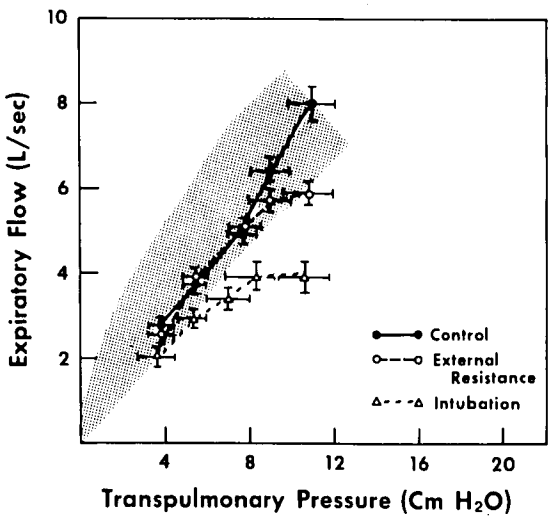

FIG. 4. Maximum-flow static recoil (MFSR) curves during the three test conditions. Simultaneous determinations of expiratory flow and transpulmonary pressure are plotted at intervals of 10 per cent of vital capacity (VC) between 70 and 30 per cent VC. Values are plotted as mean ± SEM for both flow and pressure. Shaded area indicates range of normal values taken from data of Cherniack (Pulmonary Function Testing. Philadelphia, W. B. Saunders, 1977, p 207).

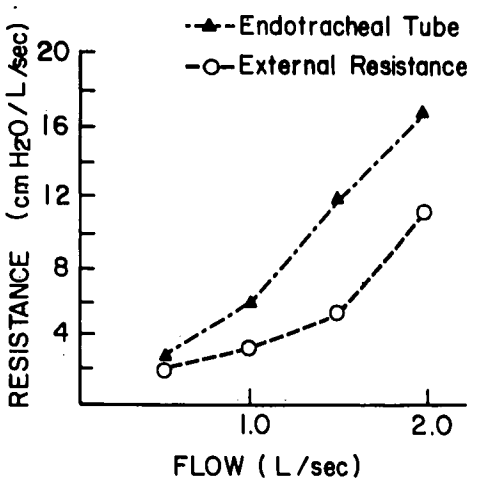

FIG. 5. Resistance measurements for the 8.0-mm ID endotracheal tube and the 8.0-mm ID endotracheal tube adaptor used as the external resistance in this study. Calculations were made from measurements of the reductions in pressure during constant flows of 0.5 to 2.0 l/sec.

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]

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 liklihood 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 FEV_1 observed during intubation. If maximal flows were decreased over a greater portion of vital capacity, FEV_1, 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

† Gal TJ, Suratt PM: Resistance to breathing with endotracheal intubation. Abstracts of Scientific Papers, American Society of Anesthesiologists, Chicago, 1978, pp 473–474.

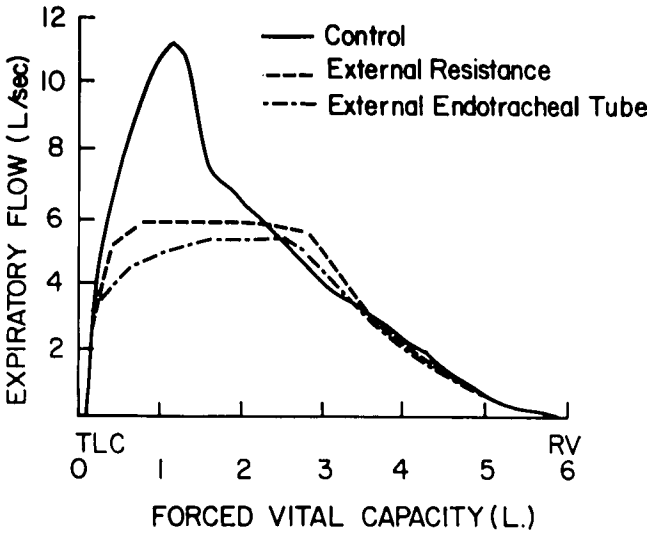

FIG. 6. Maximum expiratory flow–volume curves generated by the author with the mouthpiece as used in the study (control), an 8.0-mm ID endotracheal tube adaptor (external resistance), and an externally held endotracheal tube (25 cm long, 8.0 mm ID). Expiratory flow (l/sec) is plotted on the ordinate against forced vital capacity in liters on the abscissa. TLC = total lung capacity; RV = residual volume.

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.[15] The decreased flows below the midpoint 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.[16] The effects of helium inhalation can be explained by the concept of equal pressure points (EPP) proposed by Mead.[11] A normal $\Delta\dot{V}_{max\,50}$ with helium and a normal or decreased $V_{iso\,\dot{V}}$ 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 $\Delta\dot{V}_{max\,50}$ and increased $V_{iso\,\dot{V}}$ 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 $\Delta\dot{V}_{max\,50}$ and $V_{iso\,\dot{V}}$ 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 airway disease who demonstrated large helium-induced flow increases at mid-vital capacity when severe upper airway obstruction was simulated.[17]

Although not specific, the closing volume test has been described as a sensitive indicator of constriction is small airways.[18] Similarly, the slope of Phase III is a sensitive measure of non-uniform ventilation even in mild peripheral airway disease,[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

TABLE 2. Responses to Breathing Helium during the Three Test Conditions

	Control		External Resistance		Intubation	
	$\Delta\dot{V}_{max\,50}$ (Per Cent)	$V_{iso\dot{V}}$ (Per Cent)	$\Delta\dot{V}_{max\,50}$ (Per Cent)	$V_{iso\dot{V}}$ (Per Cent)	$\Delta\dot{V}_{max\,50}$ (Per Cent)	$V_{iso\dot{V}}$ (Per Cent)
Subject 1	50	20	45	14	35	25
Subject 2	43	23	35	13	31	14†
Subject 3	40	20	34	15	35	19
Subject 4	43	14	40	11	27	20
Subject 5	50	11	36	13	18	21
Subject 6	42	24	30	12	55	18†
Subject 7	43	25	32	12	49	29
Subject 8	35	17	38	20	37	18
Mean	43	19	36	14*	36	20
SEM	2	2	3	1	4	2

$\Delta\dot{V}_{max\,50}$ per cent = Relative increase in flow with helium compared with air at 50 per cent of vital capacity; $\Delta\dot{V}_{max\,50}$ per cent = $\dot{V}_{max\,50}$ He − $\dot{V}_{max\,50}$ air ÷ $\dot{V}_{max\,50}$ air × 100.

* 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 ($V_{iso\dot{V}}$).

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.[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.

The MFSR curve (fig. 4) is a pressure–flow curve that characterizes the resistance of the upstream segment (R_{us}).[21] The driving pressure for flow along this segment is the elastic recoil pressure (P_L). The conductance of this segment (G_{us}), the reciprocal of resistance, is lowered by decreased P_L or increased frictional resistance of small airways. Since the mean G_{us} was not decreased significantly by intubation and P_L was unchanged, small-airway constriction could not be demonstrated for the group as a whole, but five of the eight subjects showed decreased values for G_{us} when their tracheas were intubated.

Other possible causes for the decreased FVC and expiratory flows over the entire vital capacity include a leak around the cuff or herniation of the cuff over the lumen of the tube, but there was no evidence for these. Another more likely explanation is a decreased inspiratory volume at the start of the forced expiration. If subjects when intubated had not inspired fully to total lung capacity (TLC), an apparent decrease in

FVC and flow would have occurred, since the MEFV curve would have been shifted over the volume axis for its entire course.[22] 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.[23] 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 $\dot{V}_{E\,max}$ would be more greatly underestimated at lower lung volumes. This could explain the apparent increases in G_{us} 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[24] and in healthy anesthetized patients whose tracheas are intubated.[25,26] 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.[27]

‡ Gal TJ: Airway dynamics following ultrasonic mists of lidocaine in normal subjects. Abstracts of Scientific Papers, American Society of Anesthesiologists, Chicago, 1978, pp 479–480.

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.

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References

1. Colgan FS, Liang JQ, Borrow RE: Noninvasive assessment by capacitance respirometry of respiration before and after extubation. Anesth Analg (Cleve) 54:807–813, 1975
2. Jones JG, Fraser RB, Nadel JA: Effect of changing airway mechanics on maximum expiratory flow. J Appl Physiol 38:1012–1021, 1975
3. Nadel JA: Mechanisms controlling airway size. Arch Environ health 7:179–182, 1963
4. Ingram RH, Schnilder DP: Effect of gas compression on pulmonary pressure, flow, and volume relationship. J Appl Physiol 21:1521–1526, 1966.
5. Despas PJ, Leroux M, Macklem PT: Site of airway obstruction in asthma as determined by measuring maximal expiratory flow breathing air and a helium–oxygen mixture. J Clin Invest 51:3235–3243, 1972
6. Hutcheon M, Griffin P, Levison H, et al: Volume of isoflow, a new test in detection of mile abnormalities of lung mechanics. Am Rev Resp Dis 110:458–465, 1974
7. Anthonisen NR, Danson J, Robertson PC, et al: Airway closure as a function of age. Resp Physiol 8:58–65, 1969
8. Fowler WS: Lung function studies. III: Uneven pulmonary ventilation in subjects and in patients with pulmonary disease. J Appl Physiol 2:283–299, 1949
9. Buist AS, Ross BB: Quantitative analysis of the alveolar plateau in diagnosis of early airway obstruction. Am Rev Resp Dis 108:1078–1087, 1973
10. Milic-Emili J, Mead J, Turner JM, et al: Improved technique for estimating pleural pressure from esophageal ballons. J Appl Physiol 19:207–211, 1964
11. Mead J, Turner JM, Macklem PT, et al: Significance of the relationship between lung and recoil and maximum expiratory flow. J Appl Physiol 22:95–108, 1967
12. Bouhys A: The Physiology of Breathing. New York, Grune and Stratton, 1977, p 213
13. Ferris BG, Mead J, Opie LH: Partitioning of respiratory flow resistance in man. J Appl Physiol 19:653–658, 1964

14. Kreitzer SM, Saunders NA, Tyler HR, et al: Respiratory muscle function in amyotrophic lateral sclerosis. Am Rev Resp Dis 117:437–447, 1978
15. Miller RD, Hyatt RE: Evaluation of obstructing lesions of the trachea and larynx by flow–volume loops. Am Rev Resp Dis 108:475–481, 1973
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
17. Lavelle TF, Rotman HH, Weg JG: Isoflow-volume curves in the diagnosis of upper airway obstruction. Am Rev Resp Dis 117:845–852, 1978
18. McCarthy DS, Spencer R, Greene R, et al: Measurement of "closing volume" as a simple and sensitive test for early detection of small airway disease. Am J Med 52:747–753, 1972
19. Buist AS: New tests to assess lung function: The single breath nitrogen test. N Engl J Med 293:438–440, 1975
20. Benson MK, Newberg LA, Jones JG: Nitrogen and bolus closing volumes: Differences after histamine-induced bronchoconstriction. J Appl Physiol 38:1088–1091, 1975
21. Macklem PT, Mead J: The physiological basis of common pulmonary function tests. Arch Environ Health 14:5–9, 1967
22. Clement J, Van De Woestijne KP: Variability of maximal expiratory flow–volume curves and effort independency. J Appl Physiol 31:55–62, 1971
23. Ingram RH, Schilder DP: Effect of thoracic gas compression on the flow volume curve of the forced vital capacity. Am Rev Resp Dis 94:56–63, 1966
24. Cheney FW, Butler J: The effects of ultrasonically produced aerosols on airway resistance in man. ANESTHESIOLOGY 29: 1099–1106, 1968
25. Waltemath CL, Bergman NA: Increased respiratory resistance provoked by endotracheal administration of aerosols. Am Rev Resp Dis 108:520–525, 1973
26. Cheney FW, Butler J: Effects of ultrasonic aerosols on total respiratory resistance of the intubated patient. ANESTHESIOLOGY 32:456–458, 1970
27. McFadden ER, Newton-Howes J, Pride NB: Acute effects of inhaled isoproterenol on the mechanical characteristics of the lungs of normal man. J Clin Invest 49:779–790, 1970