Effects of Endotracheal Intubation on Normal Cough Performance

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To assess the effects of endotracheal intubation on normal cough function, the tracheas of eight healthy volunteers were intubated with a standard 8.0-mm-I.D. tube. Measurements were made of expiratory flow, transpulmonary pressure, and expired volume during a cycle of three successive bursts of maximum voluntary coughing initiated after inspiration to total lung capacity. Results following intubation were compared with findings with subjects breathing through a 25-mm-I.D. mouthpiece (control). Maximum cough pressures (Pmax) following tracheal intubation were similar to control values, but with the tube in place, airflow began well before Pmax was reached and did not cease fully between cough bursts. Flows at Pmax declined with each successive burst of coughing during both experimental conditions, but with tracheal intubation, the flows were significantly lower than control. Resistance to flow at Pmax increased with each successive cough and increased significantly above control during the third burst of coughing while the subjects' tracheas were intubated. The total volume of the three control coughs was 76 ± 3 per cent of vital capacity. This was similar following intubation, although cough duration was significantly longer. These findings indicate that tracheal intubation does not impair ability to develop normal cough pressures despite preventing glottic closure. The normal timing of flow and pressure is disrupted such that flow occurs early—as in a forced expiratory maneuver, and the flow is not fully interrupted during the cough sequence. Flows are submaximal as a result of the resistance of the tube and continue to decline as the lung volume decreases and airways are compressed. Because the tube is noncollapsible, high flow rates may be necessary to achieve the linear velocities required for normal airway cleansing through the tube. Such flows may be achieved only during the initial cough after full inspiration. (Key words: Cough: voluntary; Intubation, endotracheal. Lung; function; respiratory resistance.)

The normal cough mechanism involves a forceful sudden expulsion of air from the lungs, which serves to clear airways down to the level of the medium-sized bronchi.1,2 The presence of an endotracheal tube would be expected to have significant effects on this mechanism, for several reasons. First, the fixed resistance of the tube might decrease expiratory flow during coughing. Second, the presence of the tube prevents normal sphincter action of the larynx, which is important for the initial buildup of driving pressure prior to coughing. Third, the noncollapsible endotracheal tube splints the intrathoracic trachea and thus prevents the near-obliteration of the tracheal lumen by the posterior membranous tracheal wall.

The dynamics of a voluntary cough have been examined both in healthy persons and in patients with bronchitis.3-6 Although clinical observations suggest that patients whose tracheas are intubated cannot cough,7 no measurements of actual cough function in this state have been made. This article describes alterations in the mechanics of normal cough that result from intubation, and considers the possible impact of these alterations on cough efficiency.

Methods

Eight healthy men, ranging in age from 22 to 29 years, served as subjects. The work was performed in conjunction with experiments reported previously.8 Cough performance was studied with the subjects in a semirecumbent position. A cycle of three consecutive bursts of maximum voluntary coughing was recorded and the relationships between expired volume, flow, driving pressure, and time were analyzed. Measurements made in subjects whose tracheas were intubated with a 25-cm-long 8.0-mm-I.D. endotracheal tube (Shiley Laboratories) were compared with measurements obtained while the subjects breathed through a 25-mm-I.D. mouthpiece (control). The endotracheal tube was inserted to its full depth. Auscultation of the chest and measurement of vital capacity established that the tube was not endobronchial. The cuff was inflated to produce a leak-free seal, which was verified by maintenance of constant pressure (monitored by an aneroid manometer) for at least 5 sec after subjects inspired to total lung capacity and relaxed against an occluded mouthpiece. Prior to insertion of the tube, topical anesthesia of the airway was produced by lidocaine, 8–10 ml, 4 per cent, inhaled from an ultrasonic nebulizer (Mistogen®).

Vital capacity and expired volume during coughing were measured and recorded by a waterless, rolling-seal spirometer and recorder (Model #840, Ohio Medical Products). All volumes were corrected to body temperature and pressure saturated with water vapor (BTPS). Flow at the mouthpiece was determined by electronic differentiation of the spirometer volume signal. Driving pressure (transpulmonary pressure) was estimated as the difference between esophageal

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and mouth pressures. A 10-cm latex balloon containing 1.0 ml air was placed in the midoesophagus and connected to a differential pressure transducer (Validyne® MP 45; range 250 cm H$_2$O) by polyethylene (PE 200) tubing 100 cm long. The opposite port of the transducer was connected to a pressure tap in the mouthpiece by a similar length of tubing. A simultaneous record of transpulmonary pressure and flow was provided by a strip-chart recorder (Gould). The dynamic response of the system was adequate to detect and measure differences of the magnitude encountered in the experiment.

Subjects performed the coughing maneuvers by inspiring fully to total lung capacity (TLC) and then producing with maximum effort, a burst of three successive coughs uninterrupted by further inspiration. The peak deflections of the transpulmonary pressure trace for each cough burst were recorded as the maximum driving pressures (P$_{max}$). Maximum transpulmonary resistance was estimated by dividing P$_{max}$ by the expiratory flow at that point (the elastic component of resistance was ignored since it is small compared with the flow-resistive component). Onset of a cough was marked by the point at which transpulmonary pressure began to increase above baseline, and cessation of a cough was established by a return of pressure and flow to baseline after the third burst of cough. In subjects whose tracheas were intubated, the onset of flow coincided with the measurable increase in transpulmonary pressure. This point was used to measure the onset of coughing.

Before each set of coughing maneuvers, slow vital capacity was measured. During the inspiration to total lung capacity (TLC), transpulmonary pressure was recorded. A comparison with pressures obtained after the full inspiration that preceded coughing ensured that all maneuvers were initiated at TLC.

The results are reported as mean values from two successive technically satisfactory cough tracings. All values are expressed as means ± SEM. Statistical analysis consisted of the Student t test for paired data. Significant difference between the two test conditions was assessed at the 95 per cent confidence level.

**Results**

The patterns of pressure and flow during coughing in the control state are in accord with previous reports (fig. 1).\textsuperscript{3-5} As the cough sequence began, the glottis closed, expiratory muscles contracted, and transpulmonary pressure began to increase (point A). The onset of flow (point B), which corresponds to opening of the glottis, occurred abruptly, but not for almost 0.2 sec after the onset of the increase in pressure, and only after considerable pressure had developed. Flow increased rapidly, then decreased abruptly. As in the first cough, glottic closure and increased pressure preceded the onset of flow in each of the remaining cough bursts. During the initial burst of cough, the flow recorded at the point of maximum pressure (point C) approximated the peak flow. During the second and third cough bursts, brief peak-flow transients appeared prior to a more sustained plateau-like flow at maximum pressure.

When subjects coughed with the endotracheal tube in place, flow began (point B) at the same time as transpulmonary pressure began to increase. Flow increased gradually, paralleling the pressure changes. In contrast to the control state, flows did not return entirely to baseline between individual bursts of cough. Flow patterns assumed two relatively distinct forms. In four subjects whose maximum transpulmonary pressures (P$_{max}$) were below control values when their tracheas were intubated, flow and pressure tended to increase and decrease simultaneously (fig. 1B). The remaining four subjects generated P$_{max}$ values higher than control when their tracheas were intubated. During the initial cough, flow and pressure were parallel. During subsequent bursts, peak-flow transients appeared, followed by a distinct plateau-like flow as P$_{max}$ was reached (fig. 1A).

Maximum driving pressure (P$_{max}$) measured during the initial burst of cough was nearly identical to that measured during the second and third bursts. The average P$_{max}$ of the three cough bursts increased in four intubated subjects and decreased in four. The mean value of 79 ± 10 cm H$_2$O did not represent a significant change from the control value of 85 ± 7 cm H$_2$O. The time interval from the beginning of cough to the point of P$_{max}$ was 0.4 ± 0.04 sec in the subjects whose tracheas were intubated (interval B–C). This was significantly longer than the time required to reach P$_{max}$ in the control state (interval A–C), which averaged 0.2 ± 0.01 sec.

The flows achieved at P$_{max}$ decreased with each of the three bursts of cough in the control state as lung volume decreased. Flows during the second and third bursts were 68 and 39 per cent of those during the initial burst (fig. 2). With tracheal intubation, these flows were decreased significantly to about 59 per cent of control during the initial burst of cough, and they continued to decrease as more volume was exhaled during the second and third bursts to 75 and 35 per cent of their initial values.

The resistance to flow at P$_{max}$ can be computed as the ratio of P$_{max}$ to flow at this point. Control cough resistance increased progressively as more volume was exhaled with each burst of cough (fig. 3). With the endotracheal tube in place, the resistance during the...
first cough was increased by an average of 54 per cent, but four subjects demonstrated slightly decreased resistances. Thus, a significant difference from control could not be demonstrated. Resistance increased steeply with each subsequent cough, as more volume was exhaled. Only during the third burst of cough with the endotracheal tube, when resistance approached twice the control value, was the increase significant compared with the third cough of the control series.

The total volume of the three control coughs was 4.4 ± 0.2 liters, or about 75 per cent of measured vital capacity (table 1). This was not altered significantly by tracheal intubation. Of this volume, 47 per cent was exhaled during the first cough while the remaining two coughs delivered 28 and 25 per cent, respectively. These relative proportions also did not change significantly with endotracheal intubation (table 1). The duration of the three coughs was significantly longer while subjects' tracheas were intubated, averaging 2.3 sec, compared with 1.7 sec in the control state. As a result, during control cough sequences, subjects were able to exhale volume at the rate of 45 per cent of vital capacity/sec which decreased with tracheal intubation to 35 per cent of vital capacity/sec.

**Discussion**

The bursts of cough performed by the subjects, resembled a series of forced expirations at decreasing lung volumes. The difference is that with coughing, airway pressure normally builds up behind the closed glottis before flow starts. In contrast, during forced expiration, the airway-pressure increase and the onset of flow occur simultaneously. Between coughs, glottic closure allows pressure in airways to equalize. Airways regain their normal size for that particular lung volume before flow begins during the next burst. With the endotracheal tube in place, subjects were unable to use this glottic closure mechanism, but they were still able to develop the peak driving pressure for coughing.

The presence of the endotracheal tube did not lessen the buildup of peak pressure during coughing. However, by preventing normal glottic closure, the tube allowed flow to begin as soon as pressure began to increase and, in most cases, the tube allowed flow to continue between cough bursts. In the control state, glottic closure occurred normally. As a result, flow did not occur until considerable pressure had developed, which produced interruptions of flow between bursts of coughing. With tracheal intubation, the normal
rapid increase to maximal flow was slowed, and the contribution of the trachea to this maximal flow was decreased. Pressure and flow increased together, with the result that endotracheal intubation converted each cough burst into a maneuver that more closely resembled forced expiration, with the difference being that higher transpulmonary pressures occur with coughing.

Expiratory flow normally decreases with lung volume during forced expiration. Similarly, the flows achieved at $P_{\text{max}}$ during each successive burst of cough decreased as more volume was exhaled (fig. 2). Following tracheal intubation, the flows were decreased significantly at high lung volumes with the first cough burst. The flows showed a continuous decrease with expired volume that paralleled the changes in control state. This apparent parallel decrease in flow rates with tracheal intubation could be explained by a failure of subjects to inspire fully to TLC before initiating their cough sequence. However, since neither vital capacity nor transpulmonary pressure at total lung capacity differed significantly from control, this is unlikely.

The combination of decreased flows and constant high driving pressures resulted in a significant increase in calculated transpulmonary resistance at low lung volumes during the third burst of coughing.

Table 1. Volumes Expired During Coughing (±SEM)

<table>
<thead>
<tr>
<th></th>
<th>Volume of Each Cough Burst (Per Cent of Total)</th>
<th>Total Volume of 3 Coffugs (liters)</th>
<th>VC* (liters)</th>
<th>Cough Volume (Per Cent of VC*)</th>
<th>Total Cough Duration (sec)</th>
<th>Per Cent of VC* Expired per Sec</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cough 1</td>
<td>Cough 2</td>
<td>Cough 3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>$47 \pm 3$</td>
<td>$28 \pm 2$</td>
<td>$25 \pm 2$</td>
<td>$4.4 \pm 0.2$</td>
<td>$5.8 \pm 0.3$</td>
<td>$76 \pm 3$</td>
</tr>
<tr>
<td>Tracheal intubation</td>
<td>$41 \pm 5$</td>
<td>$33 \pm 3$</td>
<td>$24 \pm 4$</td>
<td>$4.2 \pm 0.2$</td>
<td>$5.6 \pm 0.2^\dagger$</td>
<td>$75 \pm 4$</td>
</tr>
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* VC = slow vital capacity determined spirometrically.  
† $P < 0.05$; denotes significance of difference from control value by paired t test.
This high resistance may reflect airway constriction. However, maximal flows over this range of lung volume are independent of effort, so values for resistance obtained may, to a great extent, reflect subject effort. The peak-flow transients, and the plateau-like flow pattern, that was observed in subjects whose $P_{\text{max}}$ was higher after intubation, suggest that marked airway collapse occurred distal to the tube as a result of this expiratory effort. Topical anesthesia of the airway, as produced for this study, may have rendered the airways more susceptible to compressive forces.†

The question then arises to what extent, if any, do the changes imposed by the endotracheal tube alter cough effectiveness. The problem of assessing the efficacy of coughing has been approached in several ways, none of which has been widely accepted. The most frequently employed measurements are maximal cough pressures⁵⁻⁷ and cough volume.⁶⁻⁶,ⁱ¹ Cough pressures largely reflect effort, while cough volume is related more to overall ventilatory efficiency. In our study, neither measurement was significantly affected by tracheal intubation.

Cough efficacy has also been assessed by measurements of peak flow.⁶⁻¹³ High flow rates are important, but for a cough to be truly effective in removing secretions from the airway, there must also be a significant decrease in airway caliber.¹⁴ Airway narrowing increases the linear velocity and kinetic energy of the flowing gas. A decrease in tracheal cross-sectional area, for example, to half normal size, will increase the kinetic energy of the gas stream fourfold. Some authors assert that this airway narrowing is even more important than the high expiratory flows.¹⁵,¹⁶ Maximal airway narrowing occurs after the transient peak flow and at the time of peak pressure development ($P_{\text{max}}$).¹⁷,¹⁸ This suggests that the flows at $P_{\text{max}}$ are more significant than the more transient peak flows in assessing cough action.

Cineradiographic and endoscopic studies have shown that the width of the tracheal shadow is decreased by half during a cough and that the lumen size is decreased from its normal area of 2.5 cm²¹⁰ to about 0.25 cm²¹⁰ Linear velocities during control coughing can be estimated by dividing the flow at $P_{\text{max}}$ by this cross-sectional area. Our estimates were 312, 212, and 124 m/sec, respectively. Assuming the endotracheal tube in place substitutes its fixed lumen size (0.5 cm²) for that of the trachea, the same calculated velocities are 92, 68, and 32 m/sec. These estimates assume that the tube extends the length of the trachea, i.e., 10–12 cm below the vocal cords. In subjects whose tracheas may have been longer, the calculations underestimate true linear velocity through the trachea, since a portion of the trachea below the tube is subject to compressive narrowing. The noncollapsible tube does not permit high velocities, and thus retards effective clearance of material through the trachea. It would appear that optimal tussive efficiency during tracheal intubation would require the shortest possible tube to minimize resistance, and that the tube should be inserted the least possible distance below the vocal cords in order to allow a longer segment of trachea to undergo compressive narrowing.

Although it is difficult to conclude on a quantitative basis to what extent endotracheal intubation alters cough efficiency, observations from this study provide some insight into the effect of an endotracheal tube on cough dynamics. Despite eliminating the normal mechanism of glottic closure, endotracheal intubation does not impair the ability to generate normal pressures during coughing. Instead, the presence of the tube alters the normal timing of pressure and flow such that a cough more closely resembles normal forced expiration. The resistance of the tube did not allow maximal flows to be reached at high lung volumes in spite of increased effort. At low lung volumes, this increased effort produced compressive narrowing of the large airways distal to the tube. This compression can, by means of a tussive squeeze, transport secretions to the area of the trachea. However, the noncollapsible endotracheal tube tends to maintain the tracheal lumen rather than allowing it to collapse. As a result, high flow rates are needed to achieve the linear velocities necessary to clear secretions. These are likely to accumulate at the area around the end of the tube unless subsequent coughs can be initiated at high lung volumes, to achieve the highest flows possible.

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