ORIGINAL ARTICLE

Changes in Respiratory Mechanics After Tracheostomy

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Objective: To determine the effects of tracheostomy on respiratory mechanics and work of breathing (WOB).

Design: A before-and-after trial of 20 patients undergoing tracheostomy for repeated extubation failure.

Setting: Surgical intensive care unit at a university teaching hospital and a level I trauma center.

Patients: A consecutive sample of 20 patients who met extubation criteria ($Pa{O_2}$, >55 mm Hg; pH >7.30; and respiratory rate, <30/min on room air continuous positive airway pressure after 20 minutes) but failed extubation on 2 occasions were eligible for the study.

Interventions: Respiratory mechanics, lung volumes, and WOB were measured before and after tracheostomy.

Main Outcome Measures: Patients in whom extubation fails often progress to unassisted ventilation after tracheostomy. The study hypothesis was that tracheostomy would result in improved pulmonary function through changes in respiratory mechanics.

Results: Data are given as means ± SDs. After tracheostomy, WOB per liter of ventilation (0.97 ± 0.32 vs 0.81 ± 0.46 J/L; $P<.09$), WOB per minute (8.9 ± 2.9 vs 6.6 ± 1.4 J/min; $P<.04$), and airway resistance (9.4 ± 4.1 vs 6.3 ± 4.5 cm H_2O/L per second; $P<.07$) were reduced compared with breathing via an endotracheal tube. These findings, however, do not fully explain the ability of patients to be liberated from mechanical ventilation after tracheostomy. In 4 patients who were extubated before tracheostomy, WOB was significantly greater during extubation than when breathing through an endotracheal or tracheostomy tube (1.2 ± 0.19 vs 0.81 ± 0.24 vs 0.77 ± 0.22 J/L).

Conclusions: We believe that the rigid nature of the tracheostomy tube represents reduced imposed WOB compared with the longer, thermoliable endotracheal tube. The clinical significance of this effect is small, although as respiratory rate increases, the effects are magnified. In patients in whom extubation failed, WOB may be elevated because of incomplete control of the upper airway. Future studies should evaluate the cause of increased WOB after extubation.

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THE TIMING of tracheostomy during the treatment of respiratory failure remains controversial.[1-3] Results of a recent report[4] suggest that early tracheostomy does not impact intensive care unit length of stay, incidence of pneumonia, or mortality compared with continued translaryngeal intubation. Yet, tracheostomy often results in clinical improvement in respiratory function and liberation from mechanical ventilation. The exact mechanisms for this improvement remain ill-defined.

Differences in work of breathing (WOB) and pressure time product (PTP) between endotracheal and tracheostomy tubes of the same internal diameter in a lung model were previously studied.[5] A slight reduction in WOB with tracheostomy tubes was found owing to a shorter length and more rigid form. Wright et al[6] demonstrated that the in vivo resistance of endotracheal tubes exceeds the in vitro resistance because of thermoliability of materials and the tortuous translaryngeal path. Davis et al[5] also showed that the misconception that tracheostomy significantly reduces dead space is mere myth.

Despite these findings, the mechanism by which tracheostomy allows weaning from mechanical ventilation after failed extubation attempts remains elusive. This study compares the changes in respiratory mechanics before and after tracheostomy in a group of non–head-injured surgical patients in whom extubation failed on 2 occasions.

RESULTS

There were statistically significant reductions in WOB per minute (8.9 ± 2.9 vs 6.6 ± 1.4 J/L per minute; $P = .04$), and PEEP

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PATIENTS, MATERIALS, AND METHODS

Twenty patients (14 men and 6 women) were studied. All patients were recovering from acute lung injury and were intubated orally via a size 7.0 (n = 4) or 8.0 (n = 16) endotracheal tube. Mean ± SD age of the patients was 58 ± 13 years (range, 26-70 years). Mean ± SD duration of ventilatory support before the study was 16 ± 3.3 days (range, 9-23 days). Mean ± SD static compliance for the group was 49 ± 6 mL/cm H_2O (range, 39-61 mL/cm H_2O). All patients had been weaned to a fraction of inspired oxygen (FIO_2) concentration of less than 0.45, a positive end-expiratory pressure (PEEP) of 5 cm H_2O, and a pressure support of less than 10 cm H_2O. Thirteen patients were injured in motor vehicle crashes, 5 patients had undergone repair of abdominal aortic aneurysms, and 2 patients had aspiration pneumonia after major abdominal surgery.

All patients were receiving enteral nutritional support via nasoenteric tubes. Energy (calorie) needs were measured by indirect calorimetry and were adjusted 1 to 2 times per week. Sedation was accomplished with administration of fentanyl citrate or haloperidol lactate as needed.

All patients had resolution of their primary lung injury and were judged ready for extubation by the intensive care unit service. All patients underwent a room air continuous positive airway pressure (CPAP) trial at 5 cm H_2O with no pressure support. After 20 minutes, an arterial blood sample was obtained and was analyzed for pH, $PaCO_2$, PaO_2, and oxygen saturation. If the patient's PaO_2 was greater than 55 mm Hg, spontaneous respiratory rate was less than 30/min, pH was greater than 7.30, and tidal volume was greater than 4 mL/kg, the CPAP trial was considered "passed." Subjective observation of the patient's mental status, ability to control the airway, and ability to handle secretions were made by the intensive care unit physicians and staff. If these were considered adequate, the patient was extubated.

In all study patients, extubation failed on 2 occasions after all the extubation criteria were met. We chose to study these patients based on our previous observations that after repeated extubation failure, many patients are liberated from the ventilator after tracheostomy. By choosing patients with 2 previous extubation failures, we believe our chances of finding differences in measured variables would be enhanced. The reasons for failure included hypoxemia (n = 9), hypercarbia (n = 3), excessive secretions (n = 3), and a combination of the 3 (n = 5). Each patient was scheduled for tracheostomy by the attending physician to aid in weaning from ventilatory support. The protocol was approved by the institutional review board of the University of Cincinnati, Cincinnati, Ohio, and informed consent was obtained.

Six to 10 hours before tracheostomy, patients had an esophageal balloon inserted transnasally (SmartCath, Bicore Monitoring Systems, Irvine, Calif) into the distal third of the esophagus to monitor esophageal pressure (Pes). Patients were placed in the semi-Fowler position with the head elevated a minimum of 30°. Correct placement of the balloon was confirmed using an airway occlusion test, as described by Baydur et al.[7] A variable orifice flow transducer and pressure tap (VariFlex, Bicore Monitoring Systems) was placed on the end of the endotracheal tube to monitor airway flow and pressure. Signals from the flow transducer and esophageal balloon were transmitted to a portable respiratory monitor (Bicore CP-100, Bicore Monitoring Systems). The performance of this device has been validated elsewhere and found to be accurate for clinical measurements.[8] Patients were disconnected from the ventilator and allowed to breathe via a continuous flow of gas at an FIO_2 concentration equal to that set on the ventilator.

After 10 minutes of stabilization, measurements were made. The following variables were measured: tidal volume, respiratory rate, airway pressure, flow, and Pes. These measurements were used to determine all other variables. Work of breathing was expressed in joules per liter and was calculated from the area subtended by the Pes created during inspiration and the relaxation curve of the chest wall (estimated chest wall compliance was 200 mL/cm H_2O). Pressure time product, an estimate of respiratory muscle metabolic work, was measured as the integral Pes and the duration of respiratory muscle contraction and expressed in centimeters of water per second per minute. Intrinsic PEEP was calculated as the difference in Pes from the end of expiration to the initiation of inspiratory flow. Expiratory airway resistance was calculated from the difference in transpulmonary pressure (airway pressure–Pes) divided by the difference in flow. All signals were recorded continuously for 10 minutes and were stored breath by breath in a personal computer for later analysis.

Ten to 12 hours after tracheostomy, the same measurements were repeated. This time was allowed to eliminate the effects of anesthetic agents on respiratory function. As before, the patient was disconnected from the ventilator and allowed to breathe from a continuous flow of gas at the previous FIO_2 concentration. The flow transducer was placed on the proximal end of the tracheostomy tube, and placement of the esophageal catheter was reconfirmed or adjusted as necessary.

Four patients were extubated before tracheostomy and were reintubated. Two patients were extubated at the attending physician's request and 2 patients were self-extubated. In these patients, the same variables were measured while the patients breathed through a mouthpiece.

Data are represented as mean ± SD, and each point represents the average of at least 40 breaths during the 10-minute measurement. Data before and after tracheostomy were compared using a paired *t* test. A probability of less than .05 was considered significant.

(2.9 ± 1.7 vs 1.6 ± 1.0 cm H_2O; $P = .02$). There was a tendency toward reductions in PTP, expiratory airway resistance, and WOB (joules per liter).

Posttracheostomy tidal volume was reduced slightly (329 ± 104 vs 312 ± 119 mL; $P = .47$), as was respiratory rate (28 ± 5 vs 26 ± 6/min; $P = .51$). **Table 1** depicts the data from the study. All patients were successfully weaned to an FIO_2 concentration of 0.40 delivered via a high-flow aerosol system through a tracheostomy collar within 24 hours of tracheostomy (duration, 8 ± 3 hours; range, 2-15 hours).

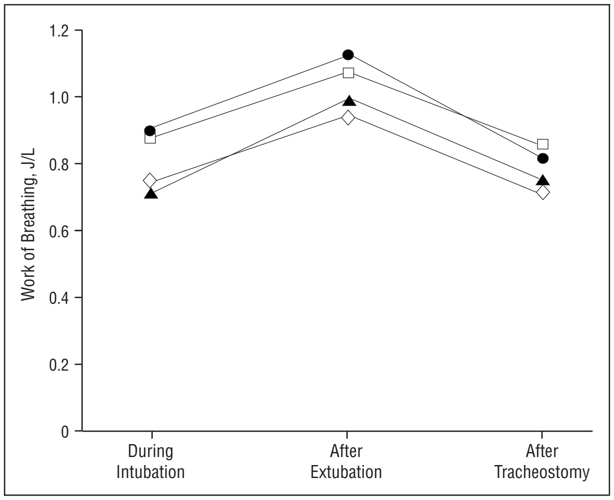
In each of the 4 patients who were extubated before tracheostomy, there was a significant increase in WOB (**Figure**) during extubation compared with breathing via an endotracheal or tracheostomy tube. Tidal volume increased, as did respiratory rate and other variables (**Table 2**).

Table 1. Changes in Respiratory Variables Before and After Tracheostomy

Variable	Before Tracheostomy	After Tracheostomy	*P*
Tidal volume, mL/breath	329 ± 104	312 ± 119	.47
Minute volume, L/min	9.2 ± 3.0	8.1 ± 3.1	.26
Respiratory rate, breaths/min	28 ± 5	26 ± 6	.51
Intrinsic positive end-expiratory pressure, cm H_2O	2.9 ± 1.7	1.6 ± 1.0	.02
Pressure time product, cm H_2O/s per minute	236 ± 122	155 ± 101	.09
Work of breathing, J/L	0.97 ± 0.32	0.81 ± 0.46	.09
Work of breathing, J/min	8.9 ± 2.9	6.6 ± 1.4	.04
Expiratory airway resistance, cm H_2O/L per second	9.4 ± 4.1	6.3 ± 4.5	.07

Table 2. Changes in Respiratory Variables in 4 Patients Who Were Extubated Before Tracheostomy

Variable	Endotracheal Tube	Extubated	Tracheostomy
Tidal volume, mL/breath	383 ± 107	429 ± 124	378 ± 81
Minute volume, L/min	11.1 ± 3.1	14.5 ± 4.2	10.6 ± 2.7
Respiratory rate, breaths/min	29 ± 8	34 ± 6	28 ± 5
Pressure time product, cm H_2O/s per minute	277 ± 109	328 ± 132	252 ± 97
Work of breathing, J/L	0.8 ± 0.2	1.2 ± 0.2	0.8 ± 0.2
Work of breathing, J/min	9.0 ± 2.7	17.4 ± 2.8	8.2 ± 2.2



Changes in the work of breathing in 4 patients during endotracheal intubation, after extubation, and after tracheostomy.

COMMENT

The major findings of this study suggest that after tracheostomy, WOB and airway resistance are reduced compared with breathing via an endotracheal tube. These findings agree with laboratory results[5] comparing WOB between endotracheal and tracheostomy tubes in a lung model. These changes are, however, small and in our opinion do not fully explain facilitated weaning often seen after tracheostomy. However, the improvements seen when breathing through a tracheostomy tube are magnified as respiratory rate and tidal volume increase. Another important finding is the statistically and clinically significant increase in WOB after extubation compared with breathing via either a tracheostomy or endotracheal tube.

The imposed WOB caused by the presence of an endotracheal tube has been studied by numerous investigators.[8-12] Decreasing the diameter of the tube and increasing flow (patient demand) through the tube increase the imposed work. Internal diameter is the most important factor, as described by the Poiseuille law, in which pressure drop across the tube is inversely proportional to the fourth power of the radius during laminar flow. In the presence of turbulent flow, pressure drop increases by the fifth power of the radius.

Compared with the number of studies evaluating the untoward effects of the endotracheal tube on respiratory work, the tracheostomy tube has been relatively ignored. Cullen[13] evaluated the effects of tracheostomy in 14 patients with chronic obstructive pulmonary disease. He found that compared with mouth breathing, tracheostomy resulted in a reduction in physiologic dead space, a slight decrease in oxygen consumption, and a slight increase in airway resistance. This work is not immediately applicable to our study because of the difference in patient disease and absence of the endotracheal tube. Cavo et al[14] found that tracheostomy tubes created airway resistance greater than that imposed by the normal upper airway. As expected, decreasing diameter resulted in greater resistance. These authors suggested that the largest diameter tube be used for prolonged tracheostomy. Yung and Snowdon[15] evaluated the resistance of 3 varieties of tracheostomy tubes in the laboratory. They found that design (radius of curvature and roughness of the inner surface) and length of the tube were important in determining resistance.

Results of a previous laboratory study[5] demonstrate that for equivalent inside diameter, endotracheal tubes created greater imposed work than did tracheostomy tubes. We believe that the additional length of the endotracheal tube contributes to the increased resistance but that the major contributor is the tortuous path traveled by the endotracheal tube. In our laboratory study, the endotracheal tube was placed through the upper airway of a resuscitation mannequin. In previous comparisons of endotracheal and tracheostomy tubes, both were placed on the bench top. Comparison of endotracheal and tracheostomy tubes in vivo, as accomplished in this study, seems to support this hypothesis. There were decreases in WOB (joules per liter and joules per minute), PTP, airway resistance, intrinsic PEEP, and minute volume after tracheostomy. These changes represent trends in many instances but seem to suggest that tracheostomy improves the efficiency of breathing compared with breathing via an endotracheal tube. In addition, Wright et al[6] found that the in vivo resistance of endotracheal tubes was significantly greater than anticipated from in vitro studies. They attributed this finding to changes in the shape of the thermoliable endotracheal tubes and adherence of secretions to the inner lumen. Wright et al also demonstrated that flow through endotracheal tubes in vivo was turbulent, causing resistance

Table 3. Comparison of Dead Space, Length, and Internal Diameter (ID) of Endotracheal and Tracheostomy Tubes

Tube	ID, mm	Length, cm	Dead Space, mL
Endotracheal			
No. 6.0	6.0	31.5	11.0
No. 7.0	7.0	34.5	15.0
No. 8.0	8.0	35.5	18.0
No. 8.5	8.5	36.5	24.0
Tracheostomy*			
Size 4	5.0	10.0	3.0
Size 6	7.0	12.0	5.0
Size 8	8.5	12.0	6.0
Size 10	9.0	12.0	8.0

*****Note that tracheostomy tube size is not equal to ID.*

to increase with decreasing diameter to the fifth power of the radius.

Our results did not show any difference in tidal volume before and after tracheostomy. It is a misconception that tracheostomy reduces dead space. Compared with mouth breathing, tracheostomy can reduce dead space, as shown by Cullen.[13] However, the added length of the endotracheal tube only results in an increase in dead space of 10 to 18 mL. **Table 3** compares dead space, length, and internal diameter values for endotracheal and tracheostomy tubes used in this study.

The genesis for this study lies in the frequent observation that patients in whom extubation repeatedly fails are quickly weaned from mechanical ventilation after tracheostomy. Results of this study suggest that tracheostomy reduces respiratory work, although this reduction may not be the only explanation. We believe the finding that patients have significant increases in respiratory work after extubation may be the most important finding of our trial. The ability to wean a patient from mechanical ventilation after tracheostomy frequently occurs after failed extubation attempts. In these patients, tracheostomy allows improved secretion clearance, allows simple initiation and discontinuation of ventilatory support, and eliminates the variable of upper airway control. Extubation criteria are frequently quoted with the caveat "ability to control the upper airway." This nebulous definition is subjective and difficult to quantify yet seemingly critically important.

Nathan et al[16] had similar findings during a study designed to use pressure support to overcome the imposed WOB caused by the endotracheal tube. They found that compared with breathing through an endotracheal tube, the WOB and PTP were increased after extubation. The increase in WOB was nearly 30% (0.74 vs 1.04 J/L) and was statistically significant. They also found the presence of "fluttering" in the flow-volume tracings of the patients who were extubated. This finding is often seen in patients with upper airway obstruction, and the authors hypothesized that after extubation, lack of upper airway control causes increased respiratory work. In a follow-up study, Ishaaya et al[17] measured tracheal and glottic diameter after extubation. They confirmed their previous results, finding a consistent increase in WOB after extubation compared with endotracheal tube breathing. However, they did not find evidence of airway narrowing. In fact, the cross-sectional area of the intact upper airway was 3 times that of the endotracheal tube. This finding may suggest that the site of upper airway narrowing is in the oropharynx. If upper airway closure in this instance is similar to that seen during sleep apnea, the use of CPAP by mask or noninvasive ventilation may be helpful. DeHaven et al[18] used CPAP by face mask to treat postextubation hypoxemia successfully.

Results of this study suggest that after tracheostomy, WOB and PTP are reduced compared with breathing through an endotracheal tube. These findings are due to reduced resistance of the tracheostomy tube compared with the thermoliable endotracheal tube, which can become deformed in the upper airway. During extubation, patients with inadequate upper airway control have WOB greater than during spontaneous breathing through either type of artificial airway. Successful discontinuation of mechanical ventilation after tracheostomy represents improved airway control compared with spontaneous breathing via the intact upper airway in select patients. The improvements in respiratory efficiency after tracheostomy compared with endotracheal tube breathing are small and unlikely to fully explain the ability to discontinue mechanical ventilation after tracheostomy.

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REFERENCES

1. Lewis RJ. Tracheostomies: indications, timing, and complications. *Clin Chest Med.* 1992;13:137-149.
2. Rodriguez JL, Steinberg SM, Luchette FA, Gibbons KJ, Taheri PA, Flint LM. Early tracheostomy for primary airway management in the surgical care setting. *Surgery.* 1990;108:655-659.
3. Plummer AL, Gracey DR. Consensus conference on artificial airways in patients receiving mechanical ventilation. *Chest.* 1989;96:178-180.
4. Sugerman HJ, Wolfe L, Pasquale MD, et al. Multicenter, randomized, prospective trial of early tracheostomy. *J Trauma Injury Infect Crit Care.* 1997;43:741-747.
5. Davis K Jr, Branson RD, Porembka D. A comparison of the imposed work of breathing with endotracheal and tracheostomy tubes in a lung model. *Respir Care.* 1994;39:611-616.
6. Wright PE, Marini JJ, Bernard GR. In vitro versus in vivo comparison of endotracheal tube airflow resistance. *Am Rev Respir Dis.* 1989;140:10-16.
7. Baydur A, Behrakis PK, Zin WA, et al. A simple method for assessing the validity of the esophageal balloon technique. *Am Rev Respir Dis.* 1982;126:788-791.
8. Banner MJ, Blanch PB, Kirby RR. Imposed work of breathing and methods of triggering a demand-flow, continuous positive airway pressure system. *Crit Care Med.* 1993;21:183-190.
9. Shapiro M, Wilson RK, Casar G. Work of breathing through different sized endotracheal tubes. *Crit Care Med.* 1986;14:1028-1031.
10. Bersten AD, Rutten AJ, Vadig AE, Skowronski GA. Additional work of breathing imposed by endotracheal tubes, breathing circuits, and intensive care ventilators. *Crit Care Med.* 1989;17:671-677.
11. Bhatt SB, Patel CB, Tan IKS, Vernasco D, Tam YH. Imposed work of breathing during tracheal pressure triggering using a demand valve CPAP system. *Respir Care.* 1996;41:1028-1031.
12. Brochard L, Rua F, Lorino H, et al. Inspiratory pressure support compensates for the additional work of breathing caused by the endotracheal tube. *Anesthesiology.* 1991;75:739-745.
13. Cullen JH. An evaluation of tracheostomy in pulmonary emphysema. *Ann Intern Med.* 1963;58:953-960.
14. Cavo J, Ogura JH, Sessions DG, et al. Flow resistance in tracheostomy tube. *Ann Otolaryngol.* 1973;82:827-830.
15. Yung MW, Snowdon SL. Respiratory resistance of tracheostomy tubes. *Arch Otolaryngol.* 1984;110:591-595.
16. Nathan SD, Ishaaya AM, Koerner SK, Belman MJ. Prediction of minimal pressure support during weaning from mechanical ventilation. *Chest.* 1993;103:1215-1219.
17. Ishaaya AM, Nathan SD, Belman MJ. Work of breathing after extubation. *Chest.* 1995;107:204-209.
18. DeHaven CB, Hurst JM, Branson RD. Post-extubation hypoxemia treated with continuous positive airway pressure by face mask. *Crit Care Med.* 1985;13:46-48.