

Response to Tubular Airway Resistance in Normal Subjects and Postoperative Patients

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Critically ill patients must often breathe spontaneously through an endotracheal tube that acts as a fixed inspiratory and expiratory tubular airway resistor. Although this practice is common, its effect on the pattern of breathing is not known. The mean breathing patterns of seven normal, healthy male subjects and eight male patients who had undergone upper abdominal surgery 2-4 days previously were studied breathing through a mouthpiece fitted in random order with a 5, 6, 7, 8, or 15 mm diameter (17 mm long) resistor. These diameters were selected because they simulate the pressure-flow relationships of adult endotracheal tubes. With the 15 mm aperture, the patients had a greater breathing frequency (f) than did the normal subjects (21 ± 5 [SD] vs. 14 ± 4 breaths/min, $P < 0.01$) as well as a smaller mean tidal volume (V_T). In both groups, minute ventilation (\dot{V}_E) and f progressively decreased as resistance was increased by decreasing the aperture size from 15 to 6 mm. In the normal subjects, but not the patients, V_T also progressively decreased. When the diameter was decreased from 6 mm to 5 mm, there were increases in V_T and decreases in f that were more marked in the normal subjects. In both groups, the changes in \dot{V}_E were accompanied by decreases in mean and peak inspiratory and expiratory flow rates. Throughout the study, oxygen consumption (\dot{V}_{O_2}) and carbon dioxide production (\dot{V}_{CO_2}) did not change. This, coupled with the decreases in \dot{V}_E resulted in decreases in the ventilatory equivalents to CO_2 and O_2 (\dot{V}_E/\dot{V}_{CO_2} , \dot{V}_E/\dot{V}_{O_2}). This study demonstrates that increasing tubular airway resistance significantly alters respiratory pattern without affecting gas exchange. When interpreting the breathing pattern of patients whose trachea is intubated, the clinician should consider the effects of the fixed tubular resistance. These results also reinforce previous admonitions that in patients with respiratory dysfunction, it is important to use as large an endotracheal tube as possible and allow spontaneous respirations only once substantial clinical improvement has occurred. (Key words: Airway: resistance. Oxygen: consumption. Ventilation: resistance; work.)

DURING INTERMITTENT MANDATORY VENTILATION (IMV) and often during continuous positive airway pressure (CPAP) breathing, critically ill patients must breathe spontaneously through an endotracheal tube that acts as

a fixed inspiratory and expiratory tubular resistor. Such patients frequently have underlying lung disease and/or injuries to the chest and abdomen, which in themselves are associated with altered breathing patterns.¹⁻³ In order for the clinician to interpret the significance of the abnormal breathing patterns in patients breathing through endotracheal tubes, the effects of breathing through equivalent resistance must be understood. Although a great deal of physiologic information is available describing the effects of loaded breathing on ventilation, data describing the specific ventilatory response to the resistances associated with endotracheal tubes and connectors in patients and normal subjects are sparse. This may be of particular clinical interest because anecdotal observations have suggested that a cause of the failure to be able to discontinue mechanical ventilation may be due to the resistance of the endotracheal tube, particularly when a tube with a small diameter is used.

This study examines the metabolic and ventilatory effects caused by fixed tubular airflow resistance in both normal subjects and in a group of postoperative surgical patients with a breathing pattern characterized by a rapid respiratory frequency and small tidal volumes (V_T). The resistances used mimicked the degrees of airway resistance caused by endotracheal tubes. However, since the subjects did not actually have an endotracheal tube in place but were breathing through resistors placed in a mouthpiece, the experimental conditions may not be completely analogous to the clinical situation. This is because during endotracheal intubation, the upper airways and oral cavity, each with their individual airflow-altering characteristics and sensory nerve endings, are bypassed while direct sensory stimulation of the trachea occurs.

Methods

SUBJECT SELECTION

Two groups were studied. Group 1 consisted of seven normal, healthy male subjects age 26-37 yr, and Group 2 consisted of eight male patients who had undergone major upper abdominal surgical procedures (e.g., gastrectomy, radical cystectomy, resection of abdominal aortic aneurysm) 2 to 4 days prior to the study. The latter group ranged in age from 27 to 83 yr (mean 49 ± 10 [SD] yr).

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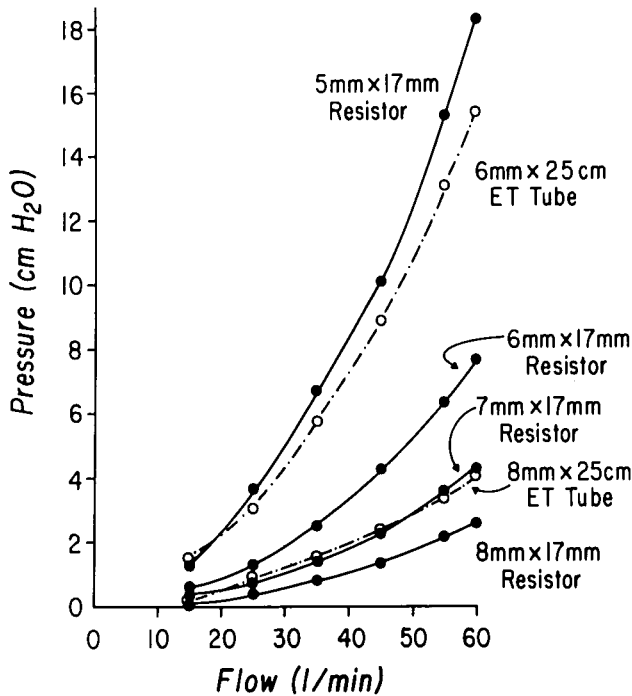


FIG. 1. Pressure-flow curves of the resistances used in this study of two sizes of endotracheal (ET) tubes used in clinical practice.

PROTOCOL

Measurements were made using a canopy-spirometer-computer system while each subject was spontaneously breathing through a mouthpiece (plus nose clip) fitted in

random order with a 5, 6, 7, 8 or 15 mm internal diameter (17 mm long) resistor. With each resistor, continuous measurements were made over a 10-min period after stable levels of oxygen consumption per min (\dot{V}_{O_2}) and carbon dioxide production per min (\dot{V}_{CO_2}) had been achieved. The dead space of the system was less than 13 ml. The studies were performed with the subjects in the supine position.

The airflow resistance caused by the tubular resistors was measured and compared with standard adult endotracheal tubes (6 mm and 8 mm diameter; 25 cm lengths). The pressure drop across the resistors at airflows of 15, 25, 35, 45, 55, and 60 l/min was determined using a Satham® transducer (Model PM 5 ± 0.7 - 350, Gould Inc, Oxnard, CA) (fig. 1).

MEASUREMENTS

The canopy-spirometer-computer system (fig. 2), which has been described in detail previously,⁴ is composed of a head canopy connected to a spirometer (Med-Science® Model 470) and a Prime 300® computer. The canopy is a rigid, transparent head chamber with a neck seal, ventilated by a continuous airstream. The spirometer (a low-friction piston) connected to the canopy provides a breath-by-breath record of changes in lung volume. Gas composition in the canopy is continuously sampled and analyzed by means of a LIRA® infrared CO₂ analyzer (Model 200 FP) and a Servomex® paramagnetic O₂ analyzer (Model OA 250). Spirometry and gas exchange data

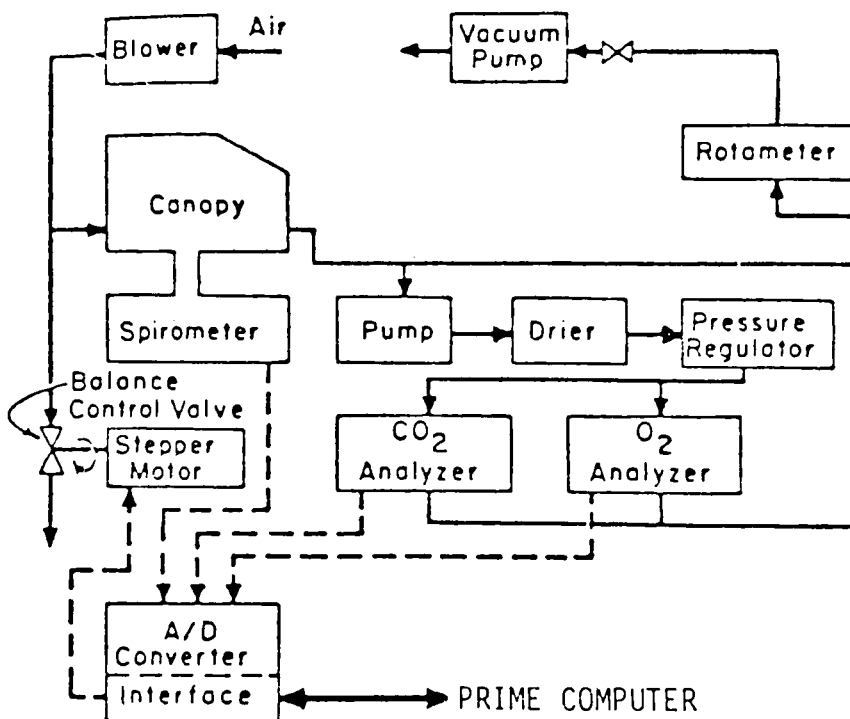


FIG. 2. The canopy-spirometer-computer system consists of a lucite head canopy ventilated by a balanced 40 l/min airflow. The spirometer is a Med-Science® Model 470.

TABLE 1. Ventilatory Variables

Resistor Diameter (mm)	\dot{V}_E (l/min)	V_T (ml)	f (min ⁻¹)	T_I (s)	T_E (s)	T_I/T_{TOT}
Group 1 (n = 7)						
15	7.99 ± 2.0	553 ± 134	14 ± 4	1.50 ± .19	2.85 ± .85	0.36 ± .05
8	7.20 ± 1.4	506 ± 113	14 ± 3	1.62 ± .17*	2.75 ± .67	0.38 ± .04
7	6.50 ± 1.2*†‡	505 ± 120*	13 ± 3	1.92 ± .31*†‡	2.84 ± 0.6	0.40 ± .04*
6	6.50 ± 1.8*†‡	502 ± 155	13 ± 4*	1.93 ± .71*	2.99 ± 1.2	0.40 ± .05*
5	6.34 ± 1.1*†‡	598 ± 183†	11 ± 3*†‡	2.46 ± .46*†‡	3.28 ± 1.2†	0.44 ± .06*
Group 2 (n = 8)						
15	8.15 ± 3.5	394 ± 154	21 ± 5¶	1.42 ± 0.7	1.85 ± 0.5	0.42 ± .05¶
8	7.63 ± 3.6	393 ± 145	20 ± 5	1.45 ± 0.4	2.08 ± 0.7	0.42 ± .05
7	6.86 ± 2.2	395 ± 147	17 ± 4*	1.44 ± 0.2	2.10 ± 0.7	0.42 ± .06
6	6.54 ± 2.0*	378 ± 136	17 ± 4*	1.57 ± 0.4*†‡	2.03 ± 0.6	0.44 ± .04
5	6.66 ± 2.1*	408 ± 201	16 ± 5*	1.77 ± 0.6§	2.13 ± 0.7*	0.45 ± .04*†‡

Values are means ± SD. \dot{V}_E = minute ventilation; V_T = tidal volume; f = frequency; T_I = inspiratory time; T_E = expiratory time. T_I/T_{TOT} = inspiratory duty cycle.

* Significantly different than 15 mm resistor ($P < 0.05$).

† Significantly different than 7 mm resistor ($P < 0.05$).

‡ Significantly different than 8 mm resistor ($P < 0.05$).

§ Significantly different than 6 mm resistor ($P < 0.05$).

¶ Significantly different than Group 1 values ($P < 0.01$).

are acquired and processed by the digital computer. Airflow to the canopy is set at 40 l/min and is controlled to provide a stable spirometer baseline position. Computer-executed algorithms quantify each breath and determine V_T , frequency (f), \dot{V}_{CO_2} , \dot{V}_{O_2} , peak inspiratory and expiratory flows, and inspiratory (T_I) and expiratory (T_E) time. Inspiration time is taken from the start of inspiration to start of expiration. Mean inspiratory and expiratory flows (V_T/T_I , V_T/T_E) and the ventilatory equivalent for O_2 and CO_2 (\dot{V}_E/\dot{V}_{O_2} , \dot{V}_E/\dot{V}_{CO_2}) at room air were calculated. An accuracy of ±10 ml in V_T measurements is achieved for breathing frequencies in the range of 5–40 breaths/min. The program excludes all V_T less than 40 ml, as these have been considered too small to represent a breath.

Repeated-measures analysis of variance was used for intragroup comparisons, and unpaired Student's *t* test was used for intergroup comparisons.

Written informed consent was obtained from all subjects. None of the subjects had previously participated in a study involving the application of a respiratory resistor. This study was approved by the Institutional Review Board of Columbia University Health Sciences Center.

Results

Measurements of the pressure–flow relationships of the tubular resistors and endotracheal tubes revealed that the 7-mm resistor had characteristics similar to that of the 8-mm endotracheal tube, while the 5-mm resistor was similar to the 6-mm endotracheal tube (fig. 1).

In the normal subjects of Group 1, \dot{V}_E and V_T progressively decreased as the diameter of resistors was decreased from 15 mm to 6 mm (table 1). With a further decrease to a 5 mm diameter aperture, V_T increased while

f abruptly decreased. Inspiratory time and T_I/T_{TOT} increased significantly as the diameter was decreased to 6 mm, while T_E increased only slightly. When the aperture of the resistor was decreased to 5 mm, both variables increased abruptly even further, as did the inspiratory duty cycle (T_I/T_{TOT}). As the diameters of the resistances were decreased, mean inspiratory flow, along with both peak inspiratory and expiratory flow, decreased, while mean expiratory flow decreased to a lesser degree (table 2). \dot{V}_{O_2} , \dot{V}_{CO_2} , and respiratory quotient were unchanged by the application of the resistances.

In the postoperative patients, \dot{V}_E and f decreased as the diameter of the resistor decreased, while V_T changed little. Respiratory frequency decreased progressively from 21 ± 5 breaths/min with the 15- and 8-mm resistors to 17 ± 4 breaths/min with the 7- and 6-mm resistances. With the 5-mm resistance, f decreased further to 16 ± 5 breaths/min. Both T_E and T_I increased as did T_I/T_{TOT} (table 1). Mean and peak inspiratory and expiratory flows also decreased progressively with decreasing diameter. \dot{V}_{O_2} and \dot{V}_{CO_2} (table 2), along with respiratory quotient, were unchanged throughout the study.

The group of patients 2–3 days after a major abdominal operation responded somewhat differently than did the normal subjects. With the 15-mm resistor, the postoperative patients (Group 2) had a significantly greater f ($P < 0.05$) and a smaller V_T when compared with Group 1. With 8-mm, 7-mm and 6-mm diameter resistors, the patients had no significant change in V_T from control (15 mm), while the normal subjects showed a significant decrease (16%). The decrease in mean frequency was 1 breath/min (7%) in the normal subjects, while it decreased 4 breaths/min (19%) in the patients (table 1). The alterations in breathing pattern seen in the normal subjects (decreased rate (15%) and increased tidal volume (18%)

TABLE 2. Mean and Peak Flow Rates and Metabolic Variables

Resistor Diameter (mm)	V_T/T_I (ml/s)	V_T/T_E (ml/s)	PIF (ml/s)	PEF (ml/s)	\dot{V}_{O_2} (ml/min)	\dot{V}_{CO_2} (ml/min)
Group 1 (n = 7)						
15	402 ± 71	257 ± 68	565 ± 98	441 ± 117	273 ± 35	214 ± 22
8	327 ± 39*	235 ± 50	472 ± 66*	377 ± 62*	267 ± 36	208 ± 28
7	296 ± 33*†‡	226 ± 47*	431 ± 93*	354 ± 68*	266 ± 40	208 ± 30
6	309 ± 70*	209 ± 52*	420 ± 79*	328 ± 62*	264 ± 40	205 ± 27
5	287 ± 45*	210 ± 42	389 ± 47*	323 ± 59*†	276 ± 38	210 ± 25
Group 2 (n = 8)						
15	372 ± 109	301 ± 107	534 ± 135	566 ± 195	232 ± 57	179 ± 37
8	341 ± 120	288 ± 117	479 ± 150	495 ± 167	234 ± 56	181 ± 41
7	293 ± 56*	254 ± 77*	406 ± 74*†‡	384 ± 174	228 ± 54	178 ± 43
6	279 ± 66*†‡	253 ± 47*	398 ± 94†‡	398 ± 84	231 ± 52	178 ± 42
5	279 ± 67*†‡	246 ± 58*†‡	400 ± 121*†‡	401 ± 100†‡	233 ± 53	182 ± 40

Values are means ± SD. V_T/T_I = mean inspiratory flow; V_T/T_E = mean expiratory flow; PIF = peak inspiratory flow; PEF = peak expiratory flow; \dot{V}_{O_2} = oxygen consumption; \dot{V}_{CO_2} = carbon dioxide production.

* Significantly different than 15 mm resistor ($P < 0.05$).

† Significantly different than 7 mm resistor ($P < 0.05$).

‡ Significantly different than 8 mm resistor ($P < 0.05$).

when the 5-mm resistor was compared with the 6-mm resistor) was less marked in the patient group. In the latter, there was only a small decrease in f (6%), with a small increase in V_T (6%). The change in frequency observed in Group 2 (patients) was due to alterations in both T_I and T_E , unlike Group 1, where most of the changes were initially in T_I alone. The mean and peak inspiratory flow rates were similar in magnitude in both the normal subjects and postoperative patients. Yet, both the mean and peak expiratory flow rates were slightly greater in the patient group. Because as airflow increases, so does resistance (fig. 2), it is possible that T_E increased at a lower level of tubular resistance in Group 2, so as to decrease the elevated V_T/T_E , thus, decreasing the airflow resistance to allow for more efficient respiration.

Discussion

Allowing spontaneous respiration through an endotracheal tube is common clinical practice in the operating room, recovery room, and intensive care unit (ICU). The effects of endotracheal tubes on respiratory pattern is of interest to those engaged in respiratory care. Most relevant studies have been performed in anesthetized patients⁵⁻⁷ or healthy volunteers.^{6,8,9} In addition, in previous studies the measurement apparatus used had considerable dead space, which may have been an important variable but was not controlled for. The present study complements these previous observations by examining the effects of tubular airway resistance in a system with very little dead space, both in spontaneously breathing, normal subjects and in a group of patients who had undergone major upper abdominal operations (e.g., gastrec-

tomy, radical cystectomy, resection of abdominal aortic aneurysm) a few days previously. Askanazi *et al.*¹ and others^{2,3} have observed that such patients frequently have an increase in respiratory frequency and a decrease in V_T . This is consistent with the finding in the present study. The normal subjects had a mean frequency of 14 ± 4 breaths/min and a V_T of 553 ± 134 ml, while the patients had a mean frequency of 21 ± 5 breaths/min with a V_T of only 394 ± 154 ml.

Previous studies of the respiratory response to resistive loading have focused mainly on the effects of selectively loading either inspiration or expiration. From these studies,^{8,10,11} as well as those that have explored the effects of loading both inspiration and expiration,^{8,9,12} it is apparent that increased resistance will substantially alter breathing patterns. In all situations, as resistance increases, respiratory rate tends to decrease while V_T increases.^{8,9,12} The slow, deep pattern adopted by these subjects apparently decreases subjective discomfort and minimizes the work of breathing.¹³ This response can be affected by personality traits,¹⁴ age,¹⁵ respiratory pattern,¹⁶ and intrinsic lung pathology.¹⁷ In addition, the greater the resistance load, the more marked the decrease in the response to hypoxia and hypercapnia.¹⁸ However, the inspiratory mouth occlusion pressure increases as resistance increases,¹⁹ indicating that central neuromuscular drive is in fact increased. In anesthetized subjects, resistive loading (inspiratory and expiratory) results in a progressive decrease in V_T but not frequency.⁶ The present study examines the ventilatory and metabolic responses of both normal individuals and postoperative patients to a fixed airway resistance, *i.e.*, both inspiratory and expiratory, this being similar but not analogous to the condition to

which intubated patients are subjected. In addition, the aim of this study was to examine the changes that occur as resistance progressively increases, the clinical correlate being a small kink or a collection of mucous in the endotracheal tube or simply the use of a smaller than optimal endotracheal tube. The degree of resistance used in the present study was less than that used in other studies, as evidenced by the fact that only at the smallest aperture was a deep, slow pattern observed. This allowed for an evaluation of progressive changes in respiratory pattern at levels of resistance less than, as well as equal, to those used in previous studies and may be a more accurate simulation of clinical conditions.

The results in the normal subjects demonstrate that a progressive series of changes occur as the diameter is decreased and as the resistance increases. A decrease in diameter from 15 mm to 8 mm resulted in a decrease in mean \dot{V}_E of 800 ml/min. Eight-millimeter diameter endotracheal tubes are commonly used in ICU patients; the resistance to airflow of these tubes is similar to that of the 7-mm resistor used in this study (fig. 1). The initial decrease in \dot{V}_E was due mainly to a decrease in V_T with a relatively minor change in frequency. When the diameter of the resistor was reduced from 6 mm to 5 mm, an abrupt change in respiratory pattern occurred. Respiratory frequency decreased and V_T increased with preservation of \dot{V}_E . Zechman *et al.*⁹ has suggested that this pattern of respiration maintains alveolar ventilation while increasing ventilatory efficiency by decreasing dead space. Further analysis of the breathing patterns using the scheme of Milic-Emili and Grunstein²⁰ revealed rather substantial alterations in breathing pattern. Mean inspiratory flow (V_T/T_I) progressively decreased as the diameter decreased. This was initially due to both decreases in V_T and increases in T_I . With a reduction to a 5-mm diameter, V_T/T_I continued to decrease due to a rather large increase in T_I and despite an increase in V_T . Also, inspiratory duty cycle increased as resistor diameter decreased. This was due to greater increases in T_I that exceed the increases in T_E (table 1). Inspiration occurs with a greater flow rate than does expiration, since inspiration is the result of inspiratory muscle action. However, expiration is normally passive and has lower flow rates.²¹ To reduce the work of breathing, V_T/T_I , thus, must decrease more than V_T/T_E . This was accomplished by increasing T_I more than T_E while decreasing V_T . Only when a deep and shallow respiratory pattern emerges does T_E increase significantly.

One index of respiratory efficiency is the ventilatory equivalents to O_2 and CO_2 . At the levels of resistance used here, oxygen consumption and carbon dioxide production were unchanged. Since minute ventilation decreased, so did the ventilatory equivalents for O_2 and CO_2 .

This indicates that at least from a metabolic viewpoint, respiration was more efficient, *i.e.*, that for a given metabolic rate there was less \dot{V}_E .

The breathing patterns of postoperative patients consist of an increased respiratory rate, generally in the range of 20 to 30 breaths/min, and a small V_T . The reason for this pattern is unknown, but it has been ascribed to the need to avoid large V_T with its associated incisional pain.² This explanation has been questioned because pain relief fails to abolish completely the alterations in respiratory patterns. Furthermore, patients with sepsis and some patients with nutritional depletion who do not have abdominal or chest wounds often have similar respiratory patterns. Thus, there is the possibility that this is caused by a central mechanism.

The results of this study demonstrate that postoperative patients with an altered pattern of respiration in the unloaded state seem to have a response to tubular airway resistance that, although similar, does differ in some respects from that observed in normal subjects. The reason for these differences is unclear, but may be due to an alteration in central or peripheral neurogenic mechanisms. Possible mechanisms include alterations in the sensitivity of the mechanoreceptors located in the respiratory muscles and pulmonary parenchyma or an inability to alter V_T because of intrinsic diaphragmatic dysfunction. Ford *et al.*²² and Simmoneau *et al.*²³ observed that upper abdominal surgery causes a marked dysfunction of the diaphragm that lasts for up to 7 days and is not reversed by relief of postoperative pain.²³ Another factor to be considered is that the functional residual capacity²⁴ and pulmonary compliance,²⁵ as well as V_T are decreased postoperatively and that further reductions in V_T , like those seen to occur in normals during resistance breathing, may be incompatible with proper gas exchange (*e.g.*, excretion of CO_2) and efficient respiration. Therefore, in situations where resistance is added to such a system, it appears that f rather than V_T is altered. The patients also were substantially older than the normal subjects. Because the perception of resistance decreases as age increases,¹⁵ this may also have been a contributing factor to the differences observed between the normal subjects and patients.

In summary, this study provides data describing the differences in the pattern of ventilation produced by tubular airway resistors of progressively decreasing diameter in normal subjects and postoperative patients. These results reinforce previous admonitions that in patients with respiratory dysfunction, *i.e.*, increased airway resistance and/or decreased compliance, it is important to use as large an endotracheal tube as possible and keep it free of obstructions and secretions as well as only allow spontaneous respirations once substantial clinical improvement has occurred. This is especially important because spon-

taneous ventilation through such tubular resistance in patients with respiratory rates commonly found in patients with impending respiratory failure (30–40 breaths/min) may hasten the onset of failure because of the inability to alter effectively lung and respiratory muscle function.

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