CRITICAL CARE

REVIEW

Equipment review: Tracheal gas insufflation

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Introduction

Tracheal gas insufflation (TGI) is an adjunctive ventilatory technique that delivers fresh gas into the trachea either continuosly or only during a specific segment of the respiratory cycle (phasic flow) [1-4]. Two mechanisms are responsible for improving the efficacy of conventional tidal breaths during TGI [5-7]. First, fresh gas introduced by the catheter during expiration flushes the series (anatomic) deadspace compartment proximal to its tip free of CO_2. Consequently, during the subsequent inspiration, less CO_2 is recycled back to the alveoli thus improving CO_2 elimination. Second, at high catheter flow rates, turbulence generated at the tip of the catheter can enhance gas mixing in regions distal to the catheter tip, thereby contributing to CO_2 removal.

Carbon dioxide elimination during TGI depends on catheter flow rate because fresh gas flushes a greater portion of the proximal deadspace at higher flow rates. Moreover, at higher flow rates, turbulence generated at the catheter tip may further enhance distal gas mixing. The volume of fresh gas introduced into the trachea during TGI depends on expiratory time (TE) and catheter flow rate (Vc). At a certain TE × Vc, fresh gas completely sweeps the proximal anatomic deadspace during expiration. At that point, increasing Vc most likely does not dilute the CO_2 residing in the series deadspace any further. This operational charactertistic of TGI, and the fact that the decrease in the partial pressure of CO_2 ($PaCO_2$) caused by a reduction in total physiologic deadspace fraction (VD/VT) is much less at lower VD/VT, limits the decrement in $PaCO_2$ afforded by TGI at high Vc [8]. Nevertheless, at high Vc (< 10–15 1/min) $PaCO_2$ continues to decrease with increasing Vc, but at a slower rate [8-10]. Once the series deadspace is flushed completely by the fresh gas during expiration, the flow dependence of $PaCO_2$ is thought to be secondary to enhanced turbulent mixing in the airways distal to the catheter tip [5,11,12]. TGI is unlikely to be very effective when the alveolar as opposed to the series compartment dominates the total physiologic deadspace; yet, at small tidal volumes (whenever series deadspace is especially high) or when alveolar ventilation is very low. TGI should be a helpful adjunct to conventional mechanical ventilation (CMV) [8].

Operational characteristics

During TGI, more distal catheter placement improves CO_2 elimination as TGI can flush a greater portion of the deadspace proximal to the catheter tip during exhalation [5,7]. Advancing the catheter also moves the turbulence zone generated by the catheter closer to the periphery, thereby improving the efficacy of TGI. However, for most clinical applications, TGI efficiency is optimized when the catheter tip is positioned within a few centimeters of the main carina [5]. This characteristic of TGI simplifies its clinical application as bronchoscopic guidance of catheter placement may not be necessary in critically ill patients.

TGI increases end-expiratory lung volume (in other words, generates auto-positive end-expiratory pressure) in three ways [5,13]. First, part of the momentum of the discharging jet stream is transferred to the alveoli [14]. Second, placement of the catheter within the trachea decreases its cross-sectional area and increases expiratory resistance and delays emptying. Third, catheter flow through the endotracheal tube, expiratory circuit, and expiratory valve during expiration can build a back pressure that impedes expiratory flow from the lung. Since the magnitude of dynamic hyperinflation depends primarily on the presence of catheter flow at end-expiration, any TGI configuration that injects gas at end-expiration will increase resting lung volume. Consequently, continuous TGI and pan-expiratory TGI (ie gas insufflation timed to occur throughout expiration) generate the same levels of auto-positive end-expiratory pressure.

Because the major mechanisms of TGI is flushing the proximal anatomic deadspace free of CO_2, if lung deflation takes place during expiration secondary to dynamic hyperinflation, the CO_2 that is constantly exhaled by the lung can decrease the efficacy of TGI. We tested this

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possibility in normal dogs, and our data suggest that the volume of fresh gas insufflated by the catheter, rather than the flow rate, determines the efficacy of TGI [14]. This implies that if TGI is used to augment alveolar ventilation during inverse ratio ventilation, one needs to apply progressively higher flow rates as the expiratory time is shortened.

Currently, there is no standard method for introducing the insufflation catheter into the trachea. In most human studies, a small-caliber catheter was introduced through an angled side-arm adapter attached to the endotracheal tube (ETT) and positioned just above the main carina [4,15-17]. Catheter placement was usually performed with bronchoscopic guidance or estimated from a recent chest roentgenogram. This type of system is simple to construct and can be duplicated in most intensive care units but suffers from some drawbacks. Placement of a catheter through the endotracheal tube interferes with suctioning of patients. Moreover, the catheter is not fixed in space and may cause bronchial mucosal injury if it whips within the trachea at high flows. New ETT designs that incorporate channels within the endotracheal tube wall would solve these problems and simplify application of TGI. Isabey et al embedded small capillaries in the walls of an ETT for tracheal injection of gas [18]. The same group have also used this modified ETT to deliver high velocity jets of O_2 desaturation during suctioning [19]. Although lung volumes did not change during suctioning when O_2 was delivered in this fashion, the rate of rise of $PaCO_2$ was similar to that observed during apnea. The modified ETT has been used in animal models [20] and can be used to perform TGI in patients. Most likely future clinical applications of TGI will use a modified ETT that incorporates the catheter in its wall attached to a standardized circuit for gas delivery.

Monitoring during TGI

Airway opening pressure and lung mechanics

TGI-induced dynamic hperinflation causes airway opening pressure (Pao) to underestimate lung volume during end-expiration [5,14]. In an experimental study, monitoring tracheal pressure 2 cm beyond the tip of the catheter seemed to gauge lung volume changes at end-expiration accurately, suggesting that tracheal pressures should be monitored beyond the jet stream during TGI [5]. During pan-expiratory TGI, however, catheter flow ceases during inspiration, and inspiratory Pao provides as much useful information as during CMV.

TGI may also interfere with the ability of the clinician to measure lung mechanics. Respiratory system compliance and auto-positive end-expiratory pressure measurements require application of a pause at end-inspiration and at end-expiration. If catheter flow continues during these measurements, the pressure within the respiratory system builds up with time. Consequently, a plateau pressure cannot be obtained and, if unnoticed, alveolar pressure can increase to hazardous levels. During pan-expiratory TGI, depending on the timing and nature of the signal that gates the solenoid to divert catheter flow to the atmosphere, these measurements can still be made safely. Nevertheless, it is advisable to test the TGI-ventilator system using a mechanical lung model under controlled conditions prior to measuring lung mechanics at the bedside.

Tidal volume (VT)

Whenever catheter flow is delivered during inspiration it contributes to total inspired VT. The contribution to total inspired VT is eliminated if TGI is timed to occur only during expiration [3]. Even then decompression of the TGI circuit into the ventilator circuit during the inspiratory phase of solenoid closure contributes to total inspired VT [21]. In most TGI circuits, however, this volume is rather small (≃ 10–20 ml at a Vc of 101/min). These problems are obviated if an independent measure of VT such as inductive plethysmography is used. The effect of TGI on total inspired VT depends on the mode of operation of the ventilator.

Flow-controlled, volume-cycled ventilation

During continuous TGI, total inspired VT is composed of two components: that delivered by the ventilator (VTv) and that delivered by the catheter (VTc). The total inspired VT is then given by the sum of these two components (VT 5 VTv 1 VTc). The contribution of continuous TGI to total inspired VT can be estimated from the duration of inspiration (TI) and Vc as: VTc 5 TI × □c. Consequently, during flow-controlled, volume-cycled ventilation, total inspired VT can be maintained relatively constant during continuous TGI by decreasing the ventilator-set VT by an amount equal to VTc [15,21].

Pressure-controlled ventilation

During pressure-controlled ventilation (PCV), application of TGI does not change the total inspired VT provided TGI does not pressurize the respiratory system beyond the ventilator set-pressure. As Vc is increased, the ventilator-delivered VT declines but the total inspired VT remains the same [10,22]. The respiratory system behaves in this manner as long as VTc is less than the VT generated by the set-pressure under PCV conditions without TGI. If VTc exceeds the VT generated by PCV in the absence of TGI, then TGI will overpressurize the circuit and peak Pao will be greater than that produced by the ventilator set-pressure. Almost all ventilators allow pressures higher than that generated by the set-pressure as long as Pao remains below the high-pressure limit of the ventilator. Consequently, excessive

pressures can be produced within the respiratory system if the product of □c × TI is too large. When this happens, the Pao time-profile becomes a hybrid of PCV and constant-flow volume-cycled ventilation, resembling that generated during volume-assured pressure support ventilation [23]. This problem can be circumvented by introducing a pressure-release valve into the ventilator circuit that dumps circuit pressure above a set threshold [24].

Monitoring efficacy of TGI

The primary effect of TGI is on physiologic deadspace due to a reduction in anatomic deadspace. However, the clinically monitored parameter at the bedside is usually $PaCO_2$ or capnography. TGI modifies the profile of the expired capnogram since fresh gas delivered by the catheter dilutes the CO_2 exhaled from the lungs. The capnogram measures exhaled CO_2 at the tip of the ETT and as a result monitors the exhaled CO_2 with a time (and volume) lag relative to the catheter tip. Nevertheless, the exhaled capnogram can be used qualitatively to gauge the completeness of expiratory washout. Clearly, if end-tidal PCO_2 declines to very low values (< 3 mmHg), expiratory washout is most likely complete and further increments in Vc may not impact $PaCO_2$ greatly. However, if more CO_2-laden gas is removed from the periphery of the lung by the distal effects of TGI as Vc is increased, end-tidal PCO_2 may remain elevated or actually rise. Consequently, the relationship between the efficacy of TGI and the capnographically measured CO_2 profile is quite complex. Nevertheless, certain observations can be made based on capnographically observed measurements.

The fraction of unperfused alveoli can be estimated in terms of $PaCO_2$ and average end-tidal PCO_2 ($PETCO_2$) as ($PaCO_2$-$PETCO_2$)/ $PaCO_2$ [25]. This ratio can be readily measured at the bedside by capnography and arterial blood gas analysis. Since increasing the fraction of unperfused alveoli decreases the efficacy of TGI (as measured by percentage change in VD and $PaCO_2$ relative to baseline conditions), the ratio ($PaCO_2$-$PETCO_2$)/ $PaCO_2$ may provide useful clinical information. Indeed, in both animal [8] and human studies [17,26] this ratio was closely correlated ($r \simeq 0.70$) with observed percentage reduction in $PaCO_2$ from baseline during TGI. The exact role of capnography to monitor the efficacy of TGI remains to be established, but available data suggest that it may be used to optimize Vc and ventilator settings.

TGI and ventilator interactions

Since TGI introduces an external flow source independent of the ventilator it can adversely affect the ability of the ventilator to monitor pressures and volumes and may cause the ventilator to alarm incessantly. Presence of catheter flow during expiration disables the monitoring role of the expiratory pneumotachograph of the ventilator causing some ventilators to alarm when the difference between the measured inspired and exhaled volumes exceed a certain value. The presence of an external flow that can pressurize the ventilator circuit also interferes with the ability of the ventilator to detect a leak. These problems can be easily circumvented if the TGI circuit is incorporated within the operational framework of the ventilator.

Clinical applications

The effect of a given TGI-Induced change in VD and $PaCO_2$ depends on the $PaCO_2$ and VD/VT values prior to the initiation of TGI, and the effect of TGI, and the effect of TGI on CO_2 production [8]. For a given fractional change in VD, the percentage change in $PaCO_2$ increases dramatically as VD/VT exceeds 0.07 [9]. This implies that the effect of TGI on $PaCO_2$ is amplified as the respiratory system is allowed to operate at higher VD/VT (in other words, permissive hypercapnia). Consequently, TGI becomes more effective in decreasing $PaCO_2$ in the setting of hypercapnia [9].

In patients with acute lung injury (ALI), part of the deadspace resides in the alveoli as alveolar deadspace. The alveolar gas originating from those ventilated but hypoperfused lung regions are CO_2-poor. Consequently, gas expired from alveolar deadspace dilutes CO_2-laden gas residing in the proximal anatomic deadspace. Thus, the impact of washing proximal deadspace free of CO_2 on alveolar ventilation diminishes [13]. Adopting a permissive hypercapnia strategy increases the amount of CO_2 that can be removed from the proximal anatomic deadspace and counterbalances the decreased CO_2 removal efficacy of TGI caused by increased alveolar deadspace.

As an adjunct to a pressure-targeted, lung-protective ventilatory strategy,TGI can be used to pursue one of two goals. It can be used to limit the extent of hypercapnia and/or to control the rate of rise of $PaCO_2$ while maintaining VT and minute ventilation (□E) as constant. Alternatively, it can be used to limit ventilatory distending forces (allowing a reduction in VT) while maintaining a constant $PaCO_2$.

TGI has been applied successfully to mechanically ventilated patients. Ravenscraft *et al* administered TGI to eight patients with acute respiratory failure while maintaining total inspired VT as constant [26]. In this study, TGI decreased $PaCO_2$ by 15% (from 53.1 ± 3.4 mmHg to 45.0 ± 2.1 mmHg) at a flow rate of 61/min. Similarly, Nakos *et al* [15] were able to decrease $PaCO_2$ by 25% (from 46.0 ± 4.8 mmHg to 34.5 ± 6.2 mmHg) by using TGI at 61/min while maintaining □E as

constant. The same group found a similar flow-dependent reduction in $PaCO_2$ during TGI in mechanically ventilated chronic obstructive pulmonary disease (COPD) patients [16]. In COPD patients with tracheostomies, however, TGI was ineffective in reducing $PaCO_2$ (from 56 ± 4 mmHg at baseline to 53 ± 5 mmHg at a □c of 61/min) due to the reduction in flushable anatomic deadspace [16]. Kuo *et al* [17] studied the effects of TGI in 20 acute respiratory distress syndrome (ARDS) patients who had baseline (Vc 5 01/min) $PaCO_2$ of 55 ± 8 mmHg. TGI decreased $PaCO_2$ by 13% and 17% (by 7.2 and 9.1 mmHg) at a Vc of 4 and 61/min, respectively. Such modest reductions in $PaCO_2$ reflect the predominance of alveolar over series deadspace in conditions of severe parenchymal lung disease. However, using smaller tidal volumes with permissive hypercapnia should increase baseline $PaCO_2$ and accentuate the relative efficacy of TGI. Belghith *et al* studied the effects of TGI in six ARDS patients with significant hypercapnia at baseline [27]. In their study, TGI at 41/min on average decreased $PaCO_2$ by 24 mmHg from 108 ± 32 to 84 ± 26 mmHg. Similarly, Kalfon *et al* using pan-expiratory TGI at 151/min were able to decrease $PaCO_2$ by 30% (from 76 ± 4 mmHg to 53 ± 3 mmHg) in ARDS patients during permissive hypercapnia [28]. The favorable response to TGI in these studies [27,28] was probably due to hypercarbia at baseline. These clinical studies suggest that, in the seting of ARDS, TGI is a useful technique to limit the level of $PaCO_2$ during a permissive hypercapnia strategy. If a $PaCO_2$ threshold of 70–80 mmHg is used to initiate TGI, then TGI can be used to decrease $PaCO_2$ by 20–30% in 15–20% of ARDS patients [29-31].

Using TGI in a canine oleic acid-induced pulmonary edema model, adequate alveolar ventilation was maintained at much smaller tidal volumes and pressures than were required without TGI [21]. Compared to CMV, the valved TGI catheter (which functioned as the ventilator in this setting) achieved the same ventilatory task at 35% of the VT and 70% of elastic end-inspiratory pressure. Using a similar strategy, Muller *et al* demonstrated that they could adequately ventilate sheep that had undergone resection of 88% of their lung tissue, without resorting to excessive VE or airway pressures [32]. Nakos *et al* [15] used TGI at 6–8 1/min to decrease VT and peak Pao by 25% and 20%, respectively, while maintaining $PaCO_2$ constant in seven patients with ALI.

In normal dogs, TGI tended to reduce venous admixture (QV/QT) and increase PaO_2 [5,10,22], a tendency we also observed in eight critically ill patients; however, the rise in PaO_2 was not significan [26]. In this clinical setting, continuous TGI (61/min) consistently increased end-expiratory lung volume (FRC) slightly (62 ± 16 ml at a □c of 61/min in four patients) [26]. The response in oxygenation, however, was variable: PaO_2/PAO_2 increased in four out of eight patients, remained the same in two, and decreased in two. In the study by Kuo *et al* [17], TGI did not significantly affect PaO_2 of ARDS patients. Similarly, in six oleic-acid injured dogs, TGI did not improve PaO_2 or QV/QT when VT and FRC were kept constant [9]. However, Nakos *et al* [15] observed a significant increase in PaO_2 during TGI at 6 1/min (from 82 ± 6 mmHg to 89 ± 6 mmHg, $P < 0.05$). Similarly, Kalfon *et al* [28] observed a significant improvement in PaO_2 during TGI (from 205 ± 28 mmHg to 296 ± 38 mmHg, $P < 0.001$). In both studies [15,28], VT was maintained constant but no attempt was made to control FRC during TGI. Current data suggest that TGI does not impact oxygenation provided that total inspired VT and FRC are not augmented during application of TGI.

When direction of catheter flow is reversed (in other words, catheter flow is directed towards the mouth) the effect of TGI on FRC diminishes [5]. With novel reversethrust catheter design, FRC can be adjusted by changing TGI flow rate. Consequently, reverse-thrust catheters can be used to eliminate dynamic hyperinflation during TGI [33].

The minute ventilation-sparing effect of TGI may also be used to reduce the work of breathing in some intubated patients. However, TGI may impair the ability of some patients to trigger the ventilator, because the patient's inspiratory effort must first outstrip catheter flow and overcome the dynamic hyperinflation caused by TGI in order to lower airway opening pressure below the trigger threshold [34]. The net effect of TGI on the work of breathing would depend on the interactions between TGI and the ventilator as well as the efficiency of TGI in decreasing deadspace and □E requirements. Obviously, incorporating TGI into a flow-by system would preserve the combined benefits of flow-triggering and improved gas exchange associated with TGI. Patients with neuromuscular weakness who have relatively normal lung parenchyma and retain CO_2 because they can only generate small tidal volumes (in other words, proximal anatomic deadspace contributes significantly to VD/VT) are excellent candidates for TGI. A reverse-thrust TGI may also maintain the patency of ETT by clearing tracheal secretions from the ETT lumen [33].

Potential complications

Although a promising adjunct to CMV, TGI is not without potential complications. When high flows are delivered into the airways, any obstruction to outflow of gas could potentially cause overinflation of the lungs within seconds, and may cause pneumothorax, pulmonary

venous air embolism, and/or hemodynamic compromise. Esophageal pressure and/or chest wall monitoring may be required to monitor changes in lung volume. A second concern is bronchial mucosal damage due to impact of the jet stream on the to the bronchial mucosa as well as the possible physical impact of the catheter tips from oscillations secondary to high flows. The force created by the jet stream impacting on the surface can be quite high and may account for the bronchial mucosal damage observed in experiments during constant flow ventilation [35]. Proper humidification of the inspired gas is essential at such high flow rates. Long-term use of TGI may result in inspiration or retention of secretions, especially if the insufflated gas is not adequately humidified. The presence of the gas stream and the catheter within the trachea may increase mucous production and may magnify this problem [36-38].

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References

1. Jonson B, Similowski T, Levy P, Viires N, pariente R: **Expiratory flushing of airways: a method to reduce deadspace ventilation.** *Eur Respir J* 1990, **3**:1202-1207.
2. Gilbert J, Larsson A, Smith B, Bunegin L: **Intermittent-flow expiratory ventilation (IFEV): delivery technique and principles of action – a preliminary communication.** *Biomed Instrument Technol* 1991, **25**:451-456.
3. Burke WC, Nahum A, Ravenscraft SA, *et al*: **Modes of tracheal gas insufflation. Comparison of continuous and phase-specific gas injection in normal dogs.** *Am Rev Respir Dis* 1993, **148**:562-568.
4. Ravenscraft SA, Shapiro R, Nahum A, *et al*: **Tracheal gas insufflation: catheter effectiveness is determined by expiratory flush volume.** *Am J Respir Crit Care Med* 1996, **153**:1817-1824.
5. Nahum A, Ravenscraft SA, Nakos G, *et al*: **Effect of catheter flow direction on CO_2 removal during tracheal gas insufflation in dogs.** *J Appl Physiol* 1993, **75**:1238-1246.
6. Slutsky As, Watson J, Leith DE, Brown R: **Tracheal insufflation of O_2 (TRIO) at low flow rates sustains life for several hours.** *Anesthesiology* 1985, **63**:278-286.
7. Nahum A, Ravenscraft SA, Adams AB, Marini JJ: **Distal effects of tracheal gas insufflation: changes with catheter position and oleic acid lung injury.** *J Appl Physiol* 1996, **81**:1121-1127.
8. Nahum A, Shapiro RS, Ravenscraft SA, Adams AB, Marini JJ: **Efficacy of expiratory tracheal gas insufflation in a canine model of lung injury.** *Am J Respir Crit Care Med* 1995, **152**:489-495.
9. Nahum A, Chandra A, Niknam J, Ravenscraft SA, Adams AB, Marini JJ: **Effect of tracheal gas insufflation on gas exchange in canine oleic acid-induced lung injury.** *Crit Care Med* 1995, **23**:348-356.
10. Nahum A, Burke W, Ravenscraft SA, *et al*: **Lung mechanics and gas exchange during pressure-controlled ventilation in dogs: augmentation of CO_2 elimination by an intratracheal catheter.** *Am Rev Respir Dis* 1992, **146**:965-973.
11. Eckmann DM, Gavriely N: **Intra-airway CO_2 distribution during airway insufflation in respiratory failure.** *J Appl Physiol* 1995, **78**:546-554.
12. Gavriely N, Eckmann DM, Grotberg JB: **Intra-airways gas transport during high-frequency chest vibration with tracheal insufflation in dogs.** *J Appl Physiol* 1995, **79**:243-250.
13. Nahum A, Marini JJ: **Tracheal gas insufflation as an adjunct to conventional ventilation.** *In Yearbook of Intensive Care and Emergency Medicine, vol 17. Edited by Vincent JL. Berlin: Springer-Verlag;* 1994.
14. Nahum A, Sznajder JI, Solway J, Wood LDH, Schumacker PT: **Pressure, flow, and density relationships in airway models during constant-flow ventilation.** *J Appl Physiol* 1988, **64**:2066-2073.
15. Nakos G, Zakinthinos S, Kotanidou A, Roussos C: **Tracheal gas insufflation reduces the tidal volume while $PaCO_2$ is maintained constant.** *Intensive Care Med* 1994, **20**:407-413.
16. Nakos G, Lachana A, Prekates A, *et al*: **Respiratory effects of tracheal gas insufflation in spontaneously breathing COPD patients.** *Intensive Care Med* 1995, **21**:904-912.
17. Kuo P-H, Wu H-D, Yu C-J, Yang S-H, Lai Y-L, Yang P-C: **Efficacy of trachal gas insufflation in acute respiratory distress syndrome with permissive hypercapnia.** *Am J Respir Crit Care Med* 1996, **154**:612-616.
18. Isabey D, Boussignac G, Harf A: **Effect of air entrainment on airway pressure during endotracheal gas injection.** *J Appl Physiol* 1989, **67**:771-779.
19. Brochard L, Mion G, Isabey D, *et al*: **Constant-flow insufflation prevents arterial oxygen desaturation during endotracheal suctioning.** *Am Rev Respir Dis* 1991, **144**:395-400.
20. Pinquier D, Pavlovic D, Boussignac G, Aubier M, Beafils F: **Benefits of low pressure multichannel endotracheal ventilation.** *Am J Respir Crit Care Med* 1996, **154**:82-90.
21. Nahum A, Reavenscraft SA, Adams AB, Marini JJ: **Inspiratory tidal volume sparing effects of tracheal gas insufflation in dogs with oleic acid-induced lung injury.** *J Crit Care* 1995, **10**:115-121.
22. Nahum A, Ravenscraft SA, Nakos G, *et al*: **Tracheal gas insufflation during pressure-control ventilation. Effect of catheter position, diameter, and flow rate.** *Am Rev respir Dis* 1992, **146**:1411-1418.
23. Amato MBP, Barbas CSV, Bonassa J, Saldiva PHN, Zin VA, Carvalho CRR: **Volume-assured Pressure support ventilation (VAPS): a new approach for reducing muscle workload during acute respiratory failure.** *Chest* 1992, **102**:1225-1234.
24. Gowski D, Delgado E, Miro AM, *et al*: **Tracheal gas insufflation during pressure-control ventilation: effect of using a pressure relief valve.** *Crit Care Med* 1997, **25**:145-152.
25. Workman JM, Penman WB, Bromberger-Barnea B, Permutt S, Riley RL: **Alveolar dead space, alveolar shunt, and transpulmonary pressure.** *J Appl Physiol* 1965, **20**:816-824.
26. Ravenscraft SA, Burke WC, Nahum A, *et al*: **Tracheal gas insufflation augments CO_2 clearance during mechanical ventilation.** *Am Rev Respir Dis* 1993, **148**:345-351.
27. Belghith M, Fierobe L, Brunet F, Monchi M, Mira J-P: **Is tracheal gas insufflation an alternative to extrapulmonary gas exchangers in severe ARDS?** *Chest* 1995, **107**:416-419.
28. Kalfon P, Rao U, Gallart L, Puybasset L, Coriat P, Rouby J-J: **Permissive hypercapnia with and without expiratory washout in patients with severe acute respiratory distress syndrome.** *Anesthesiology* 1997, **87**:6-17.
29. Hickling KG, Henderson SJ, Jackson R: **Low mortality associated with low volume pressure limited ventilation with permissive hypercapnia in severe adult respiratory distress syndrome.** *Intensive Care Med* 1990, **16**:372-380.
30. Hickling KG, Walsh J, Henderson S, Jackson R: **Low mortality rate in adult respiratory distress syndrome using low-volume, pressure-limited ventilation with permissive hypercapnia: a prospective study.** *Crit Care Med* 1994, **22**:1568-1578.
31. Amato MBP, Barbas CLV, Mederios DM, *et al*: **Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome.** *N Engl J Med* 1998, **338**:347-354.
32. Muller E, Kolobow T, mandava S, *et al*: **On how to ventilate lungs as small as 12% of normal. Intratracheal pulmonary ventilation (ITPV). A new mode of pulmonary ventilation [abstract].** *Am Rev Respir Dis* 1991, **143**: A693.
33. Trawoger R, Kolobow T, Cereda M, *et al*: **Clearance of mucous from endotracheal tubes during intratracheal pulmonary ventilation.** *Anesthesiology* 1997, **86**:1367-1374.
34. Hoyt JD, Marini JJ, Nahum A: **Effect of tracheal gas insufflation on demand valve triggering and total work during continuous positive airway pressure ventilation.** *Chest* 1996, **110**:775-783.
35. Sznajder JI, Nahum A, Crawford G, Pollak ER, Schumarker PT, Wood LDH: **Alveolar pressure inhomogeneity and gas exchange during constant-flow ventilation in dogs.** *J Appl Physiol* 1989, **67**:1489-1496.
36. Burton GG, Wagshul FA, Henderson D, Kime SW: **Fatal airway obstruction caused by a mucous ball from a transtracheal catheter.** *Chest* 1991, **99**:1520-1521.

37. Fletcher EC, Nickeson D, Costarangos-Galarza C: **Endotracheal mass resulting from a transtracheal catheter.** *Chest* 1988, **93**:438-439.
38. Christopher KL, Spofford BT, Petrun MD, McCarty DC, Goodman JR, Petty TL: **A program for transtracheal oxygen delivery. Assessment of safety and efficacy.** *Ann Intern Med* 1987, **107**:802-808.

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